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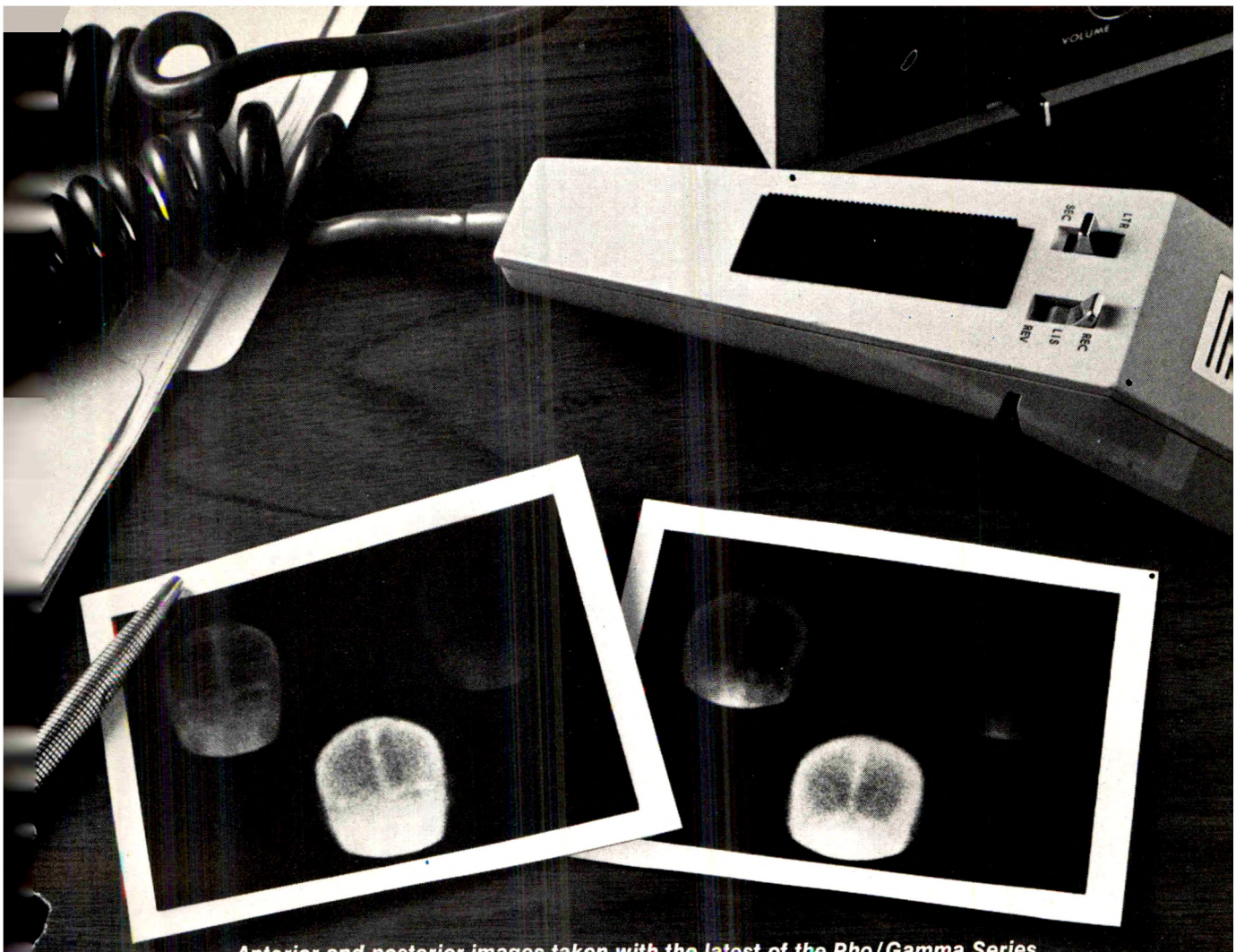
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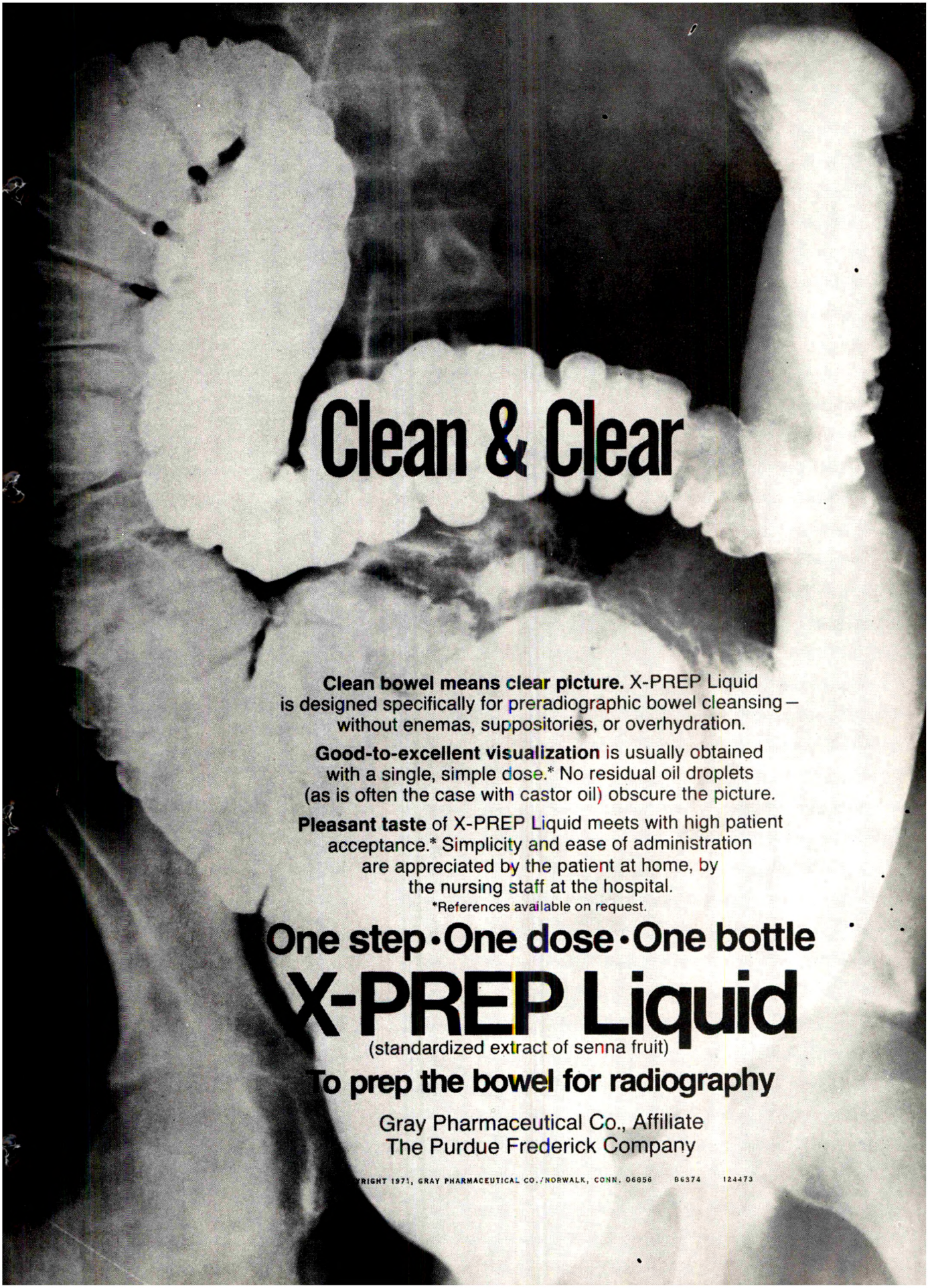
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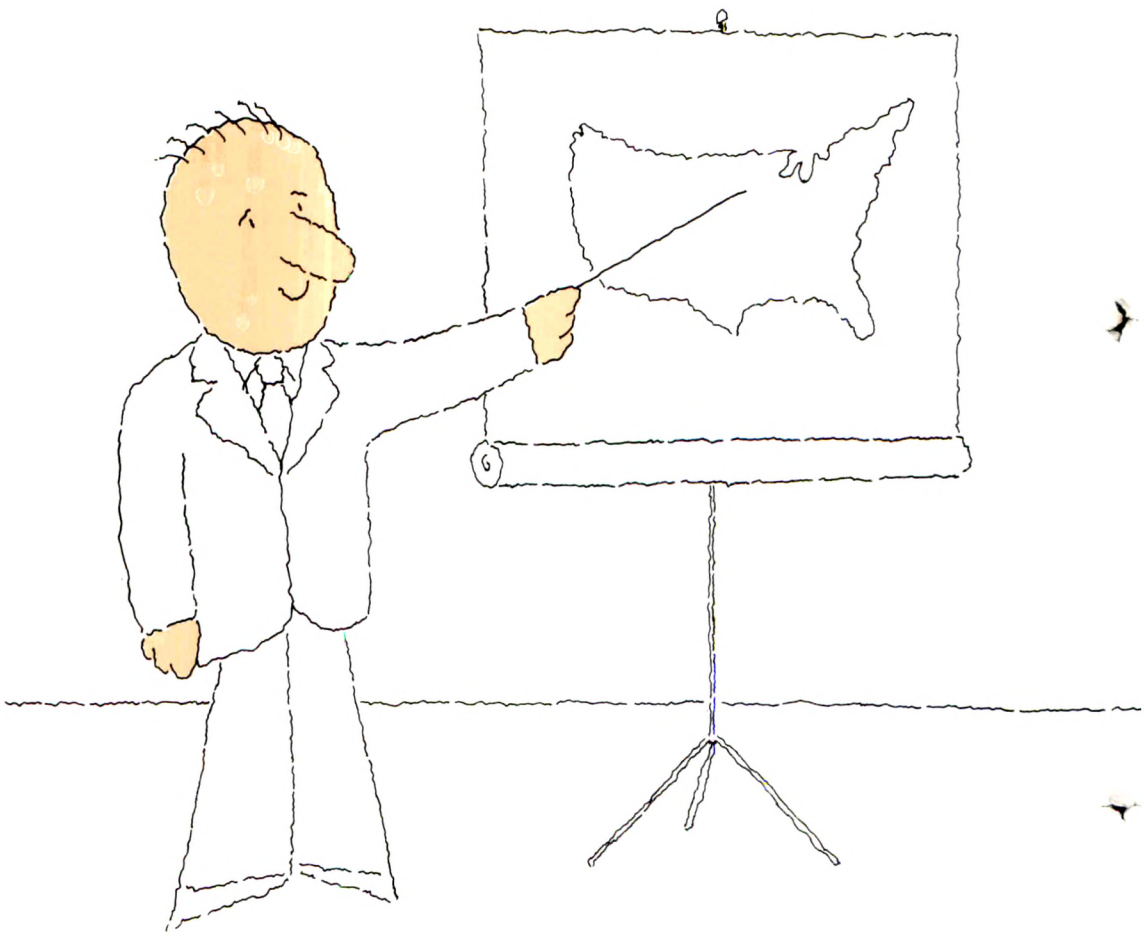
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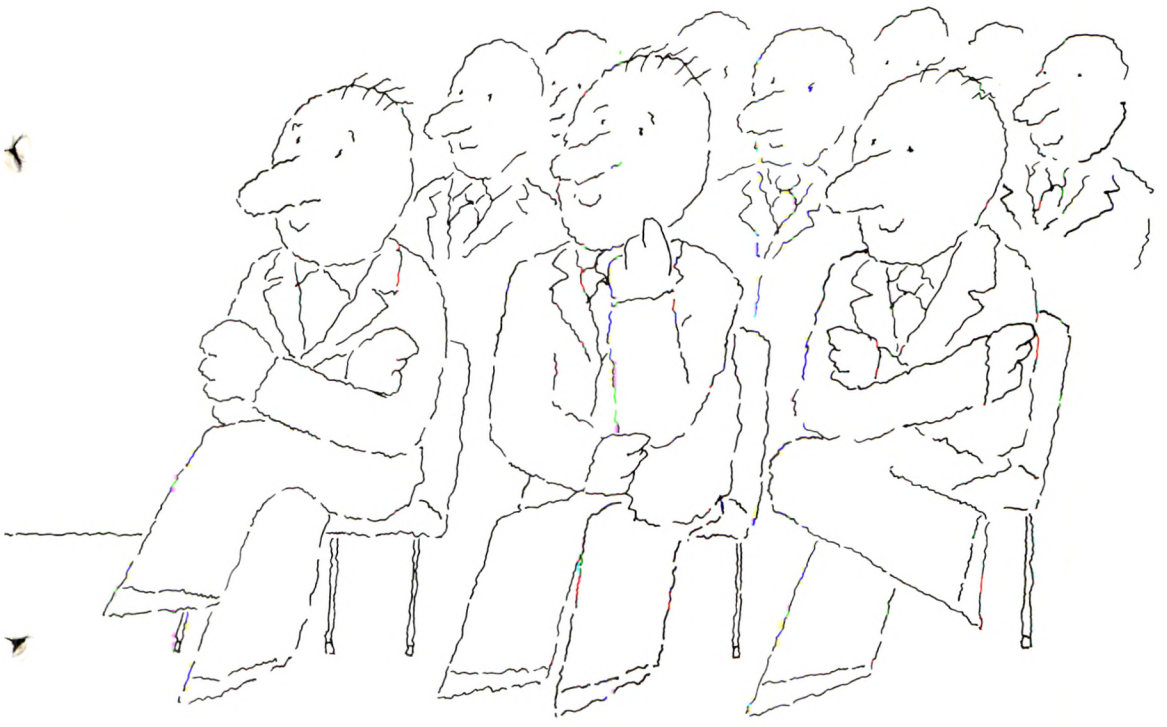
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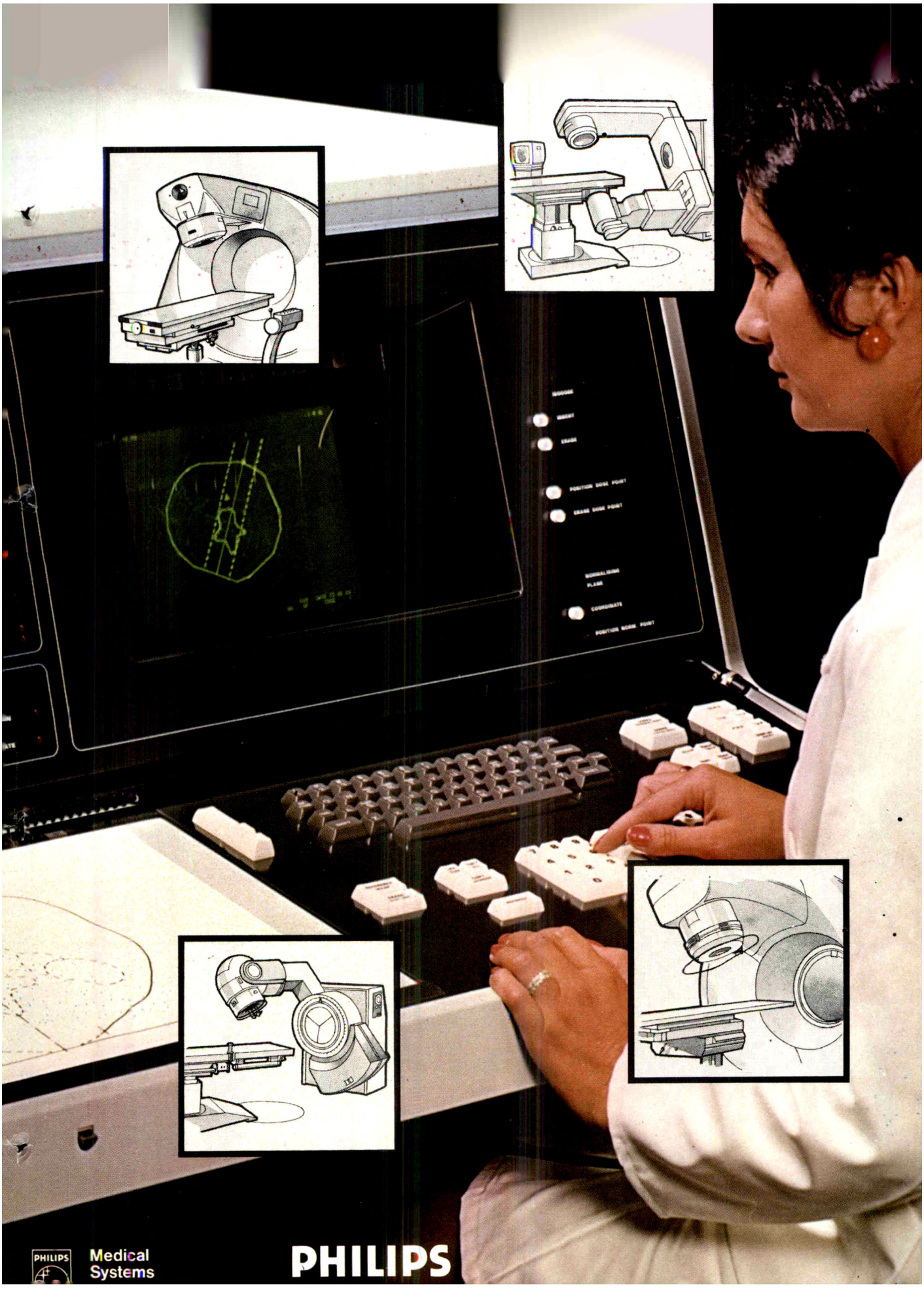
The Philips Treatment Planning System represents and demonstrates the high degree of technological design that is intrinsic to Philips entire radiotherapy programme.

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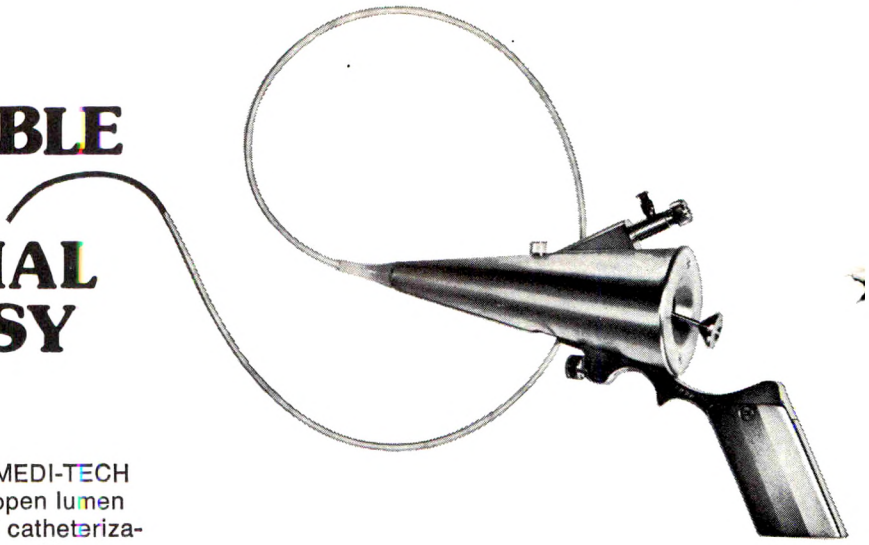
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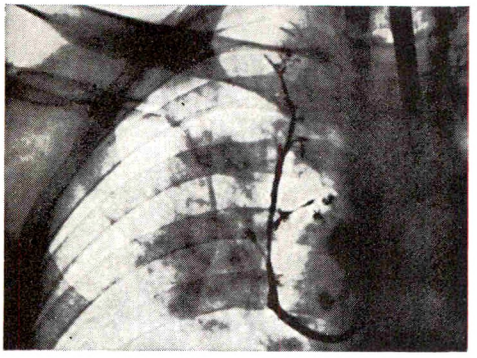
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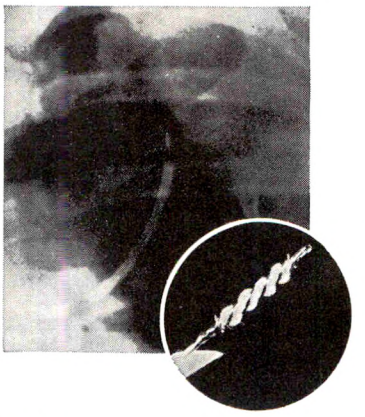
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Zavala, Rossi, Rodman, Bedell and Pixley: A New Mobile Catheter for Obtaining Bronchial Brush Biopsies: Diagnostic Results in 50 Patients with Suspicious Pulmonary Lesions and Negative Bronchoscopies, AMERICAN REVIEW OF RESPIRATORY DISEASE, Vol. 106, No. 4, pp 541-547, October, 1972.

Zavala, Rossi and Bedell: Bronchial Brush Biopsy: A Valuable Diagnostic Technique in the Presurgical Evaluation of Indeterminate Lung Densities, THE ANNALS OF THORACIC SURGERY, Vol. 13, No. 6, pp 519-528, June, 1972.

Thiede, Banaszak; Selective Bronchial Catheterization, NEW ENGLAND JOURNAL OF MEDICINE, 286: 526-528, March 9, 1972.

Norris, Tucker and Woloshin; Bronchoesophagologic Application of Recent Advances in Fluoroscopy, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 80, No. 4, pp 528-535, August, 1971.

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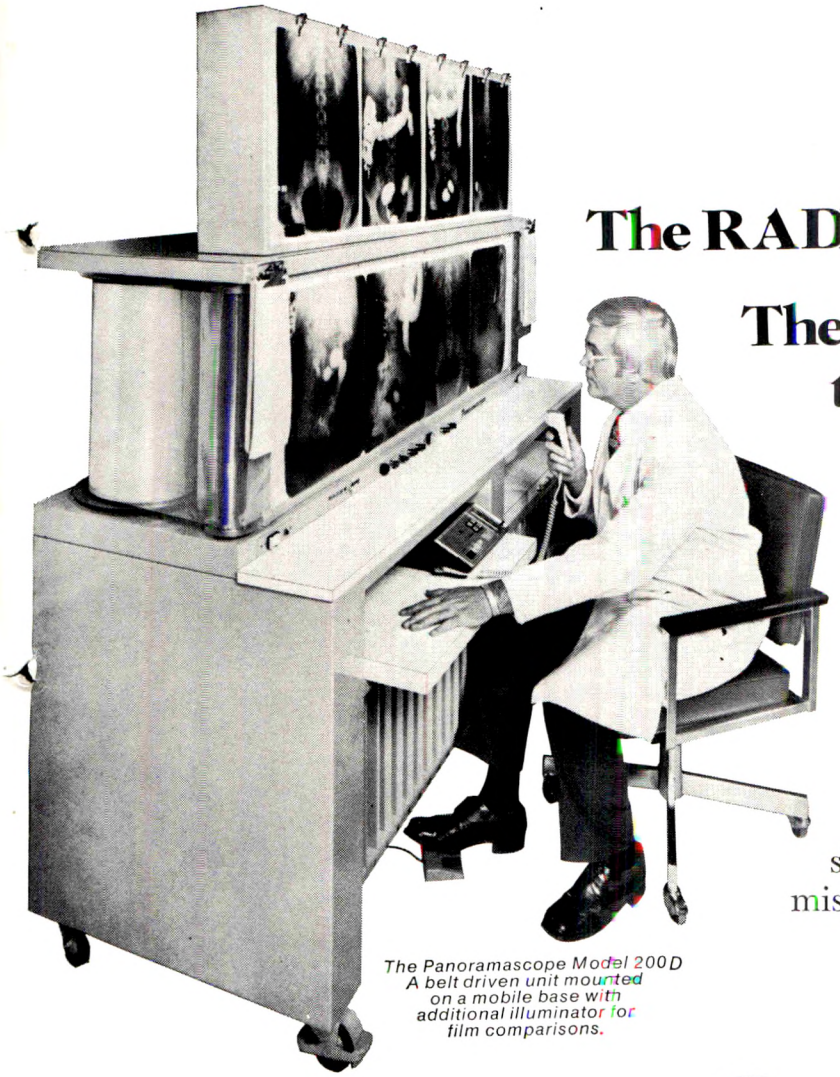
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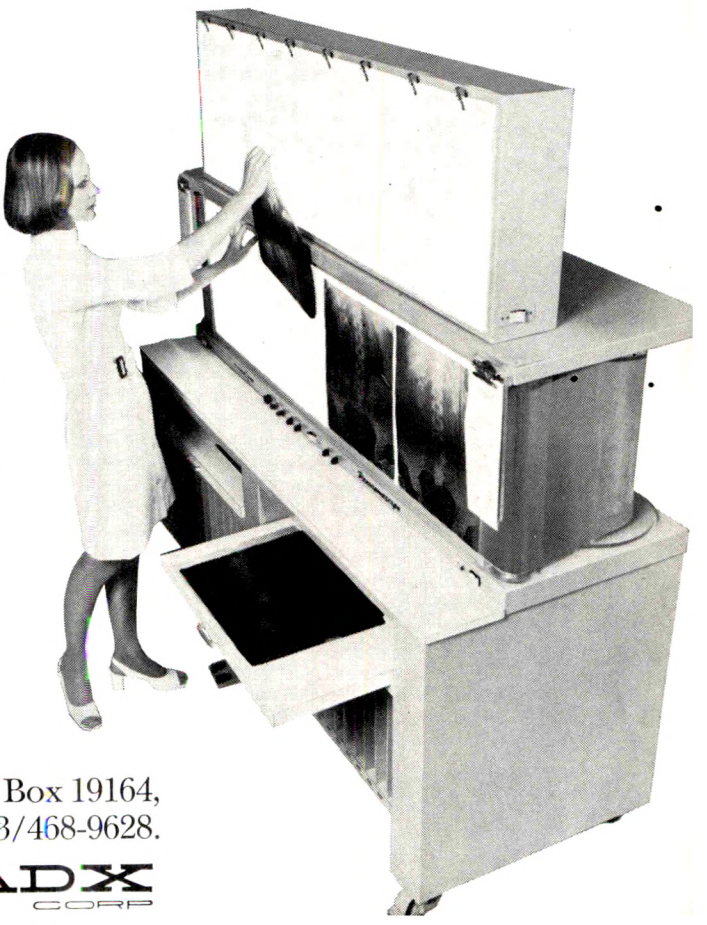
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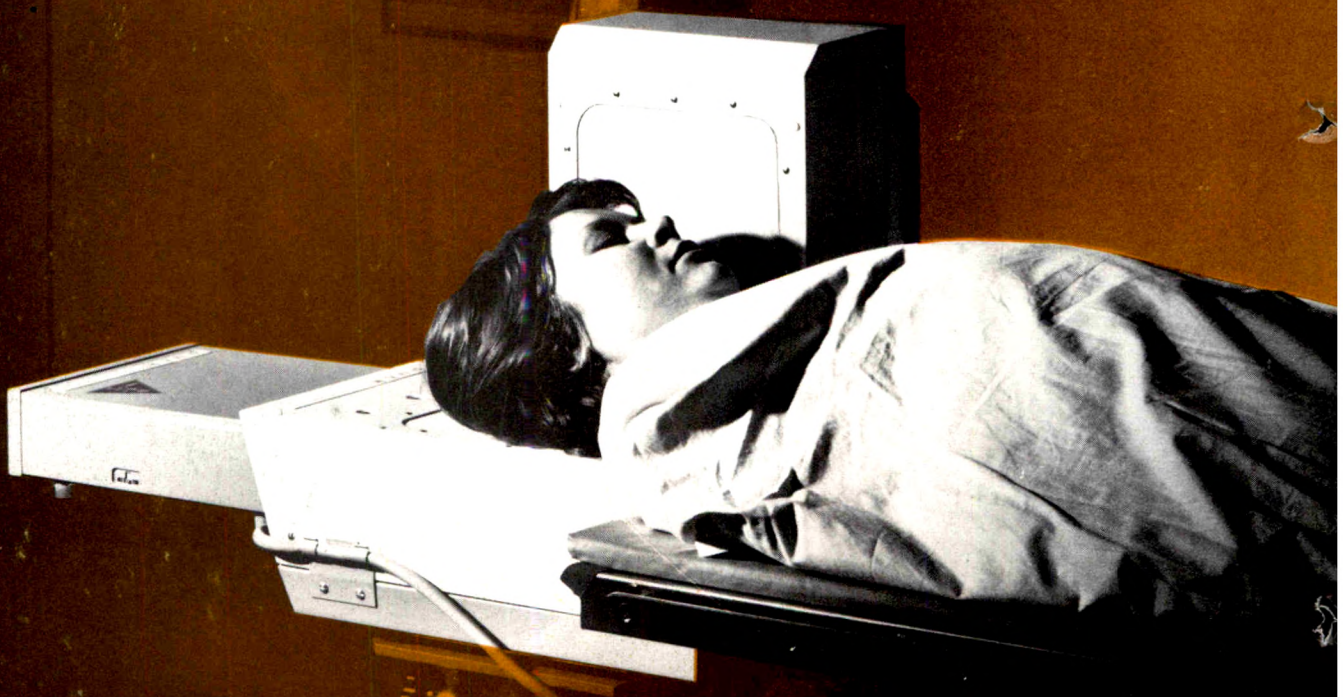


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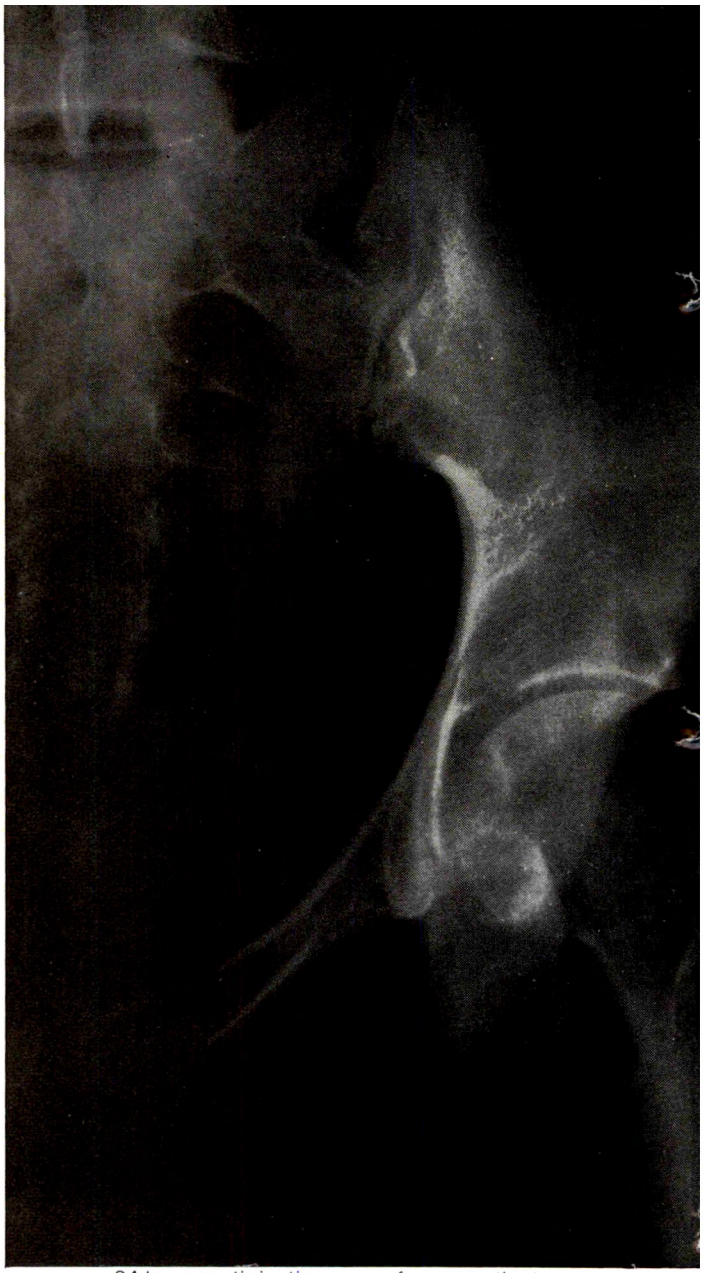


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PEDIATRIC RADIOLOGY AND SYNDROMOLOGY*

NEUHAUSER LECTURE, 1973

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Dr. and Mrs. Neuhauser, Dr. Taybi, Members of the Society for Pediatric Radiology and Guests: I am very happy and honored to give this Annual Lecture dedicated to Dr. Neuhauser on a subject of great interest to pediatricians and radiologists in their search for unification and classification in a field of kaleidoscopic diversity. In the description of signs and symptoms of multiple congenital malformations radiologic findings are essential for delineation of disease pictures, which cannot be fully recognized by external examinations. Caffey[4] has pointed out that the Boston Children's Hospital had the first roentgen department in America, organized exclusively for the use of infants and children, and that the most common subjects considered there were tuberculosis, osteomyelitis and congenital malformations. For more than 3 decades Dr. Neuhauser has upheld the tradition and thanks to his interest in rare and unusual manifestations has made many contributions to clinical teratology. At the same time he has aroused interest in this subject in many of his pupils who practice pediatric roentgenology in this country and abroad.

COMBINATIONS of congenital malformations, syndromes of structural anomalies present at birth, are striking phenomena which have attracted the attention of observers since early times. In the strict sense any case of multiple congenital malformations is a syndrome when several defects "run together" and each case of this type should be noted and analyzed. It is clear, however, that the number of combinations of congenital defects is infinite and that not every case deserves recording in the medical literature. Yet, it is difficult to decide which combination is important to the general medical public and which is not. To the patient and to the morphologist, any anatomic sign and symptom is significant, whether it has general implications or not; however, there is some unwritten consent that only those syndromes deserve attention and communication in the medical literature that occur repeatedly, particularly if they recur in the same family or kindred or in an epidemic or endemic form. Repeated occurrence results in a need for a name and classification; fur-

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thermore it holds a promise for etiologic research and preventive measures.

Although syndromes of congenital malformations have been seen by pediatricians since our specialty began, it has been only recently that syndromology has become a respectable branch of pediatrics. Some years ago our residents had little interest in patients with multiple congenital malformations and classified them as *gooks*, *geeks* or *crockheads*. Now this has changed. There is interest in syndromes for which labels are eagerly sought, but unfortunately the labels of today are often not much better than those attached by the frivolous residents of the past. I have dealt with the inconsistencies, difficulties and ironies of syndrome terminology elsewhere and should like to omit this here except when serious consequences require such discussion.[31]

The interest in syndromes is most gratifying and beneficial at a time when specialization continues to grow and flourish. Specialization, to a great extent, is directed to diagnosis and treatment of specific organs: the eye, ear, mouth, heart, lung, *etc*. Syndromes of congenital malformations indicate as a rule that a patient's disorder extends beyond a single organ. Knowledge of syndromes are educational for organ specialists, since it widens their viewpoint beyond the horizons of their specialty. Pediatrics as well as roentgenology are not "organ-specific" specialties; it is understandable that our branches of medicine have long shown interest in syndromes. The pediatrician considers the entire child, and associations of distant and heterogeneous signs and symptoms cannot escape him. If his methods of examination are not sufficient, he can call on other specialists, particularly the roentgenologist, who adds findings that are not recognizable without special diagnostic aids. The situation of the pediatric roentgenologist is somewhat different from that of the clinician at the bedside. He may be asked to look for a fracture of the bones of a forearm or leg and describe his findings. At the same time he may notice osseous changes which suggest to him the possibility of renal or cardiac or hematologic disease; or he may notice signs of disease in an area not suspected by the clinicians. We all have seen such reports by alert roentgenologists who suggest a diagnosis not considered by the clinician. How far can the roentgenologist go to substantiate his suspicion of a syndrome? His methods of examination are limited not only by costs, but also by the dangers of his method, the roentgen rays. He is handicapped, if an associated anomaly resides in an organ, such as the eye, the thyroid, the brain, whose details and functions are not readily revealed by x rays, although increasingly technical advances give him access to investigation of soft tissues previously not included in roentgen diagnosis. Limitations of diagnostic ability of the roentgenologist and the pediatrician point to the need for cooperation of these specialists which has been most fruitful in the past and must be further developed in the future. This need for cooperation in diagnosis can best be illustrated by the existence of syndromes, the existence of signs and symptoms, which "run together" and are not limited to any single organ system.

Etiology of syndromes should be of greatest interest to all of us. Pediatric roentgenology, although chiefly devoted to diagnosis and therapy, can make important contributions to etiologic research. If we consider all cases of multiple congenital malformations as syndromes, then we must distinguish between accidental associations and associations with a common cause or common root. Thus association of a cataract caused by rubella with a hereditary lobster claw is probably an accidental syndrome, whereas cataract and congenital heart disease can have a common root, namely prenatal rubella. This seems perfectly clear to us when we know the etiologies of the anomalies, but to the naive observer both associations are syndromes and in many cases we all are naive observers. Our aim must be to distinguish between syndromes that are casually and those that are causally connected. How can we make

such distinctions? One may think that accidental syndromes are irregular and rare, whereas significant syndromes with a common cause recur often. But this is not correct. Thus moles and benign cortical defects of bone are rather frequent combinations although they are accidental, whereas the association of polydactyly, encephalocele and polycystic kidneys is rare though they have a common cause when they are united in Meckel's syndrome.[21]

It would be best to speak of syndromes only if we have recognized their cause. However, we are not that cautious and patient and establish syndromes, although we do not know their origin. Thus Down's syndrome (mongolism) was a recognized syndrome for almost 100 years[7] before its dependence on trisomy 21 was discovered.[16] The "broad thumb" or Rubinstein-Taybi syndrome[22] is another example of an entity that is accepted, although we do not know its cause.

If associations of rather rare anomalies recur in families, we are inclined to accept them as syndromes and attribute them to *heredity*. In fact a few decades ago familial syndromes were always considered hereditary, since no other causes of multiple congenital malformations were known. If the syndrome was handed down from one generation to another, one considered it a dominant mendelian trait. If normal parents had more than one child with a syndrome of malformations, one suspected recessive mendelian inheritance. Such explanations were and still are given rather glibly and genetic counsel based on them is happily dispensed by newcomers in this field. I am more hesitant and less efficient as a genetic counselor, since I do not believe that repetition of syndromes always has such simple explanations. I shall cite some examples to illustrate that familial recurrence can have various and greatly heterogeneous causes. Several years ago we saw an infant with the rare *median facial cleft syndrome*.[31] This striking disease picture (Fig. 1*A*) is characterized by marked ocular hypertelorism, broad nasal root, cleft nose and lip and cranium bifidum occultum.[5] The anomaly had been observed years ago in sisters;[2] it could be thought to be a recessive hereditary trait. If the parents of such a child would have asked about the danger of recurrence, one would have had to say it is



Fig. 1. (*A* and *B*) Median facial cleft syndrome in half-sisters. (Reproduced with permission of the authors[32] and *Teratology*.)

25 per cent. Now let us assume that the parents would consider divorce after such a catastrophe and ask for counsel. It would seem that with different mates the risk of recurrence should be near zero, since it is most unlikely that either parent would encounter a new spouse who is a carrier of the same rare gene. But the mother of this child with median facial cleft syndrome, a young and phenotypically normal woman, had another girl with another husband and the second girl (Fig. 1*B*) had the same syndrome (further complicated by polydactyly). This was not a recessive trait. We can only speculate how this mother transmitted this rare syndrome to 2 daughters and we don't know how we could have predicted repetition in this case. A comparable situation was reported by Horne[14] in a couple that had 3 anencephalic children in consecutive pregnancies. The woman had a fourth anencephalic child after artificial insemination from a donor who had several normal children with other mates. Neither recessive inheritance nor empirical risk figures apply in such a situation. There are reports of recurrences in half-sibs with congenital malformations transmitted not only by mothers, but also by fathers with different spouses.[32]

Thus phenotypically normal parents can have more than one child affected by a syndrome due to unknown mechanisms which are revealed sometimes in half-sibs. However, such uncommon mechanisms can also be at work in full-sibs; the assumption of recessive inheritance may be wrong in such cases too, and genetic counsel could be based on an incorrect assumption. Observation and recording of unusual and unexplained situations are more important than emphasis on our present knowledge which is not too profound along these lines.

Various unknown causes may remain unexplored by assigning syndromes to simple or complex hereditary factors. I remember the time when a respected geneticist denied that advanced age of the mother at the time of conception had any significance in the etiology of mongolism and asserted that the defect is "inherited." Based on theoretical calculations it seemed reasonable to assume that the disorder is due "to the simultaneous presence in the germ cell of five pairs of recessive factors or two dominant and four pairs of recessive factors, carried in as many different chromosomes,"[17] or it was thought that a dominant factor with greatly reduced penetrance would be a valid explanation.[24] These examples show that one must not force unexplained phenomena into available concepts to provide hasty answers for which we are not ready. Erroneous ideas about Down's syndrome were not limited to geneticists. In 1958, one year before the discovery of trisomy 21,[18] I counted 40 theories concerning the origin of the enigmatic syndrome.[31] However, it should be pointed out that there were 3 physicians, an ophthalmologist[27] and two pediatricians,[1,10] who advanced the correct hypothesis of an extra chromosome in all the cells of patients with mongolism, at a time when the number of chromosomes in man was still uncertain.

The ability to visualize human *chromosomes*, to count them, and to distinguish them[26] became of signal importance to syndromology. Excess of chromosomal material, deficiencies and structural anomalies were found to be responsible for many syndromes. The more recent knowledge of banding chromosomes will further add to our knowledge of etiologies of multiple congenital malformations. Unpredictable advances in the laboratory may reveal causes not recognizable today.

The possibility of *environmental* or *exogenous factors* must always be kept in mind. Endemics and epidemics may help in the discovery of causes.

Figure 2*A* shows a syndrome characterized by mental retardation, deafness and dwarfism with abnormal x-ray findings of the skeleton[6,8] (Fig. 2*B*.) This disease picture has been often seen in 2 or more siblings, children of parents free of the signs mentioned. A diagram of the pedigree may suggest recessive inheritance, but the disorder is not a genetic trait;[9] it is due to

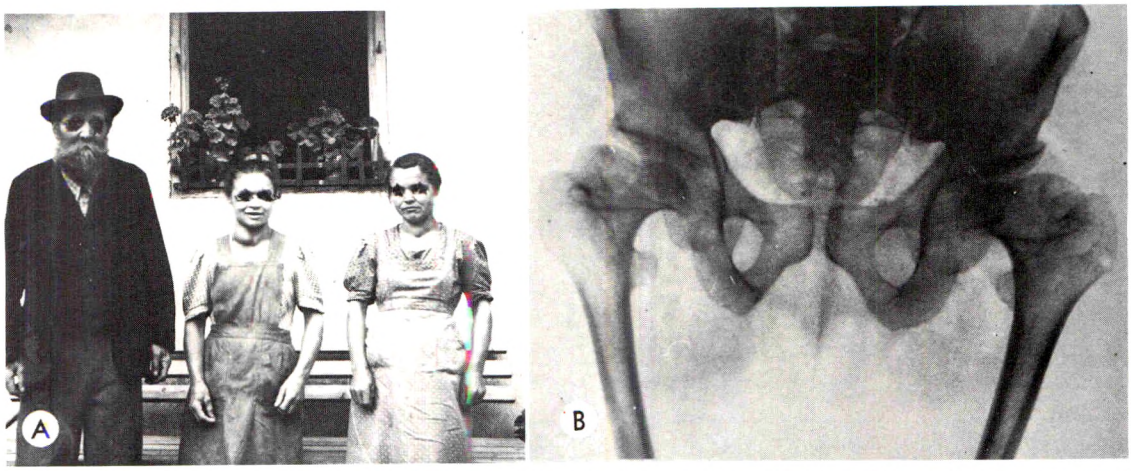


Fig. 2. (*A*) Dwarfed, deaf and goitrous sisters with endemic cretinism with their father who shows normal size. (*B*) Pelvis with hip joints of a 31 year old goitrous, endemic cretin. Coxa vara and subluxation of femoral head are seen. (Reproduced with permission of the authors[6] and Springer-Verlag, Berlin, 1936.)

iodine deficiency and the mother's abnormal thyroid. Such siblings are endemic cretins and victims of their environment. The syndrome is preventable by a simple dietary adjustment of future mothers.

Although none of you may have encountered an endemic cretin, it may be worthwhile to say a few words about this syndrome which was so prevalent in past centuries that in certain regions of the world at least one-half of the mentally retarded were handicapped by this condition. Mental retardation was equated with cretinism in the nineteenth century in many parts of Europe.[15] Since the endemic as a rule existed in remote mountainous regions where inbreeding and parental consanguinity prevailed, recessive inheritance was considered an etiologic factor by some authors. Fortunately, an association between endemic cretinism and endemic goiter of the parents and particularly the mothers was early recognized. When it was learned that endemic goiters could be prevented by dietary changes such as addition of iodine to salt, endemic goiter began to disappear and endemic cretinism went with it. However, it took more than a hundred years to transform the correct idea into effective prophylaxis. It was a fortunate coincidence that the responsible gland, the thyroid, was visible on the mother's neck; if a gland concealed in her body had mediated the disease, it could have taken much longer to recognize its etiologic role, and proof that endemic cretinism is not a recessively inherited trait might have been delayed.

There remains the question of why endemic goiter and cretinism and iodine deficiency are limited to certain locations on the earth. It seems that these locations correspond to areas covered by ice during the last (quaternary) glacial period. When the glaciers receded and these huge areas were freed from ice the eroded soil was deprived of iodine by the melting waters which carried iodine and other halogens into the oceans.[11,18,19] Those who settled in the iodine-poor valleys were afflicted by a deficiency disease in the presence of ample food. Thus the syndrome of endemic cretinism can be attributed to cosmic events that occurred millennia ago. I thought it worthwhile to discuss this fascinating and vanishing problem because it illustrates that damaging environmental factors can be much more remote than the much discussed exposures to drugs or rays during pregnancy; it shows that it may take many decades to unravel an environmental web of circumstances despite an endemic occurrence of a syndrome.

Epidemic appearance of syndromes also

can aid in recognition of their causes. The prenatal rubella syndrome was established because of the simultaneous occurrence of many cases of congenital cataract following an epidemic of rubella in Australia during World War II.[12] Subsequently other infectious diseases such as toxoplasmosis and cytomegalic inclusion disease were also shown to be teratogenic.

The ground for these discoveries had been prepared by experimental teratology which shows that almost any malformation known in man can be produced in animals by treatment of the pregnant female by radiation, nutritional deficiencies, hypoxia, toxic substances, hormones, drugs or other chemicals. Many experimental procedures result in syndromes of congenital malformations of the skeleton, of soft tissues or of both.

If a procedure is teratogenic in animals, there is great temptation to attribute similar malformations in man to the same cause. This of course, should not be done. Some experimental teratogenic conditions may never occur in man and an experiment may never be simulated by human conditions. In spite of their limitations such experiments are valuable and interesting, particularly if the patterns of syndromes produced resemble those of man. Let me illustrate this by two examples. One of the first dietary teratogenic experiments was effected in rats by riboflavin deficiency. This deficiency results in a skeletal disorder of great regularity.[28,30] In addition to cleft palate and mandibular hypoplasia, there occur a distal phocomelia and syndactylism with a definite pattern. Proximal bones are little affected, but those of the forearms and lower legs are shortened: The preaxial bones (radius and tibia) are always more abnormal than the postaxial, (ulna and fibula) (Fig. 3, *A–E*). This syndrome can be reproduced at will and, like syndromes in man, it shows variable expressivity: Some animals are more affected than others. To my knowledge there is no example of riboflavin deficiency of women that results in malformations of children which resemble the syndrome produced in rats. It is probably just a coincidence that an

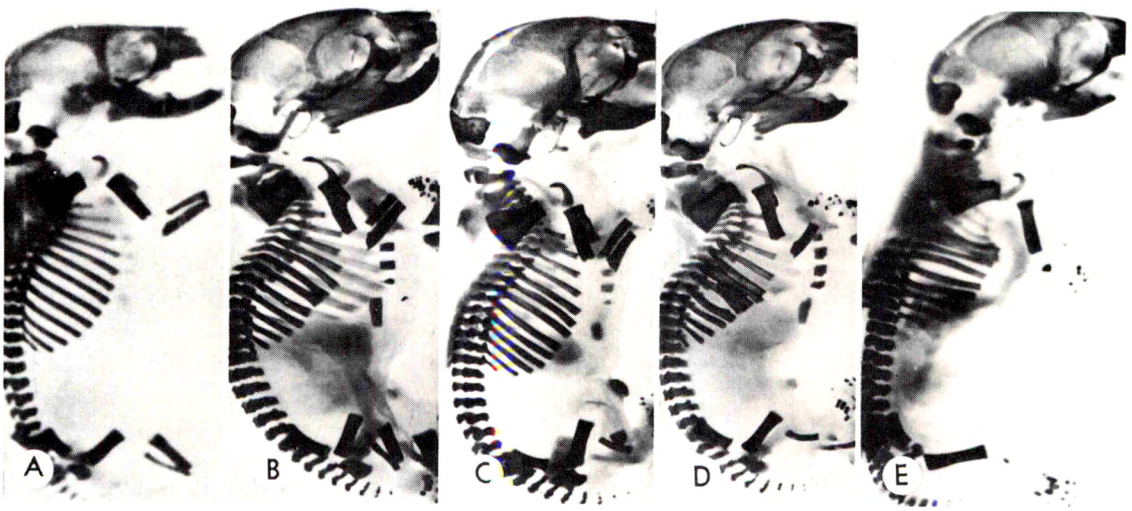


FIG. 3. Cleared specimens of newborn rats. (*A*) Control. (*B–E*) Offspring of mothers that were fed a riboflavin deficient diet before and during pregnancy.[28,30] The abnormal young show a pattern of skeletal malformations expressed in varying degrees. (*B*) Slight shortening of mandible and tibia. Two ribs are fused. (*C*) Marked reduction in size of tibia and some reduction of fibula. (*D*) Marked reduction of corpus mandibulae. Radius and tibia are not seen. Fusion and irregularities of 5 ribs. (*E*) Marked reduction of corpus mandibulae, ulna and fibula. Radius and fibula are not seen. Irregularities of ribs as in *D*. Note that the proximal long bones are little affected. Some of the absent bones are shown in histologic sections as irregular cartilaginous structures.

entirely different environmental interference with embryonic development caused—20 years after this experimental demonstration—a somewhat similar pattern of limb anomalies in man: Many of the children exposed to thalidomide in early embryonic life showed distal phocomelia with the tibia and fibula more involved than their postaxial correspondents. The existence of experimental mammalian teratology helped in the quick recognition of the thalidomide syndrome as an embryopathy of environmental causation.

Another example shows a direct relationship of experimental malformations to a human syndrome. Folic acid deficiency was thoroughly investigated as a teratogen in rats by the late Marjorie Nelson and her co-workers.[20] Dietary folic acid deficiency does not cause congenital malformations, since the deficiency can be compensated by folic acid production of the intestinal flora; but folic acid *antagonists* can induce a deficiency in man which is embryotoxic and teratogenic. Thus it was early recognized that aminopterin is a powerful abortive and it was used as such in illegal and therapeutic abortions. It was employed rather successfully by Thiersch[25] to achieve abortion in tuberculous women. However, when it failed in some cases, the attempt was followed by surgical removal of the embryos and in some of these exencephaly, hydrocephaly and cleft palate were noted. In a case reported by us[29] the drug failed to produce abortion. The pregnancy continued and toward the end of gestation roentgenograms of the fetus taken *in utero* failed to show bones of the calvarium. Anencephaly was suspected, but when the child was born—42 weeks after conception—the brain was well developed and covered by skin but the cranial bones were not ossified. In addition to this induced cranial dysostosis, the infant had a most unusual facial appearance (Fig. 4, *A* and *B*), posterior palate cleft and club feet. She died 24 hours after birth, but there are a few children on record who survived attempts to abort them by aminopterin and they have a rather characteristic appearance.[3,23] There exists a folic acid deficiency syndrome, a syndrome induced by a chemical. Theoretically this syndrome as well as

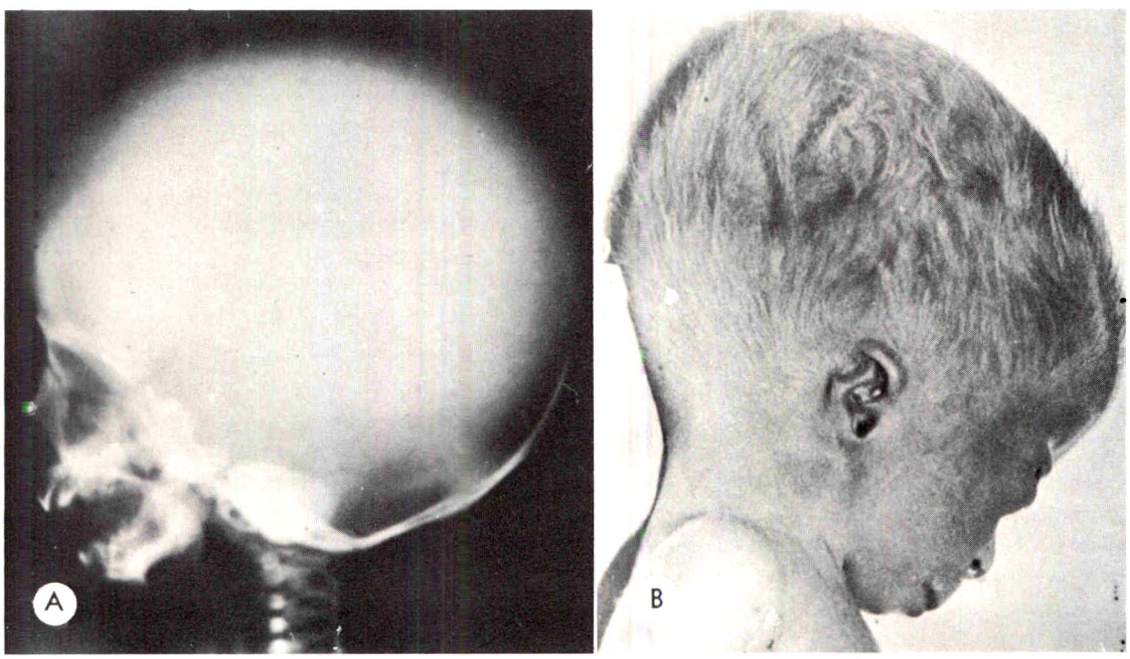


FIG. 4. Newborn child exposed to aminopterin in early gestation.[29] (*A*) Roentgenogram of skull showing lack of ossification of cranial membrane bones. (*B*) Deformed head and ears. Marked micrognathia.

the thalidomide embryopathy could recur in families if exposure to the drug were repeated.

Thus we have learned that genes, chromosomes and environmental factors can bring about multiple malformations and syndromes. And it seems likely that often combinations of these heterogeneous factors are responsible for the disastrous results encountered by us. Under certain relatively rare conditions, syndromes can recur in families. To consider only one strand of the causative web in such cases and to attribute the anomalies to a single damaging factor is not always advisable. Unitary explanations and tubular vision usually are characteristic of newcomers in the field of congenital malformations.

The roentgenologist is at a disadvantage in etiologic investigations because he is not often in direct contact with the patient and his family. He sees the results, but generally he is removed from etiologic information. He has advantages too; because of his central position so many cases are channeled through radiology departments that he sees more of them and of the anomalies than other pediatricians. He may notice differences and new dimensions of malformations that seem uniform and equal to the bedside observer. He may recognize connections and similarities which escape external inspection.

There is now much hope—and seemingly too much hope—for prevention of congenital malformations by genetic counseling. Genetic counseling is an application of etiologic classification and knowledge. The role of the pediatric roentgenologist in this endeavor cannot be overrated or overemphasized. Genetic or parental counseling is not possible without a firm diagnosis. This may sound like a truism, but it should be realized that in the field of congenital malformations advice is often requested and given for disorders that are insufficiently diagnosed and understood. To illustrate the importance of roentgenology one can cite the chondrodystrophies and osseous dystrophies which had long been classified by clinicians and pathologists. With the help of x rays they have been reclassified and recognized as many heterogeneous entities which often differ in etiology. Whereas in the past pathologists reigned supreme and dominated the field, now roentgenologists often have the final say in diagnosis and indirectly in genetic counseling. Consequently they carry also great responsibilities, since family planning may depend on their findings. It may be good to avoid dogmatic statements beyond description of the changes actually seen in the roentgenograms. X rays do not reveal the chemical anomalies of the tissues, the family histories nor the clinical observations which must be considered in the classification of a case and in the advice given to the parents. The roentgenologist's view and point of view is important, but it is not the only one. It is clear that roentgenology can make its best contributions to syndromology by cooperation with other observers and that those who establish good communication and cooperation with other specialists are likely to contribute most to the solutions of the problems faced by all of us.

SUMMARY

Syndromes of congenital malformations are interesting and challenging phenomena which deserve observation, description and treatment of the children and adults who are afflicted by these multiple birth defects. However, the highest level of syndrome research is etiologic research, because knowledge of the causes of multiple congenital malformations promises prevention. Although naming syndromes and classifications are important auxiliary activities, they must not be our final goal. The infinite number of combinations of congenital defects suggests that terminology and classification should be tentative and flexible and that rigidity be avoided. The same holds for statements of causation. I tried to make it clear that we are far from a full understanding of all the principles involved in the etiology of malformations. Many unexpected discoveries along these lines have

been made during my lifetime and many of those who pretended to have the final answers were shown to be wrong. It was particularly distressing to see legislation based on insufficient knowledge which resulted in sterilization and even death of handicapped persons.[13] There has been progress and fortunately there has been recognition of our ignorance. However there is still too much happiness about, and promotion of, genetic counseling which is often inadequate and not based on solid knowledge. I refer to the enthusiasm of newcomers who believe that etiologies of congenital malformations and syndromes are always clear-cut, easily identifiable and applicable to all cases with similar signs and symptoms. Experienced medical geneticists will agree with my caution, since they are fully aware of the gaps in our knowledge of the causes of congenital malformations.

My attitude is by no means pessimistic or nihilistic. Attention should be paid to all details that surround the origin of syndromes and prevention should become possible by recognition and elimination of causative factors. The senior members of this Society will remember diseases which have been wiped out because their causes were recognized and removed. Similar methods can be applied to prevention of multiple congenital malformations. In the meantime our guidelines should be collecting of facts and delay of conclusions. In medicine no disease problem is solved until the disorder disappears and becomes history.

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REFERENCES

1. Bleyer, A. S. Indications that mongoloid imbecility is gametic mutation of d·gressive type. *Am. J. Dis. Child.*, 1934, *47*, 342–348.
2. Bougon and Derocque. Fissure médiane de la face. *Rev. orthop.*, 1908, *9*, 219–224.
3. Brandner, M., and Nusslé, D. Foetopathie due à l'aminoptérine avec sténose congénitale de l'espace médullaire des os tubulaires longs. *Ann. de radiol.*, 1969, *12*, 703–710.
4. Caffey, J. First sixty years of pediatric roentgenology in United States-1896 to 1956. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 437–454.
5. De Myer, W. Median cleft face syndrome: differential diagnosis of cranium bifidum occultum, hypertelorism and median cleft nose, lip, and palate. *Neurology*, 1967, *17*, 961–971.
6. De Quervain, F., and Wegelin, C. Der endemische Kretinismus. Springer-Verlag, Berlin, 1936.
7. Down, J. L. H. Observations on ethnic classification of idiots. *Clin. Lect. & Rep. London Hospital*, 1866, *3*, 259–262.
8. Eggenberger, H. Kropf und Kretinismus. Handbuch der inneren Sekretion. Leipzig, Kabitzsch., 1928.
9. Eugster, J. Zur Erblichkeitsfrage des endemischen Kretinismus-Untersuchungen an 204 Kretinen und deren Blutsverwandten. *Arch. der Julius Klaus-Stiftung für Vererbungsforschung, Sozialantropologie und Rassenhygiene*, 1938, *13*, 383–494.
10. Fanconi, G. Die Mutationstheorie des Mongolismus. *Schweiz. med. Wchnschr.*, 1939, *69*, 81–86.
11. Goldschmidt, V. M. Geochemistry. Edited by A. Muir. Clarendon Press, Oxford, 1954.
12. Gregg, N. M. Congenital cataract following German measles in mother. *Tr. Ophth. Soc. Australia*, 1942, *3*, 35–46.
13. Gütt, A., Rüdin, E., and Ruttke, F. Gesetz zur Verhütung erbkranken Nachwuchses vom 14. Juli, 1933. J. F. Lehman, München, 1934.
14. Horne, H. W. Anencephaly in four consecutive pregnancies. *Fertil. & Steril.*, 1958, *9*, 67–68.
15. Kanner, L. A History of the Care and Study of Mentally Retarded. Charles C Thomas, Publisher, Springfield, Ill., 1967.
16. Lejeune, J., Gautier, M., and Turpin, R. Étude des chromosomes somatiques de neuf enfants mongoliens. *C. R. Acad. Sc.* 1959, *248*, 1721–1722.
17. Macklin, M. T. Mongolian idiocy: manner of its inheritance. *Am. J. M. Sc.*, 1929, *178*, 315–337.
18. Marine, D. On occurrence and physiological nature of glandular hyperplasia of thyroid (dog and sheep) together with remarks on important clinical (human) problems. *Johns Hopkins Hosp. Bull.*, 1907, *18*, 359–364.
19. Merke, F. Die Eiszeit als primordiale Ursache des endemischen Kropfes. *Schweiz. med. Wchnschr.*, 1965, *95*, 1183–1192.
20. Nelson, M. M., Asling, C. W., and Evans, H. M. Production of multiple congenital abnormalities in young by maternal pteroylglutamic acid deficiency during gestation. *J. Nutrition*, 1952, *48*, 61–80.
21. Opitz, J. M., and Howe, J. J. Meckel syndrome

(dysencephalia splanchnocystica, Gruber syndrome). In: Birth Defects: Original Article Series. Volume V, Part II, 1969, pp. 167–178.
22. Rubinstein, J. H., and Taybi, H. Broad thumbs and toes and facial abnormalities. *A.M.A. Am. J. Dis. Child.*, 1963, *105*, 588–608.
23. Shaw, E. B. Fetal damage due to maternal aminopterin ingestion. *A.M.A. Am. J. Dis. Child.*, 1972, *124*, 93–94.
24. Snyder, L. H. Medical Genetics. Duke University Press, Durham, N. C. 1941.
25. Thiersch, J. B. Control of reproduction in rats with aid of antimetabolites and early experiences with antimetabolites as abortifacient agents in man. *Acta endocrinol.*, 1956, *28*, 37–45.
26. Tjio, J. H., and Levan, A. Chromosome number of man. *Hereditas*, 1956, *42*, 1–6.
27. Waardenburg, P. J. Das Menschliche Auge und Seine Erbanlagen. Martinus Nijhoff, The Hague, 1932.
28. Warkany, J., and Nelson, R. C. Skeletal abnormalities in offspring of rats reared on deficient diets. *Anat. Rec.*, 1941, *79*, 83–100.
29. Warkany, J., Beaudry, P. H., and Hornstein, S. Attempted abortion with aminopterin (4-amino-pteroylglutamic acid). *A.M.A. Am. J. Dis. Child.*, 1959, *97*, 274–281.
30. Warkany, J. Experimental production of mammalian limb malformations. Edited by C. A. Swinyard. In: Limb Development and Deformity. Charles C Thomas, Springfield, Ill., 1969.
31. Warkany, J. Congenital Malformations. Notes and Comments. Year Book Medical Publishers, Inc., Chicago, Ill., 1971.
32. Warkany, J., Bofinger, M. K., and Benton, C. Median facial cleft syndrome in half-sisters: dilemmas in genetic counseling. *Teratology*, 1973, *8*, 273–285.

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TAKAYASU'S ARTERITIS*

AN ANGIOGRAPHIC STUDY WITH REMARKS ON ETHNIC DISTRIBUTION IN ISRAEL

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TAKAYASU's arteritis is a diffuse nonspecific arteritis of unknown etiology usually affecting young women. It may involve the aorta as well as any of the larger elastic arteries, including the pulmonary arteries, and may progress to stenosis, occlusion, or aneurysmal dilatation of the affected vessels. Symptoms are protean and may precede clinical manifestations of vascular involvement. The first reported cases were apparently seen in the XIX century.[2,10,38] Twentieth century interest was stimulated by a Tokyo ophthalmologist, Migito Takayasu, who described a case of advanced chronic ischemic retinopathy in a young woman before the Twelfth Congress of the Japanese Ophthalmological Society in 1908.[20] Although Takayasu himself did not mention any additional vascular abnormalities, in the discussion that followed Takayasu's report, 2 other ophthalmologists, Onishi and Kagoshima,[20] described their patients, who had similar ocular findings together with absence of the radial pulse. In the following years the disease was recognized as a clinical entity and became known under the name of Takayasu's disease,[32] pulseless disease[45] and later as aortitis syndrome.[50] Thereafter, as the disease was appreciated in other countries, it was described under a large number of eponyms or descriptive names excellently traced by Gotsman *et al.*[13]

Although initial reports stressed that this is a disease of the aortic arch, subsequent reports of involvement of the abdominal aorta and pulmonary arteries suggested a more generalized process. The similarity in the histopathologic findings from those cases of aortitis affecting the thoracic aorta, particularly the arch and its branches, and the aortitis which causes coarctation and aneurysms of the lower thoracic and abdominal aorta and its branches led to the unifying concept that these conditions are various manifestations of the same disease.[19] The aortic arch and brachiocephalic form seem more common in Japan, while the abdominal form is more prevalent in Africa.[33,48]

The distribution of case reports of Takayasu's arteritis may only reflect the clinical interests and enlightenment of the reporting physicians. Nevertheless, until the mid-fifties and even into the sixties, the vast majority of cases were reported from Oriental countries: Japan, Singapore, Korea, China, India, Malaysia and the Philippines, or from the "coloured" and Bantu peoples of South Africa.[1,7,9,11,13,17–19,21,22,28,33,40,44,51] In contrast, the disease was uncommon among the white population of South Africa.[42] Large series reported from the United States and European countries are relatively infrequent[3–6,14,20,29,34,35] and when the ethnic origins of the patients from the United States are stated, it appears to affect the Black, Oriental, and Spanish (Mexican, Puerto Rican, Central or South American) groups to a greater extent than those of Northern European origin.[18,20,29,30]

In reviewing the angiograms and the clinical files of 22 cases of Takayasu's arteritis investigated in the last 12 years at the Chaim Sheba Medical Center in Israel, we found an unexpected distribution among the various ethnic groups comprising the Israeli population—observations which

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may contribute to an understanding of the geographic incidence of the disease and the variations in its anatomic distribution.

ETHNIC BACKGROUND

In order to clarify the ethnic origins of the various groups forming the contemporary Jewish community of Israel, a short historic background is necessary.[15,36,37,52] Jewish history in the lands comprising the present State of Israel dates to about 1200 B.C. From that historic time there were 3 major migrations which eventually led to the formation of large Jewish communities in the Diaspora, outside the land of Israel.

The first of these migrations, known as the Babylonian captivity, took place in 586 B.C. Seventy years later, a large group of these exiled Jews were allowed to return to the Holy Land, but the rest remained and eventually spread to nearby lands. The present Iraqi, Kurdish and Persian Jews are, to a large extent, descendants of this original community of exiled Babylonian Jews.

The second migration from ancient Israel occurred during the period following the conquests of Alexander the Great. The spread of Greek culture induced large groups of Jews to settle in North Africa, Syria, the Balkans and Asia Minor, where their descendants have continued to live until the present time.

The third large migration took place during the Roman period when Jews emigrated primarily to Spain and Germany. The 2 groups, for the most part, remained quite separate, establishing different traditions and customs. The descendants of these original German Jews populated Eastern Europe and are known as Ashkenazim from the Hebrew word for Germany. The Ashkenazim form the majority of the present day Jewish communities of North and South America, England and South Africa. The Spanish Jews, called Sepharadim from the Hebrew word for Spain, were largely expelled from Spain in 1492 at the time of the Inquisition. The majority settled in what was then the Turkish Empire. Their descendants can be found in the Jewish communities of Greece, Turkey, Bulgaria and other Mediterranean countries, as well as in North Africa, where they mingled with Jews who had arrived earlier. Today the differences between the Ashkenazim and Sepharadim include not only tradition, language and ritual, but also physical appearance, suggesting that intermarriage and conversion have contributed to these ethnic characteristics.

Small relatively isolated Jewish communities also existed in Yemen, Ethiopia and India. The Yemenite Jews are believed to have originally settled in Yemen during the period of the Second Temple before the Roman Era. According to Arab writers many indigenous tribes of Yemen embraced Judaism prior to the spread of Islam. The descendants of both groups have lived in Yemen, at the southern tip of the Arabian desert, continuously since that time.

The Ethiopian Jews, or Falashas, are believed to be descendants of Jews who lived in Ethiopia from ancient times and intermarried with local proselytes whom they introduced to the Jewish religion. Others are inclined to the theory that they originated entirely from local tribes which had been converted to Judaism.

The origin and dates of the arrival of Jews into India is controversial. According to their own tradition, they are descendants from one of the Ten Lost Tribes of Israel. Some of them are known to have arrived as refugees from Spain, while others emigrated from Iraq and Yemen. However, a dark skinned group, known as the Black Jews of India, at least partly has to be considered as offsprings of converted local inhabitants.

The contemporary Jewish community of Israel is composed of approximately equal numbers of Ashkenazim and Sepharadim, 5 per cent Yemenite and less than 0.5 per cent Ethiopian and Indian Jews.

MATERIAL AND METHOD

Twenty-two patients with Takayasu's

arteritis were investigated in the Division of Cardiovascular Radiology of the Chaim Sheba Medical Center during the period from 1965 to 1973. The medical center contains one of the largest hospitals (1,100 beds) in Israel and serves as a referral center for all parts of the country. The patient population is almost half Ashkenazim and half non-Ashkenazim, corresponding approximately to the proportions of these groups in the country as a whole.[47]

The age, sex and ethnic origin of the patients are summarized in Table 1. All patients were under the age of 40 years, except for 2, in whom the diagnosis was proven at autopsy. Three other patients also had postmortem confirmation of diffuse arteritis of the Takayasu type. Otherwise, the diagnosis of Takayasu's arteritis was based mainly on the results of angiographic studies supported by the clinical and laboratory findings. There were 12 females and 10 males, a ratio which does not conform to the 8:1 female-to-male ratio usually stated for this disease. Nine of the 22 patients were Arab or Beduin, while the remainder were Jewish. None of the 13 Jewish patients were Ashkenazim.

TABLE 1

ETHNIC DISTRIBUTION OF CASES

Case No.	Age	Sex	Origin*
1	18	F	India
2	22	F	India
3	12	F	India
4	22	M	India
5	40	M	Beduin
6	40	M	Beduin
7	23	F	Beduin
8	27	F	Beduin
9	40	M	Beduin
10	40	M	Arab
11	24	F	Arab
12	36	M	Arab
13	27	M	Arab
14	42	F	Bulgaria
15	30	F	Iran
16	26	M	Ethiopia
17	45	M	Greece
18	36	F	Iraq
19	24	M	Yemen
20	26	F	Syria
21	21	F	Tunisia
22	38	F	Turkey

* All patients, except 5-13, were Jewish. See text for discussion of ethnic background.

Laboratory tests included complete blood cell count, erythrocyte sedimentation rate, serum protein electrophoresis, Wassermann serologic test for syphilis, antinuclear factor, and tuberculin skin test. Plain chest and abdominal roentgenograms were obtained in all 22 cases. Aortography of the entire aorta and its major branches was performed in 18 cases, thoracic and abdominal aortography in 3, and abdominal aortography alone in 1. In addition, the peripheral arteries of the lower and/or upper extremities were visualized in 13 cases. Pulmonary angiography was performed in 5 cases. Repeat aortography was obtained in 6 cases at intervals from 2 to 5 years. Additionally, 1 patient had repeat examinations at 2 and 5 years. Aortography was performed by the percutaneous method through the femoral artery in 17 cases, by the axillary route in 4, and by direct lumbar aortography in 1.

RESULTS

A. CLINICAL AND LABORATORY EXAMINATIONS

The clinical and laboratory findings in our 22 patients, in general, corresponded to those described in the literature. Constitutional symptoms found during the early acute stage of the illness were present in 3 cases in the form of fever or night sweats. Other symptoms were referable to the areas of vascular involvement. Twenty-five per cent exhibited cerebral signs or symptoms including cerebral hemorrhage, epileptic attacks, syncope, vertigo and visual disturbances. Absence or weakness of peripheral pulses were present in 72 per cent of cases and hypertension in 54 per cent. Aortic insufficiency, pericarditis and abdominal angina were present in 1 patient each. Seven patients suffered from left heart failure.

Laboratory investigations disclosed elevated erythrocyte sedimentation rate (ESR)

in 13 of 19 patients studied. Leukocytosis greater than 10,000 per mm.^{3} was present in 6 of 18 patients. A positive tuberculin test was found in 4 of 10 patients. The Wassermann test was negative in all 8 patients in whom it was performed, and the antinuclear factor was negative in 4 out of 5 patients.

B. PLAIN FILM CHEST ROENTGENOGRAPHY

The plain film chest roentgenograms showed mild to moderate dilatation of the thoracic aorta and cardiomegaly in half of the patients. Localized aneurysmal dilatation of the thoracic aorta was noted in 3 patients (Fig. 1*A*). In 1 of them a thin laminar calcification was present in the wall of a tubular aneurysm (Fig. 2, *A* and *B*). Rib notching was not observed. Narrowing of the pulmonary vessels in one of the upper lung fields (Fig. 2*A*) was evident in 2 cases. Tuberculous lesions of the lung or pleura were not observed. In 1 case with apparent cardiomegaly on plain film roentgenogram, pericardial disease was diagnosed by angiography and confirmed at operation.

C. PLAIN FILM ABDOMINAL ROENTGENOGRAPHY

The plain film abdominal roentgenograms did not show calcification of the abdominal aortic wall in any case. Significant differences in the size of the kidneys were observed in 4 cases with hypertension, while bilaterally small kidneys were seen in another 3. These findings were due to renal artery stenosis or occlusion.

D. ANGIOGRAPHY

The aortographic abnormalities were variable and often bizarre in appearance. Various forms of narrowing, occlusion, dilatation or a combination of these were observed. In this series, generally, stenotic lesions were more common than dilatation or aneurysm formation. This may be related to regional or ethnic differences in the way the disease is manifested or to the stage of pathologic development at the time of presentation. Stenotic lesions may progress to occlusion and relatively uninvolved segments may become aneurysmal. This latter point is well demonstrated by one of our patients who was investigated 3 times during a period of 5 years (Fig. 3, *A–D*).

1. *Heart and Aortic Arch.* In the ascending aorta mild to moderate dilatation was encountered in 7 patients and marked dilatation in 1. Aortic insufficiency was demonstrated in only 1 patient. The coronary arteries could be evaluated from the thoracic aortograms in 5 patients and no pathologic changes were detected. One patient had an effusive constrictive pericarditis.

Although the disease may involve all of the brachiocephalic vessels, in our material the subclavian arteries were the most commonly involved (Fig. 5; 6; and 7). Pathologic changes were observed in the subclavian arteries of 11 patients: 7 of them bilateral, while the other 4 had involvement only of the left. These lesions were obstructive in all cases except 1 in whom aneurysms were present. Complete occlusion or stenosis occurred just beyond the thyrocervical trunk in most cases (Fig. 6, *A–C*). In 3 patients the occlusion or stenosis involved the proximal left subclavian artery, but even then, the most proximal 2–3 cm. of the subclavian artery was patent (Fig. 7*B*). In 2 cases (Fig. 6, *A* and *D*) the common carotid arteries were markedly stenosed and the vertebral arteries hypertrophic. Three patients had axillary artery involvement, in each case on the right side (Fig. 7*A*).

2. *Descending Thoracic Aorta.* A lesion of the thoracic aorta was encountered in 11 of 21 patients in whom this segment of the aorta was visualized. The lesion was stenotic in 4 (Fig. 8) and aneurysmatic in 7 (Fig. 1; 2; and 5). It should be stressed that all 4 patients originating from India had aneurysmal changes of the thoracic aorta.

3. *Abdominal Aorta.* The abdominal aorta was involved in 14 cases (Fig. 3; 4; and 9). Stenotic or obstructive lesions were present in 9 and aneurysms in the rest. The aneurysmal lesions varied in shape and size, were either diffuse or localized (Fig.

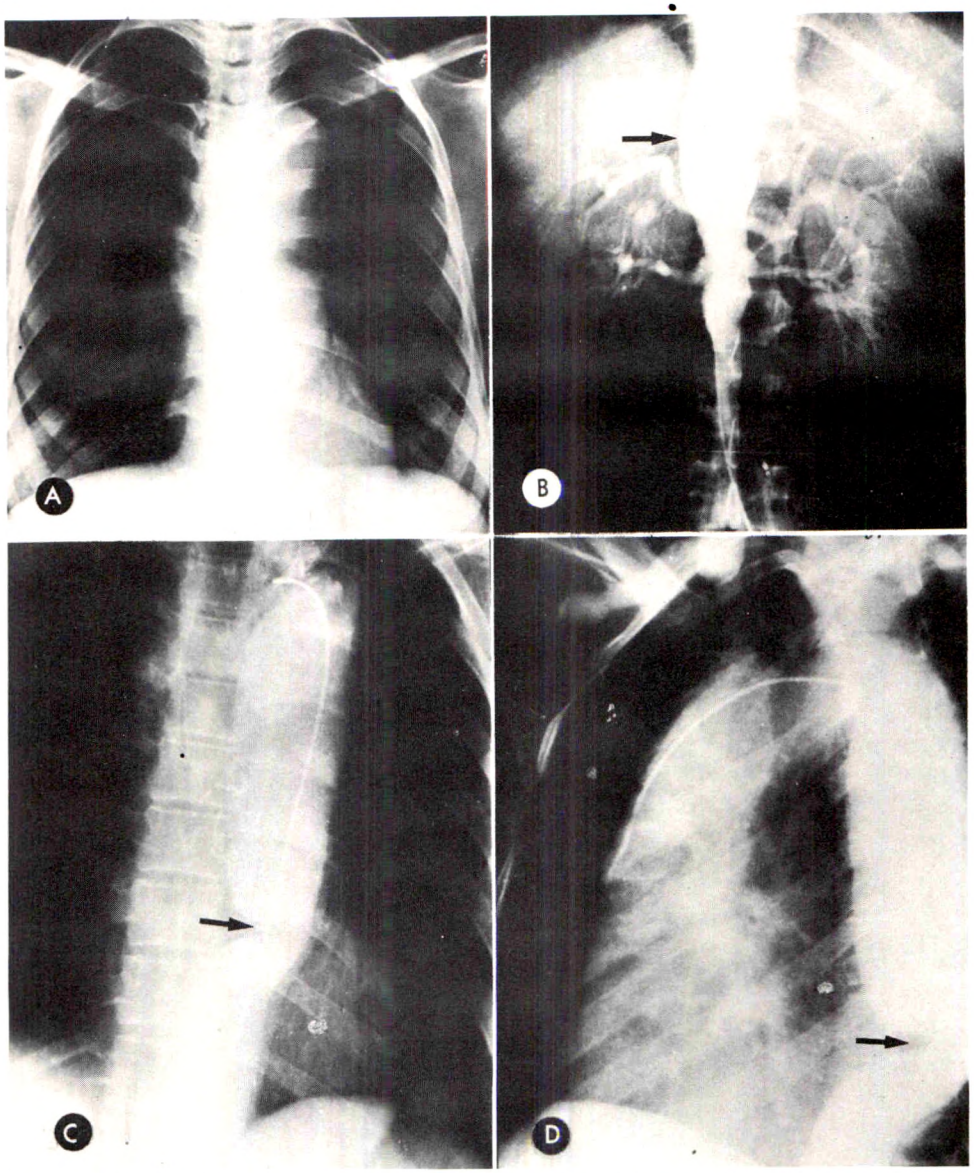


FIG. 1. Case 21. (*A*) Roentgenogram of the chest, overpenetrated frontal view. Dilatation of the thoracic aorta, apparently extending to T-10. (*B*) Abdominal aortogram. Aneurysmal dilatation of the thoracic aorta (arrow) tapering to a more normal diameter, then dilating again below the renal axis. There is a gradual tapering of the lower aneurysmal segment into a normal distal aorta. (*C* and *D*) Frontal and right posterior oblique (RPO) views of thoracic aortograms. The entire thoracic aorta is aneurysmal with characteristic kinking of the lower thoracic segment (arrows). Note the smooth luminal surface and only slight thickening of the aortic wall.

3; and 5), saccular or fusiform. Multiple aneurysms in the same case were also observed. Complete occlusion of the abdominal aorta occurred at all levels, from above the origin of the celiac trunk to below the inferior mesenteric artery (Fig. 4; and 9). It is remarkable that 3 out of 5 Beduin patients and 3 out of 4 Arab patients had stenosis or complete occlusion of the abdominal aorta.

4. *Visceral Branches of the Abdominal Aorta.* The celiac artery was occluded at its origin in 3 patients (Fig. 10) and stenosed in another 3. Complete occlusion of the

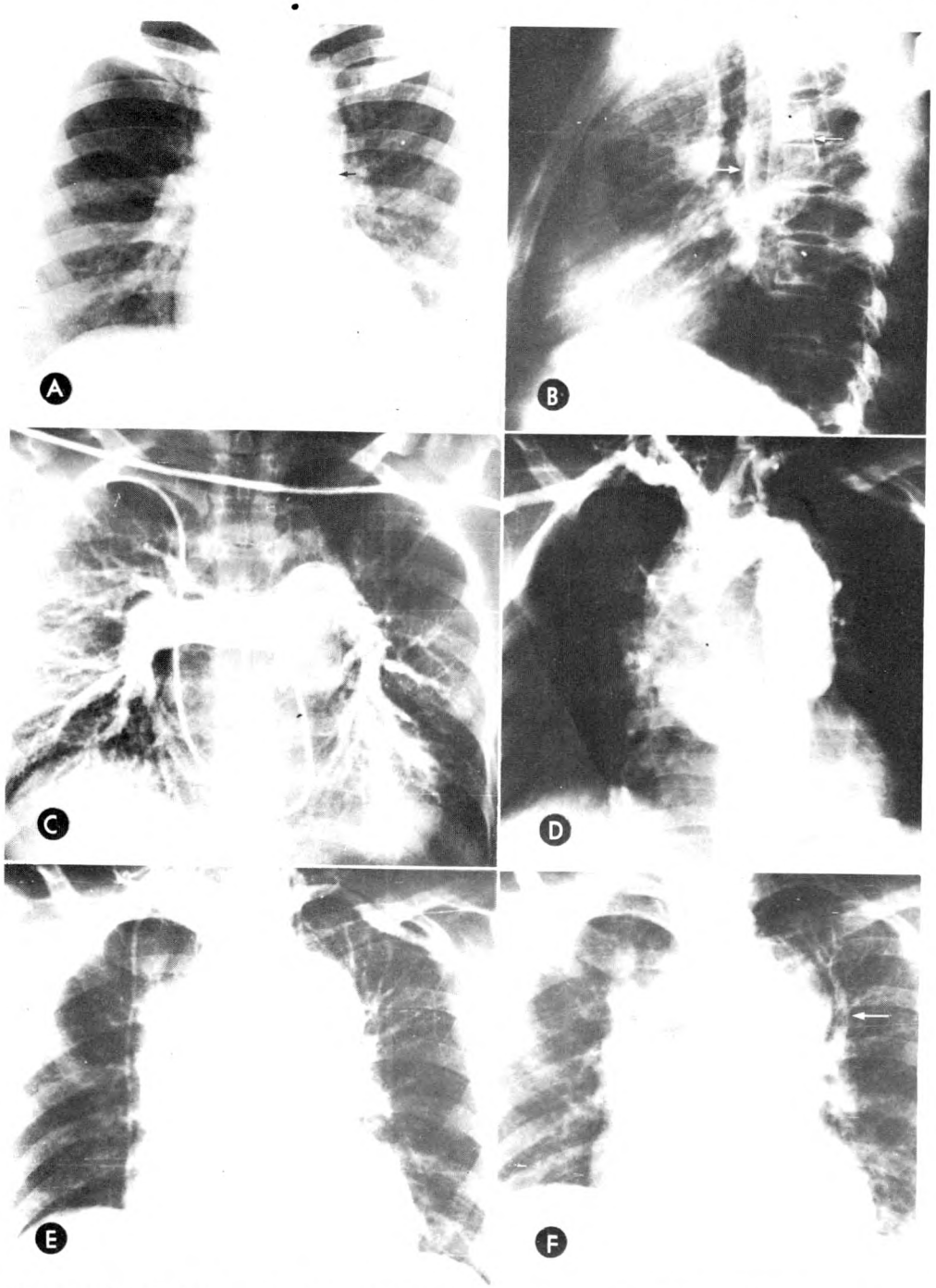


FIG. 2. Case 2. (*A* and *B*) Frontal and lateral chest roentgenograms. A calcified aneurysm is visible in the upper descending aorta (arrows). (*C*) Pulmonary angiogram. There is diminished flow to the left upper lobe and narrowing of the proximal portions of the segmental pulmonary arteries to the right upper lobe. (*D*) Thoracic aortogram. There is a saccular aneurysm of the upper thoracic aorta with irregular walls. The proximal portion of the left subclavian artery is narrowed. (*E*) Later phase of thoracic aortogram, intentionally underpenetrated to show the pulmonary vessels. Large bronchial arteries communicate with the pulmonary arteries and supply the left upper lobe. (*F*) Still later a large pulmonary vein (arrow) draining the left upper lobe indicates that this lobe receives its primary supply *via* bronchial arteries.

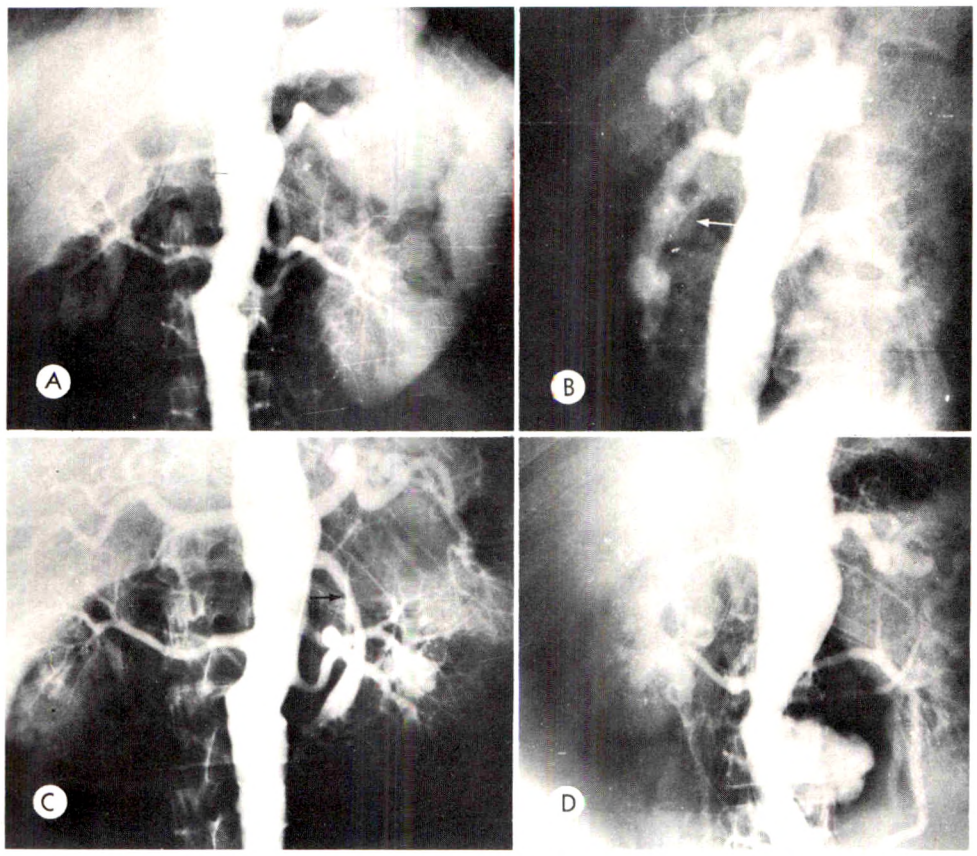


Fig. 3. Case 19. (*A* and *B*) Frontal and lateral views of initial abdominal aortogram. There are irregular aneurysmal changes in the abdominal aorta. The superior mesenteric artery is also aneurysmal, seen best in the lateral view (arrow). (*C*) Two years later, the aneurysmal changes in the abdominal aorta are more prominent. A long segment of the superior mesenteric artery has become narrowed (arrow); but the more distal segment is still dilated. (*D*) Five years after initial study. The upper segment aortic aneurysm is still present. A stenosis has developed below the renal arteries and a large aneurysm has appeared below this narrow segment. The superior mesenteric artery is now totally occluded. Collateral supply from the inferior mesenteric artery is visible.

superior mesenteric artery was present in 4 and stenosis in 2 (Fig. 8). In 1 of the cases with occlusion (Fig. 3) the superior mesenteric artery initially showed aneurysmal dilatations, but on later examination it was completely occluded. In all 4 cases with occlusion of the superior mesenteric artery, stenosis or occlusion was also present in the celiac artery.

The inferior mesenteric artery was occluded in 4 patients (Fig. 9). In all cases with visceral artery involvement prominent collateral vessels compensated for the circulation either from the inferior mesenteric to the superior mesenteric and celiac arteries or from the superior to the inferior mesenteric artery.

Two of the cases with visceral artery involvement were exceptions: 1 showed localized aneurysmal dilatation of the main hepatic artery (Fig. 8), and the other stenosis of the main hepatic artery (Fig. 10).

5. *Renal Arteries.* Renal artery stenosis or occlusion was present in 13 patients (Fig. 3; 4; 8; and 9). There was bilateral involvement in all except 3. Among the cases with bilateral lesions, unilateral complete occlusion was present in 4. Renal artery stenosis manifested itself as a short segmental constriction of the ostium, with

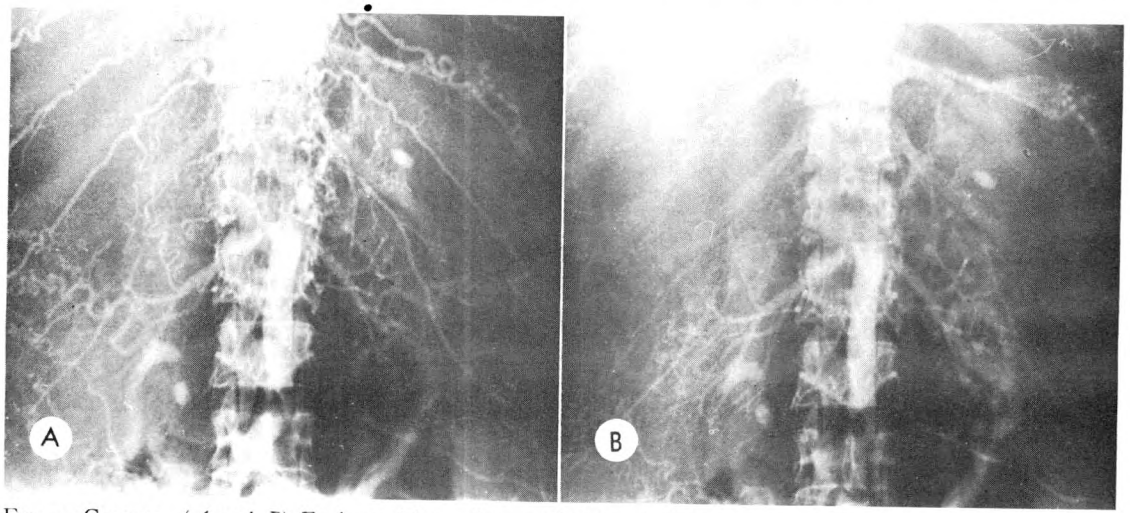


FIG. 4. Case 11. (*A* and *B*) Early and late phases of abdominal aortogram. Injection into thoracic aorta. There is total interruption from above the origin of the celiac artery to above the renal arteries. The entire distal aorta, renal and inferior mesenteric arteries fill *via* intercostal collaterals. The celiac and superior mesenteric arteries are filled in turn, *via* collaterals from the inferior mesenteric artery.

or without poststenotic dilatation, or as an elongated narrowing throughout the entire length of the main renal artery.

6. *Iliac and Lower Extremity Arteries.* In the cases with complete occlusion of the distal aorta, the proximal segment of the common iliac arteries was also occluded. However, the distal segments of the com-

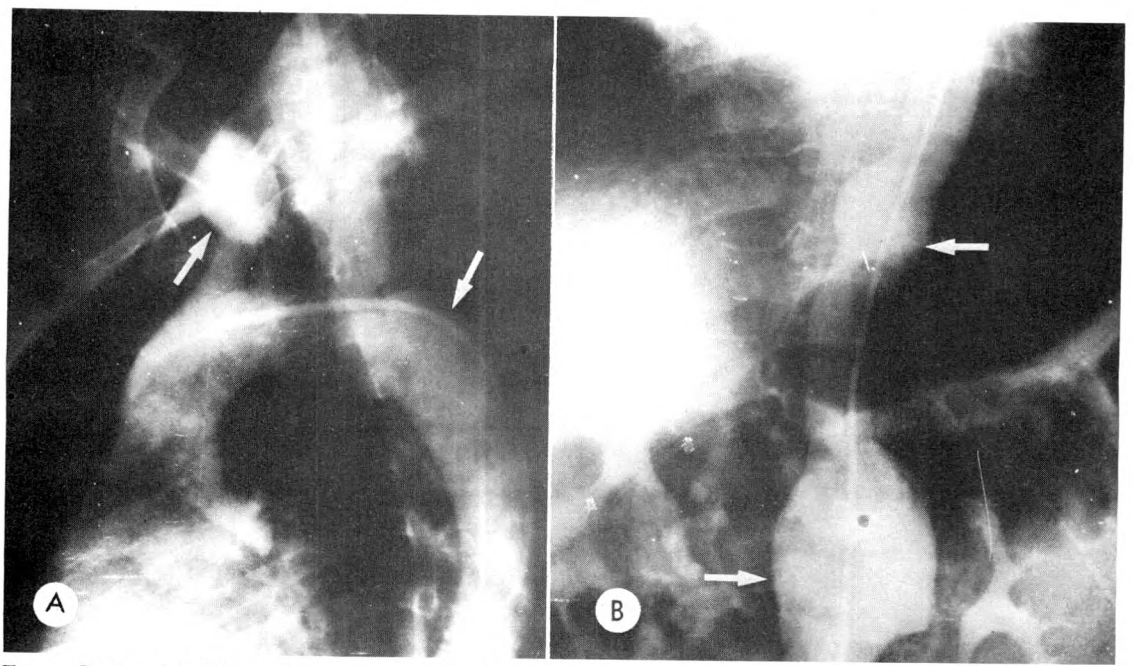


FIG. 5. Case 4. (*A*) Thoracic aortogram, RPO view. Aneurysm of the innominate artery (right arrow) and the thoracic aorta just distal to the left subclavian origin (left arrow). Smooth luminal surfaces with tapering margins of the aneurysms are noteworthy. (*B*) Thoraco-abdominal aortogram, frontal view. There are localized aneurysms of the lower thoracic and mid abdominal aorta (arrows) with intervening skip areas free of disease.

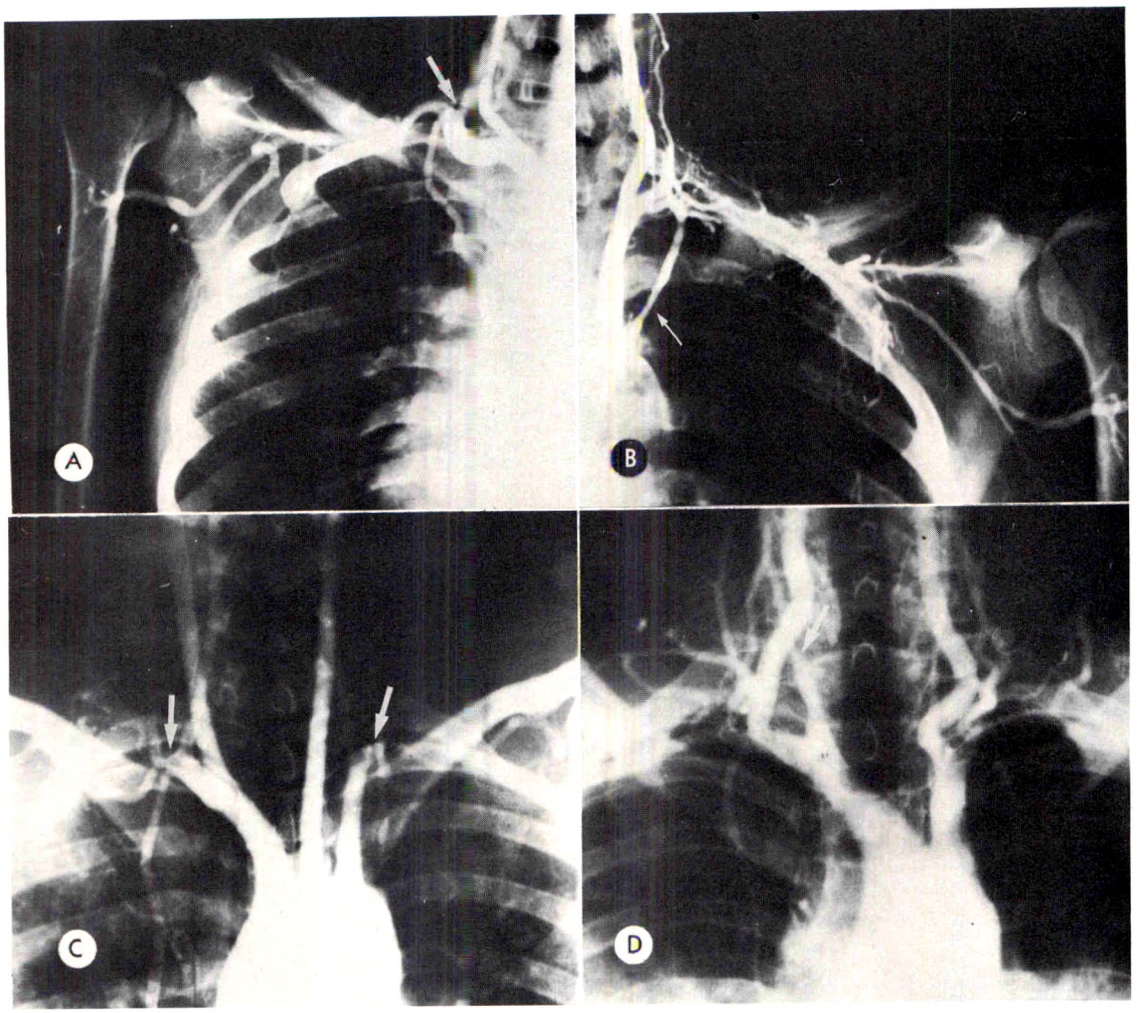


FIG. 6. Case 7. (*A*) Selective innominate arteriogram. There is diffuse narrowing of the right common carotid artery and hypertrophy of the right vertebral artery. The right subclavian artery is totally occluded beyond origin of the vertebral artery (arrow). (*B*) Left subclavian arteriogram. The left vertebral artery is hypertrophied. The subclavian artery is totally occluded distal to origin of the internal mammary artery. Segmented narrowing of the mammary artery (arrow) was an unusual finding. (*C*) Case 15. Bilateral subclavian artery occlusion distal to origins of vertebral arteries (arrows). (*D*) Case 1. There is stenosis of the right common carotid artery (arrow) and diffuse narrowing of the left common carotid artery with marked hypertrophy of both vertebral arteries and cervical branches of the thyrocervical trunks.

mon iliac, the external iliac and the hypogastric arteries were patent and were visualized *via* collaterals. In 2 cases in whom the inferior mesenteric artery was not involved, it served as the main pathway for collateral circulation to the iliac arteries and the arteries of the distal extremities. The arteries of the lower extremity were partially or completely visualized in 7 cases. In all of the cases the main arteries of the lower extremity were free of atheromatous changes or occlusion.

7. *Pulmonary and Bronchial Arteries.* Pulmonary angiography was performed in 5 patients (Fig. 2; and 11). Two patients had areas of abnormal perfusion suspected from the plain film chest roentgenogram and confirmed by angiography. The other 3 were selected for study at random, and in 2 of them unsuspected segmental occlusions of the pulmonary arteries were present. In all the cases with segmental lesions the bronchial arteries of the corresponding segment were hypertrophied and were vis-

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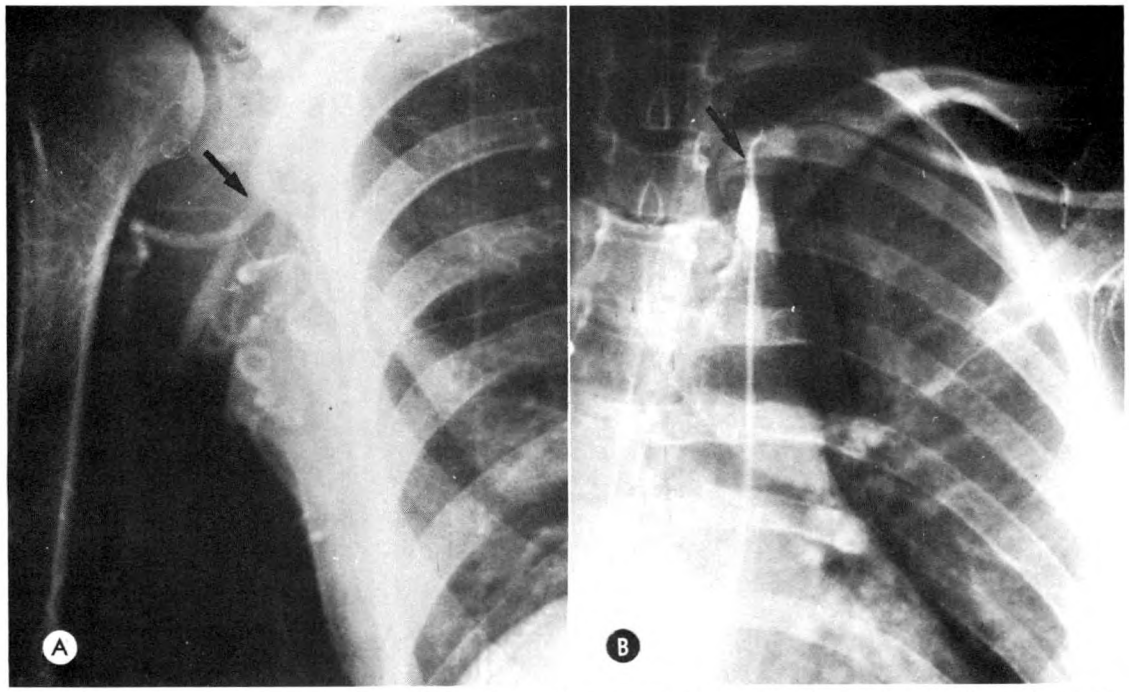


FIG. 7. (*A*) Case 20. Stenotic lesion of the right axillary artery (arrow) with prominent collateral vessels. Distal lesions were only seen in the right axillary artery. (*B*) Case 8. Proximal "flame-shaped" stenosis of left subclavian artery (arrow). Lesions proximal to the origin of the vertebral artery tend to occur on the left side only.

ualized either in the levophase of the pulmonary angiogram or during aortography. In 2 other cases prominent bronchial arteries were present, but pulmonary angiography was not performed.

E. POSTMORTEM STUDIES

Seven of the 22 patients expired and postmortem studies were available in 5. Macroscopically, a good correlation was found between the angiographic studies and the necropsy findings. Histologic examination revealed signs of panarteritis with lymphocytic infiltration of the adventitia, disruption of the muscular and elastic tissue of the media, and thickening of the intima with superimposed thrombi.

DISCUSSION

In the present series the diagnosis of Takayasu's arteritis was based primarily on the angiographic findings of involvement of the thoracic or abdominal aorta and their major branches. Cases with lesions of the smaller vessels or with peripheral arterial involvement were excluded. According to some reports Takayasu's arteritis may rarely affect the femoral arteries,[41] but we felt that without histologic proof of arteritis in these patients, atherosclerosis was a more likely etiology. Consequently, we performed peripheral arteriography in 13 of the cases, even in the absence of symptoms referable to the legs, to demonstrate normal femoral arteries. For the same reason, we only included patients under the age of 40 years at the time of initial study, except for 2 patients in whom a definite diagnosis was established at autopsy.

Since the diagnosis may be suspected from the deformation and irregularities of the aorta seen on plain film chest roentgenograms, patients with characteristic presenting symptoms or signs should receive careful roentgenologic attention. The diagnosis is established, however, on the basis of the angiographic studies which

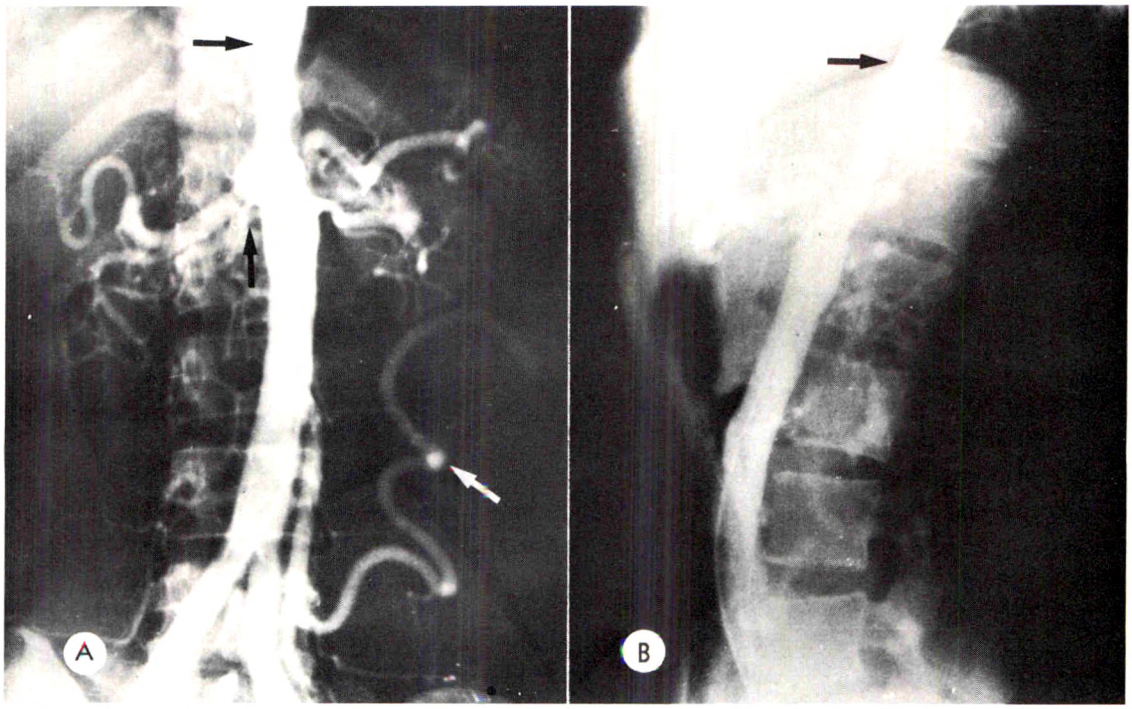


Fig. 8. Case 14. Abdominal aortograms, frontal and lateral view. (*A*) Long stenotic lesion of thoraco-abdominal aorta (horizontal arrow) gradually tapering to approximately half its original diameter, localized aneurysm of mid abdominal aorta (vertical arrow), and total occlusion of the superior mesenteric artery with prominent collateral supply from the inferior mesenteric artery (white arrow) were demonstrated. (*B*) Lateral view shows character of stenotic segment (arrow) of thoracic aorta. Also visible is a shallow aneurysm below the celiac artery, aneurysmal dilatation of the hepatic artery, and total occlusion of the superior mesenteric artery.

should include the entire aorta and proximal peripheral arteries.

We believe that the incidence of pulmonary artery involvement has been underestimated in the past.[25] Four out of 5 patients that we studied had segmental stenosis or occlusions. In 2 patients abnormal perfusion was suspected on the chest roentgenograms, but pulmonary angiography was performed in the others even though there were no symptoms or suspicious findings on the chest roentgenograms. All of the occluded segments received ample collateral supply from the corresponding bronchial artery. Other authors have described perfuse collateral supply from intercostal arteries. We did not observe this in our small series.[41] We believe that the presence of pulmonary artery lesions supports the diagnosis of Takayasu's arteritis in those cases where differentiation between arteritis and atherosclerosis is difficult.

Although Takayasu's arteritis is usually said to be a disease of young women, we found almost an equal incidence of affected men. Female predominance is especially noteworthy in the reports from the Far East, but this sex incidence is a lesser feature in the reports from Africa and Northern European countries.[19,34] However, the 1.2:1, female-to-male ratio which we observed is unusual.

In most geographic areas the brachiocephalic vessels and the aortic arch are found to be the most frequently involved.[3,4,20,21,24] Aortitis of the ascending aorta with dilatation of the aortic ring, aortic insufficiency and obliteration of the coronary arteries is not rare in Japan, South Africa and Thailand.[13,42,50,51] In certain countries, however, such as India, the

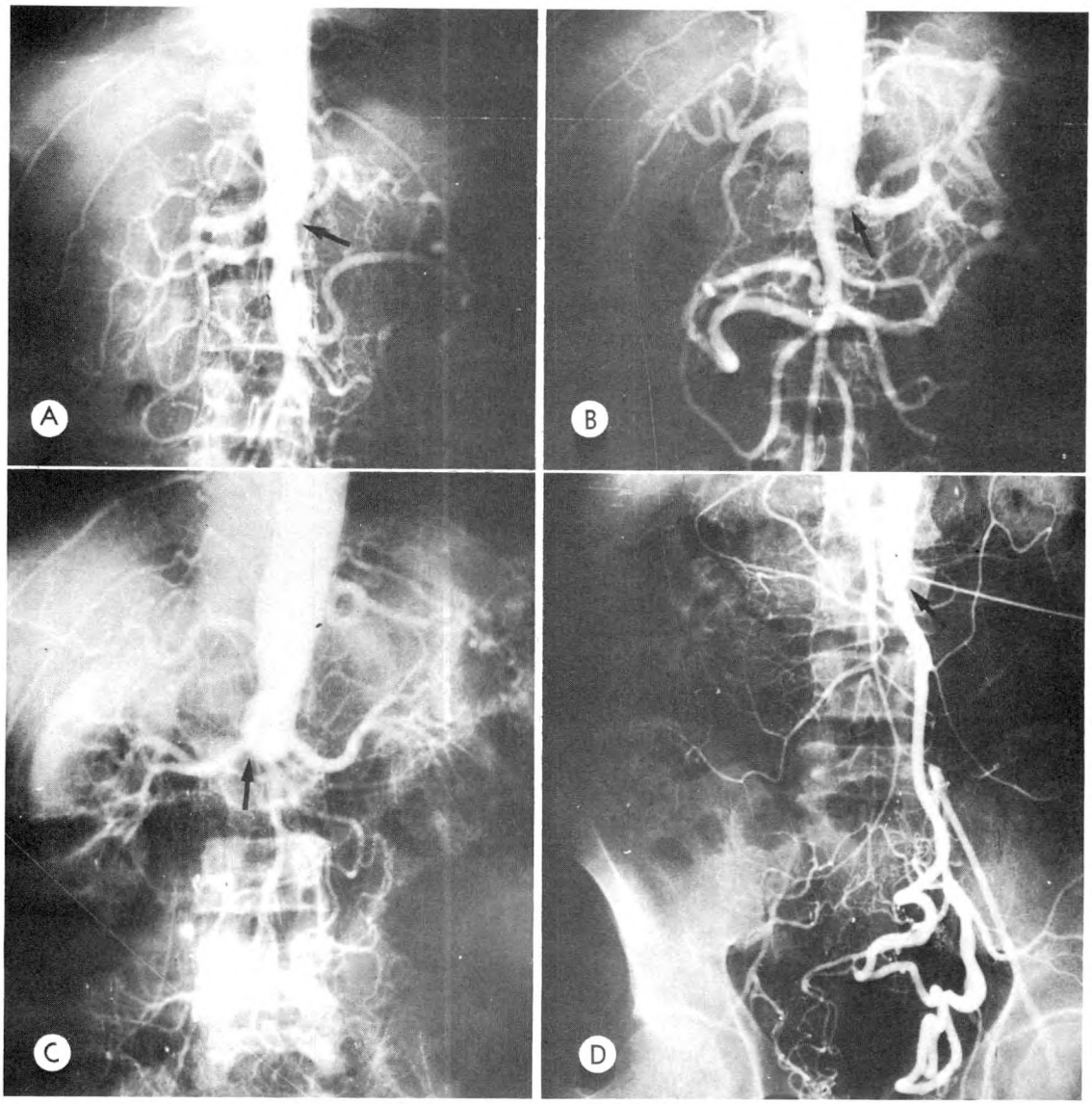


FIG. 9. Abdominal aortograms in 4 different patients. (*A*) Case 5. Occlusion of aorta (arrow) at level of renal arteries. The origin of the right renal artery is stenotic; the left renal artery is occluded. The intercostal and superior mesenteric arteries serve as collaterals and are markedly hypertrophied. (*B*) Case 6. Occlusion of aorta (arrow) at level of renal arteries. The right renal artery is totally occluded; the left renal artery is stenotic at its origin. Collateral circulation to distal aortic branches is *via* hypertrophied superior mesenteric and intercostal arteries. (*C*) Case 9. Total occlusion of aorta (arrow) just below origin of renal arteries. Shallow aneurysms of the upper aortic segment. Slight stenosis at origin of the right renal artery. (*D*) Case 17. Total occlusion of distal aorta just below the origin of the inferior mesenteric artery (arrow). The markedly enlarged inferior mesenteric artery serves as the main collateral route to the lower extremities *via* connections between its sigmoid and rectal branches and the hypogastric arteries.

descending thoracic aorta is the most commonly involved and the ascending aorta and coronary arteries are free of disease.[23] Involvement of the abdominal aorta, particularly its upper and mid segment is reported with varying frequency.[21,44] In contrast, however, reports from Mid and West African countries state that the distal aorta is the most frequently diseased segment.[19,33]

Geographic variation is also observed in the type of lesions found. In Europe, Japan and the U.S., vascular stenoses or occlusions are common and aneurysms rare, in contrast to India, Thailand and Africa, where aneurysms are a prominent feature of the disease.[18,51] In this regard it is of interest that all 4 Indian patients in our series had aneurysms of the thoracic aorta, while only 3 other patients had similar aneurysms. Occlusion or stenosis of the abdominal aorta was a predominant feature in 3 of 5 Beduins from the Sinai peninsula and 3 out of 4 Arabs from Gaza, again suggesting that ethnic origin may be a factor influencing the pattern of regional involvement. The only part of the world from which reports of distal aortic involvement are common is Africa,[19,33] and it is likely that an African influence is to be found among the Arabs and particularly the Sinai Beduins.

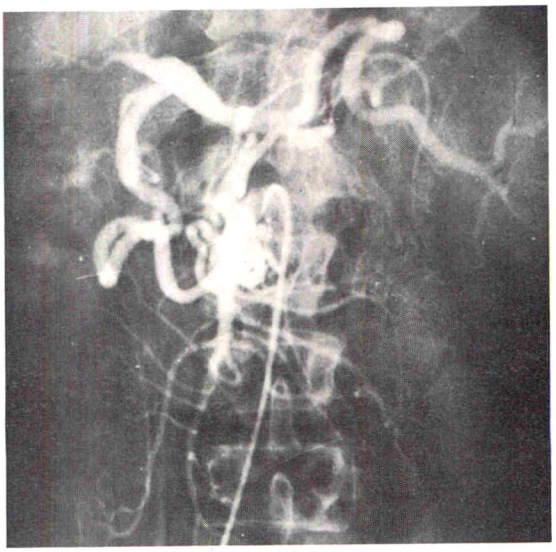


Fig. 10. Case 10. Selective superior mesenteric arteriogram. The celiac artery is occluded at its origin and fills *via* pancreatico-duodenal arcade. Note aneurysmatic dilatation of the proximal and stenosis of the distal part of the hepatic artery.

The ethnic distribution of the patients in our series is of great interest (Table 1). It seems more than coincidental that none of our patients were Jews of Ashkenazi origin. The average population at the Chaim Sheba Medical Center is approximately half Ashkenazi and half Sephardi, which corresponds to the general division of the population in Israel.[47] Very small numbers originate from India or Ethiopia. Therefore, the fact that 4 patients in our small series were born in India would support the view that the disease is more common among Indian born patients than among the other ethnic groups. In addition, the fact that among the 13 Jewish subjects all were of Sephardic

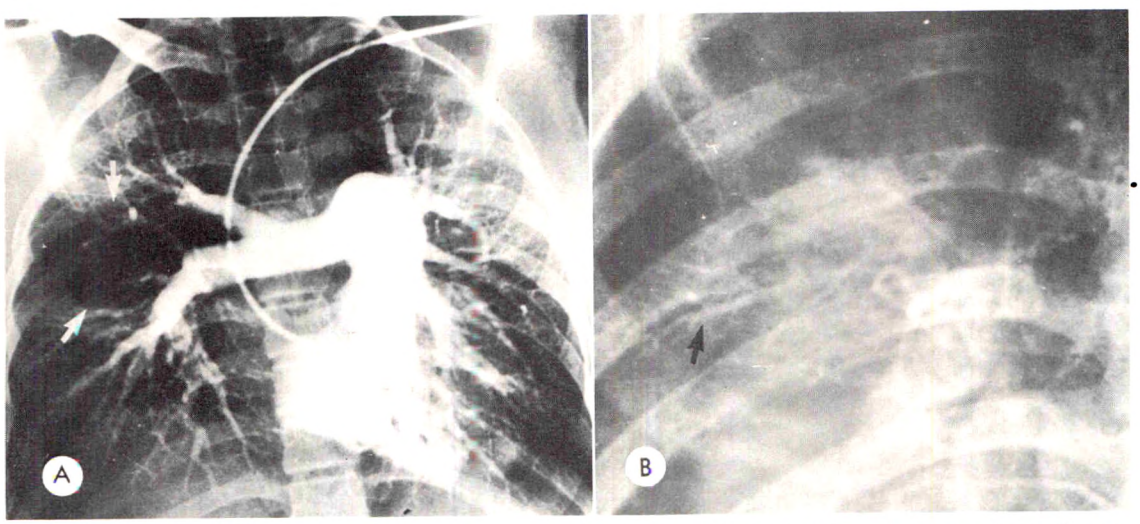


Fig. 11. Case 1. (*A*) Pulmonary angiogram. Arrows indicate area of diminished flow. (*B*) Late phase of aortogram. The area that is poorly perfused on pulmonary arteriogram has compensatory bronchial artery flow which communicates with the pulmonary artery branches (arrow).

origin without a single Ashkenazi suggests that the ethnic-genetic factor may be more important than the environmental. In this regard it should also be noted that 3 of the Sephardic patients came from Turkey, Bulgaria and Greece, where the environmental conditions are similar to those of Western Europe. The prevalence of Takayasu's arteritis among Sephardic Jews in Israel and its low incidence among Ashkenazim was also found by our colleagues in Haifa.[39] This is in direct contrast with thromboangiitis obliterans[12] which has the opposite distribution among the Israeli population. It should be emphasized that the observed ethnic distribution emerged in retrospect, after the cases of Takayasu's disease were collected.

As described earlier, the Jews living in Spain and North Africa have a close relationship to each other and there is little doubt that they were under the genetic influence of the North African population through intermarriage. Further confirmation of a possible genetic factor is found in the reports from the U.S. of non-Jewish subjects with Takayasu's arteritis where there is a relatively common occurrence of the disease among those of Spanish or African origin.[14,29]

Others have speculated as to the etiology of Takayasu's arteritis. Most authors believe that it is one of the autoimmune diseases.[34,50] The ethnic factor, which seems to be a prominent feature in our series, may influence the susceptibility of various population groups and may determine the target areas in the aorta.

Some authors have suggested that streptococcal or tuberculous infections may exert a triggering effect in the development of the disease.[23] The antigen-antibody reactions resulting from these infections may be type specific and may also have an immunologic affinity for the aorta and its branches. Only 4 patients in our series had a positive tuberculin skin test and 3 had tuberculous lymphadenopathy. Our material, therefore, neither supports nor refutes the post infection theory.

Six of our patients had repeat aortography 2 to 5 years following the initial study. These patients had been treated with steroids and/or antituberculous therapy and had shown clinical improvement. However, roentgenologically no improvement could be demonstrated. Five of the patients showed no change whatever in the angiographic pattern of occlusions and stenoses. One patient followed from the acute stage showed aneurysms which progressed to stenosis and occlusion, and also the reverse, stenotic segments which became aneurysmal. Although the natural history of the disease has not been fully elucidated, it is thought to be progressive, although at an unpredictable rate. Our material suggests that successful therapy will impede the progression of the vascular lesions, but should not be expected to reverse them. Vessels once affected do not revert to normal.

CONCLUSION

Takayasu's arteritis is found among the Sephardic and Oriental Jewish, Arab and Beduin populations of Israel, but not yet among the Jews of Ashkenazic origin.

Among the 22 patients in this series the sex distribution was approximately equal, in contradistinction to the female predominance usually reported from the Far East.

The regional distribution and character of the aortic involvement bore a relationship to the ethnic origin of the patient.

Aneurysms of the thoracic aorta had a predilection for Indian patients, while coarctation or occlusion of the abdominal aorta was characteristic in the Arabs and Beduins.

While the disease may be suspected from the clinical findings and the plain film chest roentgenogram, aortography is required for the diagnosis. Complete evaluation should include pulmonary angiography, since segmental involvement of the pulmonary arteries may be unsuspected and probably occurs in a high percentage of patients with the disease. Involvement of the pulmonary arteries will support the diagnosis

of Takayasu's disease *vs.* atherosclerosis in those patients in whom the differentiation may be difficult.

Patients may show clinical improvement following appropriate therapy, although the angiographic findings are usually unchanged. In 1 case progression of the disease was documented over a 5 year period.

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REFERENCES

1. Abrahams, D. G., and Cockshott, W. P. Multiple non-luetic aneurysms in young Nigerians. *Brit. Heart J.*, 1962, *24*, 83–91.
2. Adams, R. Quoted from Reaves, L. E., III.[35]
3. Ask-Upmark, E. On "pulseless disease" outside of Japan. *Acta med. scandinav.*, 1954, *149*, 161–178.
4. Ask-Upmark, E., and Fajers, M. C. Further observation on Takayasu's syndrome *Acta med. scandinav.*, 1956, *155*, 275–291.
5. Birke, G., Ejrup, B., and Olhagen, B. Pulseless disease: clinical analysis of 10 cases. *Angiology*, 1957, *8*, 433–455.
6. Caccamise, W. C., and Whitman, J. F. Pulseless disease. *Am. Heart J.*, 1952, *44*, 629–633.
7. Chang, H. T., Chang, A., and Chin, F. H. Quoted from McKusick V. A.[30]
8. Coon, C. S. The Races of Europe. Macmillan Company, New York, 1939, p. 432.
9. Danaraj, J. T., and Ong, H. W. Primary arteritis of abdominal aorta in children causing bilateral stenosis of renal arteries and hypertension. *Circulation*, 1959, *20*, 855–863.
10. Davy, J. Quoted from Keun-Soo, L., *et al.*[21]
11. Domingo, R. T., Maranba, T. P., Torres L. F., and Wesolowski, S. A. Acquired aorta arteritis: worldwide vascular entity. *A.M.A. Arch. Surg.*, 1967, *95*, 780–790.
12. Goodman, R. M., Elian, B., Mozes, M., and Deutsch, V. Buerger's disease in Israel. *Am. J. Med.*, 1965, *39*, 601–615.
13. Gotsman, M. S., Beck, W., and Schrire, V. Selective angiography in arteritis of aorta and its major branches. *Radiology*, 1967, *88*, 232–248.
14. Harrell, J. E., and Manion, W. C. Sclerosing aortitis and arteritis. *Seminars in Roentgenol.*, 1970, *3*, 260–266.
15. Herlitz, G., and Kirschner, B. Judisches Lexikon. *Judischer Verlag, Berlin*, 1930, *4*, 1146–1151.
16. Hirsch, M. S., Aikat, B. K., and Basu, A. K. Takayasu's arteritis: report of five cases with immunological studies. *Bull. Johns Hopkins Hosp.*, 1964, *115*, 29–64.
17. Isaakson, C. Idiopathic aortitis in young Africans. *J. Path. & Bact.*, 1961, *81*, 69–79.
18. Joffe, N. Idiopathic panarteritis with aneurysm formation. *Clin. Radiol.*, 1965, *16*, 251–255.
19. Joffe, N. Aortitis of obscure origin in African. *Clin. Radiol.*, 1965, *16*, 130–140.
20. Judge, D. R., Currier, D. R., Gracie, A. W., and Figley, M. M. Takayasu's arteritis and aortic arch syndrome. *Am. J. Med.*, 1962, *32*, 379–392.
21. Keun-Soo, L., Ki-Yong, S., Chank-Yee, H., Suk-Rin, K., and Knut, B. Primary arteritis (pulseless disease) in Korean children. *Acta pediat. scandinav.*, 1967, *56*, 526–536.
22. Kim, C. S. Quoted from McKusick, V. A.[30]
23. Kinare, S. G. Etiopathological aspects of non-specific aortitis. In: Cardiology, Current Topics and Progress. Edited by H. Eliakim, and H. N. Neufeld. Academic Press, Inc., New York, 1968, pp. 322–325.
24. Koszewski, B. J. Branchial arteritis or aortic arch arteritis: new inflammatory arterial disease (pulseless disease). *Angiology*, 1958, *9*, 180–186.
25. Kozuka, T., Nosaki, T., Soto, K., and Ihara, K. Arteritis syndrome with special reference to pulmonary vascular changes. *Acta radiol.* (Diag.), 1968, *7*, 25–32.
26. Kozuka, T., Nosaki, T., Soto, K., and Tachiiri, H. Aneurysm associated with aortitis syndrome. *Acta radiol.* (Diag.), 1968, *7*, 314–320.
27. Kussmaul, A. Quoted from McKusick, V. A.[30]
28. Kwak, N. O., Chang, J. H., and Lee, S. H. Quoted from McKusick, V. A.[30]
29. Lande, A., and Gross, A. Total aortography in diagnosis of Takayasu's arteritis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 165–178.
30. McKusick, V. A. Form of vascular disease relatively frequent in Orient. *Am. Heart J.*, 1962, *63*, 57–64.
31. Nasu, T. Pathology of pulseless disease. *Angiology*, 1963, *14*, 225–242.
32. Niimi, X. X. Quoted from Nasu, T.[31]
33. Pepler, N. J., and Simson, I. W. Occlusion disease of abdominal aorta associated with panarteritis. *A.M.A. Arch. Path.*, 1959, *68*, 525–532.
34. Poloheimo, J. A. Obstructive arteritis of Takayasu's type: clinical, roentgenological and laboratory studies on 36 patients. *Acta med. scandinav.*, 1967, Suppl. 4, pp. 1–42.
35. Reaves, L. E., III, and Wasserman, A. J. Takayasu's aortic arch syndrome: report of case with associated hypoplasia of aorta and

review of literature. *Angiology*, 1964, *15*, 280–282.

36. ROTH, C. Encyclopaedia Judaica, Jerusalem, 1973. Volume 6, pp. 1143–1154; Volume 16, pp. 739–758; Volume 8, pp. 1349–1362.
37. SACHS, H., and BAT-MIRIAM, M. Genetics of Jewish population. I. Fingerprint patterns in Jewish population in Israel. *Am. J. Human Genet.*, 1957, *9*, 117–126.
38. SAVORY, W. S. Quoted from Reaves, L. E., III.[35]
39. SCHRAMEK, A. Personal communication.
40. SCHRIRE, V., and ASHERSON, R. A. Arteritis of aorta and its major branches. *Quart J. Med.*, 1964, *33*, 439–463.
41. SCHRIRE, V. Arteritis of aorta and its major branches. *Australasian Ann. Med.*, 1967, *16*, 34–40.
42. SCHRIRE, V. Discussion in symposium of non-specific arteritis of aorta and its branches. In: Cardiology, Current Topics and Progress. Edited by H. Eliakim, and H. N. Neufeld. Academic Press, Inc., New York, 1968, pp. 336–338.
43. SEN, P. K. Obstructive disease of aorta and its branches. In: Cardiology, Current Topics and Progress. Edited by H. Eliakim, and H. N. Neufeld. Academic Press, Inc., New York, 1968, pp. 329–331.
44. SEN, P. K., KINAR, S. G., KULKARNI, T. P., and PARULKAR, G. B. Stenosing aortitis of unknown etiology. *Surgery*, 1962, *51*, 317–325.
45. SHIMUZU, K., and SANO, K. Pulseless disease. *Neuropath. & Clin. Neurol.*, 1951, *1*, 37–47.
46. TAKAYASU, M. Quoted from Judge, D. R., *et al.*[20]
47. Tel-Hashomer Hospital Statistical Records, 1956, p. 34; 1957, p. 9; 1958, p. 10; 1959, p. 7; 1960, p. 10; 1961, p. 13; 1962, p. 6. Government Printing Press, Israel.
48. UEDA, H., ITO, I., and SAJTO, Y. Quoted from Keun-Soo, L.[21]
49. UEDA, H., ITO, I., OKUDA, X., *et al.* Quoted from Kozuka, T., *et al.*[25]
50. UEDA, H. Etiology and clinical manifestations of "aortitis disease." In: Cardiology, Current Topics and Progress. Edited by H. Eliakim, and H. N. Neufeld. Academic Press, Inc., New York, 1968, pp. 320–321.
51. VINIJCHAIKUL, K. Pathological spectrum of sclerosing aortitis. In: Cardiology, Current Topics and Progress. Edited by H. Eliakim, and N. H. Neufeld. Academic Press, Inc., New York, 1968, pp. 326–328.
52. WIGODER, G. Jewish Art and Civilization. Volume 1. Weidenfeld Nicolson, Jerusalem, 1972, pp. 76–77; 108–109.

Vol. 122, No. 1

COARCTATION OF THE ABDOMINAL AORTA WITH ANEURYSM OF MIDDLE CEREBRAL ARTERY*

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COARCTATION of the abdominal aorta is a rare entity and comprises approximately 2 per cent of all coarctations of the aorta. This may be part of a disease complex or may be seen as an isolated entity. The patients typically present either with systemic hypertension or intermittent claudication, with surgical by-pass of the coarctation being the treatment of choice.

The patient described here represents the second case of abdominal aortic coarctation associated with intracranial aneurysms reported. In view of the limited attention abdominal coarctation has received in the radiologic literature, the details of this case and a review of the general topic are presented.

REPORT OF A CASE

B.B., a 16 year old white female, entered the hospital in November, 1968, with symptoms of fainting, nausea, vomiting, neck and back pain. Physical examination revealed a blood pressure of 230/130 mm. Hg; the patient was lethargic, but without focal neurologic signs. The findings on a lumbar puncture—numerous red cells and increased cerebrospinal fluid pressure—indicated a subarachnoid hemorrhage. A carotid angiogram showing an aneurysm at the knee of the left middle cerebral artery (Fig. 1), was followed by a left frontotemporal craniotomy through which clips were placed at the neck of the aneurysm. Subsequent to surgery, the patient had right upper and lower extremity paresis which resolved over the ensuing months. She was then discharged and readmitted later for evaluation of hypertension.

A coarctation was clinically suspected because of weak femoral pulsations. An abdominal aortogram obtained *via* a left brachial approach revealed a 7 cm. in length segment of coarctation extending from just above the celiac artery to include the superior mesenteric and renal arteries. Significant stenoses of the celiac, superior mesenteric, and both renal arteries were present. A large inferior mesenteric artery supplied collateral flow to the superior mesenteric artery *via* the arc of Riolin (Fig. 2).

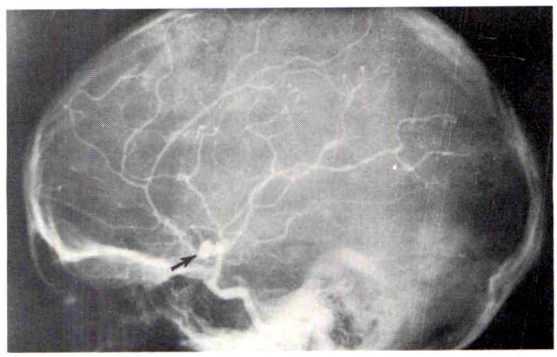


FIG. 1. There is a lobulated aneurysm of the left middle cerebral artery (arrow).

At surgery, confirmation of the roentgenologic findings was obtained. A dacron graft was used to by-pass the area of coarctation. An end-to-side anastomosis of the left renal artery to the dacron graft was performed as well as an end-to-side saphenous vein graft extending from the coarcted aorta to the right renal artery. Subsequent to surgery, the blood pressure dropped to 180/110 mm. Hg, but showed no additional improvement on follow-up examination. For this reason, she was placed on antihypertensive medication. A second arteriogram demonstrated that all the grafts were functioning with apparently normal contrast medium flow (Fig. 3). Abnormally high bilateral renal vein renin levels indicated the possibility of persistent renal ischemia.

DISCUSSION

The first report of an abdominal coarctation was in 1884 by Quain.[13] Bahnson *et al.*[1] were the first to describe the angiographic features of the lesion. Glenn *et al.*,[9] in 1962,

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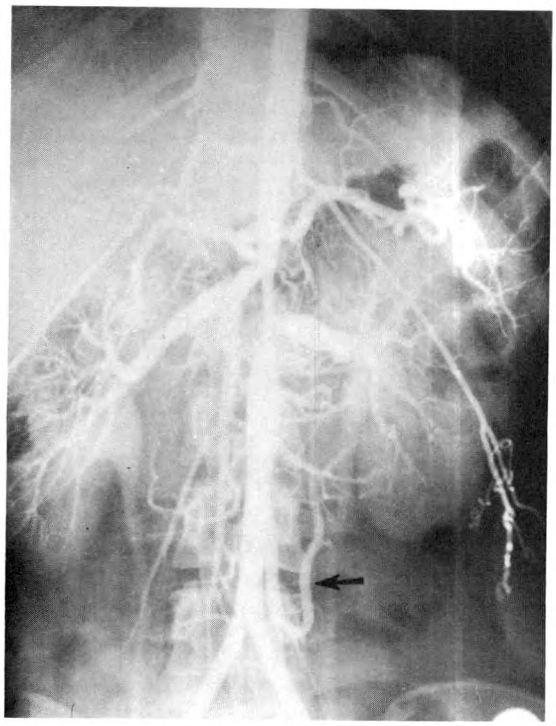


FIG. 2. Coarctation extends from just above the celiac artery to the infrarenal segment with poststenotic dilatation. Stenoses involve the proximal celiac, superior mesenteric artery (SMA), and both renal arteries. A large arc of Riolin extending from the inferior mesenteric artery provides collateral flow to the SMA (arrow).

described the first operation for this anomaly in which the splenic artery was used to by-pass the coarctation. Maycock[12] states that the lesion is secondary to failure of fusion of 2 dorsal aortas which is followed by disappearance of one of these with resultant diminutive size of the remaining dorsal aorta. In Orientals the lesion is often of an inflammatory etiology, very likely part of the spectrum of Takayashu's disease,[17] with abdominal aortic coarctation almost as common as that of the thoracic lesion.[11] In the United States, the evidence supports its congenital nature. It is especially common in children and young adults; is often seen with other congenital anomalies; and histopathologically lacks inflammatory signs.[3,10,17]

In abdominal coarctations the female to male ratio is 2:1 as opposed to the thoracic coarctation female to male ratio of 3:1.[14] The mean age at diagnosis is 21 years. Abdominal aortic coarctations are classified as to their segmental or diffuse nature and a suprarenal, interrenal, or infrarenal location.[3,7] These lesions may be seen with coarctation of the thoracic aorta; multiple pulmonary arterial stenoses, valvular aortic stenoses, mitral insufficiency, subclavian stenoses,[2,13] renal artery aneurysms, idiopathic hypercalcemia, neurofibromatosis, rubella, Takayashu's disease, or as an isolated entity.[14,15,17] Two cases of abdominal aortic and renal artery hypertension have been reported in children after radiotherapy for abdominal tumors.[5] Fisher and Corcoran[8] reported a 14 year old boy with severe systemic hypertension, abdominal aortic coarctation, renal artery stenoses, and aneurysms of the left anterior cerebral and lenticulostriate arteries, seen at postmortem examination.

Should the stenoses be supra- or infrarenal, the patient typically will present with systemic hypertension. When the most significant narrowing is in the infra-

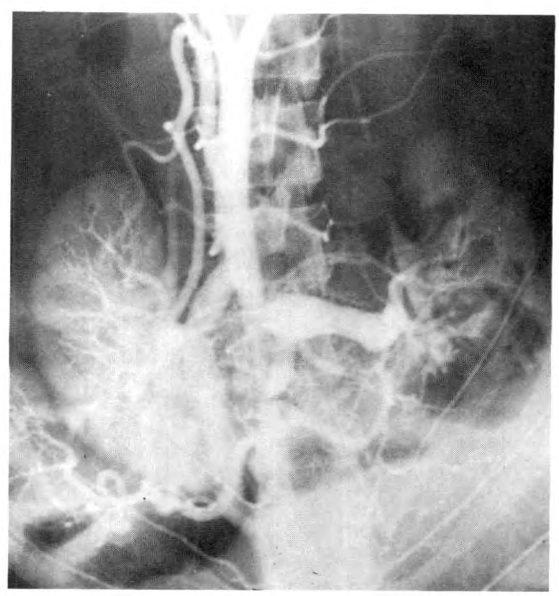


FIG. 3. A dacron graft by-passes the area of aortic coarctation. A left renal artery to dacron graft and an end-to-side right saphenous vein graft extends from the coarcted aorta to the renal artery.

renal segment of the aorta, intermittent claudication with or without hypertension is the predominant complaint. Headache, palpitation, dyspnea on exertion, giddiness, lethargy, abdominal angina, cerebrovascular accident, seizures, or congestive heart failure are occasionally manifested. Systemic hypertension may be due to either mechanical effects of the coarctation itself, or to renal ischemia.[4] Sometimes bruits are heard and notching of the inferior 4 ribs is present.

Angiography should define the location and extent of the lesion, the condition of the renal and splanchnic vessels, and any collateral circulation.

PATHOLOGY

Most of the specimens submitted are histopathologically normal. On occasion, marked intimal proliferation, fibromuscular dysplasia, nonspecific inflammatory reaction of the aortic wall, intraluminal thrombosis, atresia or stenosis are seen.[13] Arteriosclerotic changes may coexist proximal to the coarctation, or occasionally plexiform neurofibromata encase the aorta in patients with neurofibromatosis.

TREATMENT

Without major peripheral vessel involvement a localized coarctation may be resected, and an end-to-end anastomosis or small patch graft applied. When the coarctation is extensive, with involvement of the renal or splanchnic branches, then a by-pass graft extending from above to below the coarctation is efficacious. Due to Bernoullie's principle, as the flow through the coarcted segment is diminished, the pressure on the side walls of the aorta is increased, and this pressure will lead to increased flow through the branch vessels. However, this may not be sufficient for an effective blood flow, and therefore side grafts often are performed to the kidneys with either dacron or saphenous vein material. A nephrectomy may be necessary, if there are multiple areas of renal artery stenoses or the stenotic areas are of excessive length. There is typically no need for splanchnic revascularization as collaterals usually suffice.[16] Most of the patients who undergo surgery become normotensive.[6,14]

SUMMARY

(1) Coarctation of the abdominal aorta is rare, the incidence being 2 per cent of all aortic coarctations.

(2) The patient reported is only the second case associated with intracranial aneurysms.

(3) Symptoms most often are those of systemic hypertension and/or intermittent claudication.

(4) Angiography defines the extent of the coarctation and any collateral circulation.

(5) Most specimens are histopathologically normal.

(6) Surgical by-pass of the lesion often cures the hypertension.

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REFERENCES

1. Bahnson, H. T., Looley, R. N., and Sloan, R. D. Coarctation of aorta at unusual sites: report of two cases with angiographic and operative findings. *Am. Heart J.*, 1949, *38*, 905–913.
2. Baird, R. J., Evans, J. R., and Labrosse, C. L. Coarctation of abdominal aorta. *A.M.A. Arch. Surg.*, 1964, *89*, 466–474.
3. Ben-Shoshan, M., Rossi, N. P., and Korus, M. E. Coarctation of abdominal aorta. *A.M.A. Arch. Path.*, 1973, *95*, 221–225.
4. Brust, A. A., Howard, J. M., Bryant, M. R., and Godwin, J. T. Coarctation of abdominal aorta with stenosis of renal arteries and hypertension: clinical and pathologic study of two cases and review of literature. *Am. J. Med.*, 1959, *27*, 793–802.
5. Colquhoun, J. Hypoplasia of abdominal aorta following therapeutic irradiation in infancy. *Radiology*, 1966, *86*, 454–456.
6. DeBakey, M. E., Garrett, H. E., Howell, J. F., and Morris, G. C. Coarctation of abdominal aorta with renal arterial stenosis: surgical considerations. *Ann. Surg.*, 1967, *165*, 830–843.

7. DOBENECK, R. C., and VARCO, R. L. Congenital coarctation of abdominal aorta. *Lancet*, 1968, *88*, 143–150.
8. FISHER, E. R., and CORCORAN, A. C. Congenital coarctation of abdominal aorta with resultant renal hypertension. *A.M.A. Arch. Int. Med.*, 1952, *89*, 943–950.
9. GLENN, F., KEEFER, E. B. C., SPEER, D. S., and DOTTER, C. J. Coarctation of lower thoracic and abdominal aorta immediately proximal to celiac axis. *Surg., Gynec. & Obst.*, 1962, *94*, 561–569.
10. HUANG, T. T., WOLMA, F. J., and TYSON, K. R. Coarctation of abdominal aorta. *Am. J. Surg.*, 1970, *120*, 598–601.
11. KIMOTO, S. Surgical treatment of coarctation of aorta with special reference to atypical coarctation. *Clin. Surg.*, 1960, *15*, 5.
12. MAYCOCK, W. A. Congenital stenosis of abdominal aorta. *Am. Heart J.*, 1937, *13*, 633–646.
13. QUAIN, R. Partial coarctation of abdominal aorta. *Tr. Path. Soc.* London, 1884, *1*, 244–245.
14. RIEMENSCHNEIDER, T. A., EMMANOUBIDES, G. C., HOUSE, F., and LIODE, L. M. Coarctation of abdominal aorta in children: report of three cases and review of literature. *Pediatrics*, 1969, *44*, 716–726.
15. SERRING, A., and JOHANSON, L. Coarctation of abdominal aorta. *J. Thoracic & Cardiovasc. Surg.*, 1960, *40*, 517–523.
16. SPROUL, G., and PINTO, J. Coarctation of abdominal aorta. *A.M.A. Arch. Surg.*, 1972, *105*, 571–573.
17. UEDA, H., MORROKA, S., ITO, I., YAMAGUCHI, H., TAKEDA, T., and SAITO, Y. Clinical observations of 52 cases of aortic syndrome. *Jap. Heart J.*, 1969, *10*, 277–288.

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CRANIAL GROWTH RETARDATION FROM MATERNAL PHENYLKETONURIA*

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WE wish to report a unique and interesting biochemical cause of retarded brain growth, which is being recognized with increasing frequency. Study of this specific cause of retarded growth emphasizes both the importance and the variability of well known but often overlooked radiologic signs of slowed brain growth and the fallibility of radiologic measurement in such cases.

MATERIAL

Three families of mentally retarded children of mothers with phenylketonuria (PKU) have been seen at the Alvin Buckwold Centre, University of Saskatchewan (Table 1). These 3 mothers have produced 8 retarded and 2 borderline retarded children. In addition 1 child was stillborn and 3 died neonatally with congenital anomalies. These children represent one of the largest series collected; in the last 5 years we have discovered more such retarded but biochemically normal children of PKU mothers than new cases of the disease, PKU (9 *vs.* 7). Each child presented radiologically with sometimes subtle but definite evidence of retarded brain growth.

RESULTS

The radiologic findings in children with retarded brain growth born of mothers with phenylketonuria, are often subtle and overlooked (Fig. 1, *A–D*; and Table 1). It is common for a smooth-walled vault in a child of 3 to 10 years to be interpreted incorrectly as normal, whereas well developed normal convolutional markings may raise unjustified alarm of increased intracranial pressure. Convolutional markings normally appear between 1 and 3 years of age; between 3 and 10 or 12 years, absent or diminished markings are a sign of abnormality.[3] As yet we have not encountered a normal child of this age group with absent convolutional markings. The absence of convolutional markings in our patients therefore is abnormal. Premature sclerosis of the sutures, particularly occurring at 6 and 8 years of age, as in 2 of our patients (Fig. 1*C*), is another significant sign of decreased brain growth. Flattening of the occiput was pronounced in 1 patient; this is nonspecific but occurs more frequently in retarded and inactive children who spend a great deal of time lying on their backs. Children in this series have not had noticeable thickening of the cranial vault.

Measurement of the skull roentgenograms proved disappointing in these cases. Since 85 per cent of brain growth and cranial vault growth has been achieved before 3 years of age, a child of 6 years with a vault size of only 2 years, represents only a 5 per cent difference in any one dimension; this is not apparent by casual inspection. Only rather advanced degrees of microcephaly are evident to the observer; this small size was readily apparent on visual inspection of the roentgenograms in only 3 of our cases.

Several of our children whose roentgenographic skull measurements fall well within the range of normal, based on values given by Lusted and Keats,[6] have head circumference measurements below minus 2 standard deviations (below the second percentile). In Table 1, the measurements falling below Lusted and Keats' range of normal are italicized. Comparison of head circumference of our patients with normal values is shown in Figure 2. It is apparent

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TABLE I

THREE FAMILIES WITH MENTALLY RETARDED CHILDREN OF MOTHERS WITH PKU

	Bio-chemistry	Birth Weight	Age	I.Q.	Head Circumference	Breadth	Length	Height	Convolutional Markings
D Family									
Mrs. M.	PKU	?	31	55	54.6	158	191	148	(Adult)
Miss D	N	2.3	8	34	46.5	129	178	149	Decreased
Miss A.[1,3]	N	2.4	6	32	44.7	140	164	139	Decreased
Mstr. D.[3]	N	2.3	5	36	45.4	*128*	166	*132*	Decreased
Miss M.[3]	N	2.8	3	26	43.2	*129*	*159*	135	Decreased
P Family									
Mrs. E.[4]	PKU	?	32	58	52.0	155	195	142	(Adult)
Prem. Girl[1]	N	2.0	2 da						
Prem. Boy[1]	N	2.0	2 da						
Mstr. Dn.[1,3]	N	2.2	7	42	47	147	*170*	150	Absent
Mstr. Dl.[1,3]	N	2.4	6	45	46	143	*165*	143	Absent
Stillbirth Boy	N	1.6	—	—					
Mstr. De.[3]	N	2.3	3	44	42	*131*	*158*	*132*	Absent
Prem. Boy[2]	N		2 da						
(One spontaneous abortion)									
K Family									
Mrs. R.	PKU	?	32	80	54.4	156	209	161	(Adult)
Mstr. J.[3]	N	2.5	5	85	49.3	146	178	146	Absent
Miss A.M.	N	2.1	3	75	44.2	*130*	167	*132*	Absent
Miss L.	PKU	2.6	2	77	45.5	*131*	166	*130*	Absent
(One spontaneous abortion)									

N = Normal
PKU = phenylketonuria
da = days

1 = congenital heart disease
2 = esophageal atresia and cleft palate
3 = increased muscle tone, abnormal gait
4 = cleft palate

that the head circumference measurement is an accurate, better standardized and much easier method of assessing skull size and must be preferred to roentgenographic measurements. The small cranial vault is most evident at birth; during childhood, no longer in an abnormal biochemical environment, the size of the cranial vault seems to "catch up" to some degree.

DISCUSSION

When an individual child or particularly when several children of one family are seen with subtle but definite radiologic signs of diminished brain growth, it is not enough to test the children biochemically, but one must also test the mother for PKU.[5] This is important for diagnosis, genetic counselling and family planning and has major economic significance, since custodial care of a severely retarded child is a heavy burden on the family and on society.

Since routine testing for PKU throughout most of this continent in the past 2 decades has resulted in the correct diagnosis of most PKU children, allowing treatment with low phenylalanine diets, many have avoided the severe brain damage that otherwise might have occurred. More PKU patients are functioning normally in the community, and in years to come will marry and have children, although their doctors and even their husbands may be unaware of their diagnosis. A man or a woman with phenylketonuria has a genetic risk of having a child with the same disease only on the 1:70 chance that

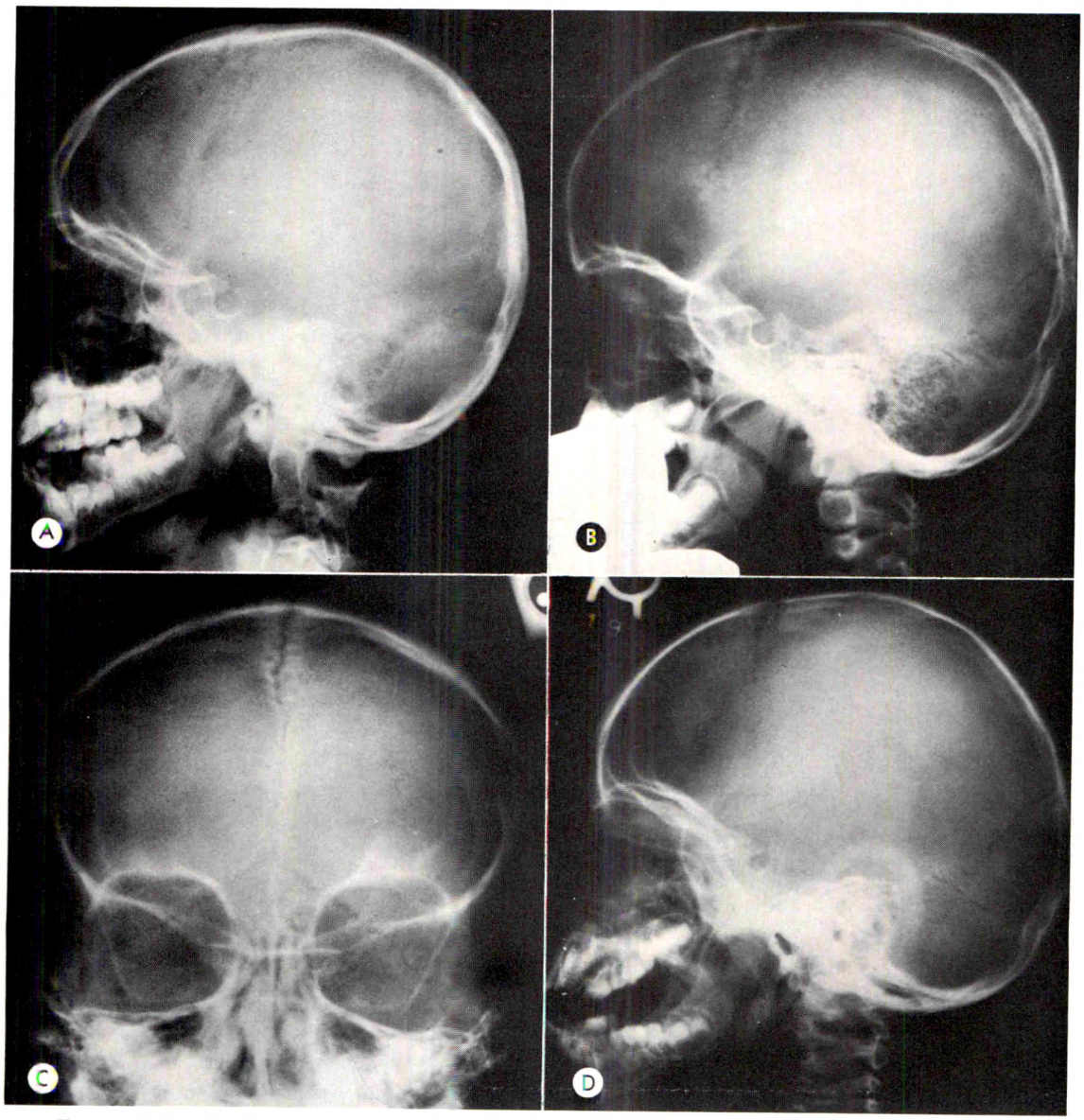


Fig. 1. *P Family*. (*A*) Boy 7 years of age. (*B* and *C*) Boy 6 years of age. (*D*) Boy 3 years of age. Note that the convolutional markings are absent.

the mate is a heterozygote carrier of PKU (autosomal recessive). Yet the metabolic risk to *all* children of PKU mothers is very great and the extent of this increasing problem is not yet widely appreciated. On review of the literature it was found that 68 of 69 children born of PKU mothers were mentally retarded,[11] whereas Reed and Reed's study[8] of unselected retarded mothers showed that only 16.7 per cent had mentally retarded offspring.

The placenta normally functions to maintain amino acid levels in the fetus higher than those in the mother. This is a conservation measure designed to ensure the fetus adequate amounts of normal amino acids, the essential "building blocks" for protein. Thus phenylalanine is actually transported across the placenta with nearly twice the concentration in the umbilical

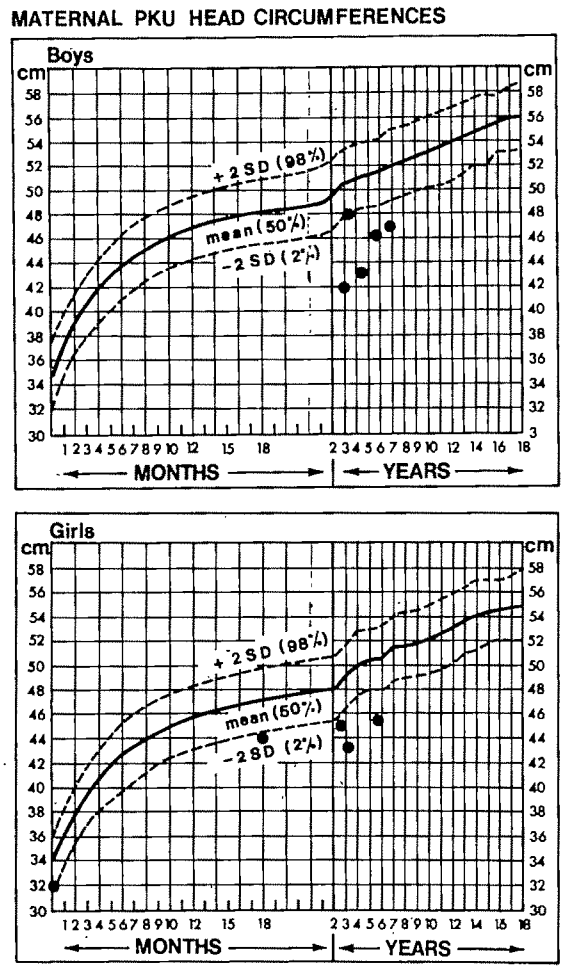


FIG. 2. *Head Circumferences.* All patients (black dots) are at least 2 standard deviations below the expected mean for their age.

cord blood as compared to maternal blood.[2,4] In the case of PKU mothers, whose phenylalanine levels are already 5 to 7 times normal, these levels may be further increased in the infant, with serious results.

In typical PKU, as seen in these mothers, there is a reduced activity of phenylalanine hydroxylase.[9] In both clinical and experimental situations, high phenylalanine levels cause incomplete myelination, abnormal cerebral lipids and distorted amino acid concentrations in the brain with irreversible neurologic damage.[7] Because of the very high risk of biochemical damage to the fetus, maternal phenylketonuria can be a valid indication for therapeutic abortion and sterilization.[5] A strict, low phenylalanine diet throughout pregnancy is the only alternative, although its success in preventing fetal abnormalities is not yet proved.[1,10]

As a result of these cases, the Guthrie test for PKU is now done on all premarital blood samples in Saskatchewan. A woman with an I.Q. of 98 but a plasma phenylalanine of 27 was recently diagnosed as PKU. She was treated with a low phenylalanine diet through the last half of her pregnancy. The infant has a coarctation of the aorta, but seems to be developing normally at 1 year.

The biochemically normal children of PKU mothers, reported in this paper, have suffered brain damage through exposure to excessive levels of phenylalanine *in utero.* They differ in a number of ways from their biochemically abnormal mothers with phenylketonuria. Their intelligence quotient is usually lower. Their bones are small but with normal architecture, without the metaphyseal changes described in PKU. Unlike PKU, there is a greatly increased incidence of spontaneous abortions, stillbirths and congenital abnormalities (Table 1).[10] Unlike PKU, no dietary treatment is needed after birth.

These children are a tragic demonstration of chemistry gone wrong. It is obvious that the mere exposure to abnormal levels of phenylalanine *in utero* is sufficient to cause life-long impairment of growth and development, particularly of the brain.

SUMMARY

Three women with phenylketonuria have produced 3 infants with lethal congenital anomalies and 10 biochemically normal but mentally retarded children.

These children, exposed to abnormal levels of phenylalanine *in utero*, have life-long impairment of growth and development. Decreased brain growth results in absent or greatly diminished convolutional markings and sometimes premature sclerosis of the sutures.

Radiologic measurement of the skull is less useful than simple measurement of head circumference.

Recognition and genetic counselling are necessary, for otherwise maternal phenylketonuria will be an increasingly common cause of mental retardation.

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REFERENCES

1. Allan, J. D., and Brown, J. K. Maternal phenylketonuria and foetal brain damage: attempt at prevention by dietary control. In: Some Recent Advances in Inborn Errors of Metabolism. Edited by K. S. Holt, and V. P. Caffey. E. & S. Livingstone, Ltd., Edinburgh, 1968, pp. 14–35.
2. Boggs, D. E., and Waisman, H. A. Influence of excess dietary phenylalanine on pregnant rats and their fetuses. *Proc. Soc. Exper. Biol. & Med.*, 1964, *115*, 407–410.
3. Davidoff, L. M. Convolutional digitations seen in roentgenograms of immature human skulls. *Bull. Neurol. Inst. New York*, 1936, *5*, 61–71.
4. Howell, R. R., and Stevenson, R. E. Offspring of phenylketonuric women. *Soc. Biol.*, 1971, *18*, Suppl. S19–S29.
5. Johnson, C. F. Phenylketonuria and obstetrician. *Obst. & Gynec.*, 1972, *39*, 942–947.
6. Lusted, L. B., and Keats, T. E. Atlas of Roentgenographic Measurement. Third edition. Year Book Medical Publishers, Inc., Chicago, 1972, pp. 30–37.
7. Menkes, J. H., and Aeberhard, E. Maternal phenylketonuria: composition of cerebral lipids in affected offspring. *J. Pediat.*, 1969, *74*, 924–931.
8. Reed, E. W., and Reed, S. C. Mental Retardation: A Family Study. W. B. Saunders Company, Philadelphia, 1965, p. 38.
9. Scriver, C. R., and Rosenberg, L. E. Amino Acid Metabolism and Its Disorders. W. B. Saunders Company, Toronto, 1973, p. 298.
10. Stevenson, R. E., and Huntley, C. C. Congenital malformations in offspring of phenylketonuric mothers. *Pediatrics*, 1967, *40*, 33–45.
11. Yu, J. S., and O'Halloran, M. T. Children of mothers with phenylketonuria. *Lancet*, 1970, *1*, 210–212.

A PATHOPHYSIOLOGIC MECHANISM FOR VENTRICULAR ENTRY OF RADIOPHARMACEUTICAL AND POSSIBLE RELATION TO CHRONIC COMMUNICATING HYDROCEPHALUS

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THE cerebrospinal fluid (CSF) imaging pattern of ventricular entry and stasis in patients with chronic hydrocephalus has been described and documented.[4,8] The cause of this ventricular "penetration" and the reason radiopharmaceutical remains there for prolonged time periods is not known. Radiopharmaceutical presence in the lateral ventricular area so soon after lumbar subarachnoid injection is too rapid to be explained by molecular diffusion.[8] Neither can it be accounted for by rapid transfer into the blood and secretion by the choroid plexus, since most of the labeled substances do not readily pass the blood-CSF barrier.

When considering the possible explanations of these phenomena, one must relate them to the generally accepted relations of production and absorption of cerebrospinal fluid. CSF appears to be produced, at least in part, by an active process, since it does not represent an ultrafiltrate of blood. The site of greatest CSF production is in the ventricular system. The choroid plexus seems mainly responsible for its secretion, although a significant contribution may be made by the ventricular ependyma.[2]

Absorption of CSF in some species such as dogs is possible by the arachnoid villi of the spinal cord, the optic and otic tracts, through lymphatic pathways and other unidentified secondary sites.[7,15] However, in the human and subhuman primate the major site of absorption appears to be the arachnoid villi in the parasagittal region.[16,17] Alternative pathways may account for some absorption in the normal and appear to represent the major pathways if the arachnoid villi are unavailable or in some manner physiologically impaired.[10,11,14,17] Thus, if the bulk of cerebrospinal fluid is formed in the ventricles and absorbed over the cerebral cortex, there is a resultant net flow of CSF out of the ventricles. This flow and the anatomic arrangement of the foramina of the fourth ventricle as suggested by Di Chiro seem reasonable explanations for radiopharmaceutical not entering the ventricles in normal patients.[4]

DESCRIPTION OF MECHANISM

When the pathways of CSF flow between the ventricles and the arachnoid villi are either obstructed or obliterated, normal absorption of CSF at these sites cannot occur and movement of CSF and its constituents from the ventricles outward is diminished. We have reconstructed this circumstance in an experimental model of chronic communicating hydroceph-

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alus.[7,9,10,16,17] From these data and correlation with the appearance of the cisternograms, as well as clinical and physiologic observations, we propose that the following events are important in the development of communicating hydrocephalus and are correspondingly reflected by the cisternographic changes observed.[10,18] Initially, even though there is reduced absorption of CSF by the arachnoid villi, production is not diminished.[2,6,12] This relative overproduction of CSF causes a slow and intermittent increase in pressure in both the ventricles and the communicating subarachnoid space surrounding the brain.[9] At the same time, histologic and ultrastructural studies have shown enlargement of the extracellular space in the periventricular region suggesting edema[11,14] and perivascular edema selectively.[10,16] The brain, being a viscoelastic substance, resists these forces, but is somewhat compliant due to the presence of cerebral veins.[6] The small periventricular cerebral veins and capillaries may collapse as their critical closing pressure is reached or the lumen may narrow sufficiently to fail to support adequate blood flow resulting in a combination of pressure and ischemic damage initially localized to the periventricular white matter.[10,11,14,16] Following these changes, the lateral ventricles have been shown to enlarge (Fig. 1*A*), especially at their dorsolateral angles. These changes have been observed in acute models for noncommunicating hydrocephalus, but are also present in the chronic experimental model for communicating hydrocephalus, but are not as striking initially. With ventricular dilatation in all forms of hydrocephalus, the ependyma loses its normal cuboidal pattern in the stretched regions, becoming flattened with loss of cilia or even entirely denuded at areas of greatest stress (Fig. 1*B*).

With these morphologic changes, the ventricular ependyma no longer acts as a partial barrier to the larger molecules in the CSF. Larger molecules (such as albumin) in the CSF pass into the brain parenchyma, not by diffusion as in the normal brain,[13] but by facilitated diffusion (bulk flow). Studies of direct ventricular infusion of I^{131} serum albumin in normal animals and those with chronic communicating hydrocephalus (to be subsequently reported) demonstrate patterns easily differentiated by plotting radioactivity *versus* distance from the ventricular ependymal surface on probability paper.[13] Gross and semiquantitative autoradiography (Fig. 1, *C* and *D*) performed in our laboratory have also confirmed these differences in movement.[16]

As the ventricles enlarge, flattening and destruction of ependymal cells continue, increasing the loss of integrity of the ependymal layer and widening of the space between supporting cells which apparently facilitates transependymal movement. The pathologic changes lead to an increase in the periventricular extracellular space[13,14,17] (Fig. 2) initially and at greater depth in the brain parenchyma in more chronic models.

After an unspecified period of time "compensation" occurs, probably because alternative pathways for absorption of CSF are established and ventricular enlargement ceases.[11,13,14] However, this stable circumstance is not so rapidly achieved if the cranial sutures are not fused. This is probably due to the fact that the calvarium does not act as a permanent resistance to continued enlargement of the ventricles. Prior to sutural closure, marked ventricular dilatation will be seen, often with only minimal neurologic deficit in both children and in very young animals.[5]

This proposed sequence of events correlates with the changes noted on cisternographic images. With ventricular enlargement and transependymal migration of CSF, the net flow out of the ventricles may be reduced and could be ultimately reversed resulting in radiopharmaceutical penetration. Since the ventricles may not participate in CSF absorption, after radiopharmaceutical enters the ventricles from the cisterna magna, it will pass across the

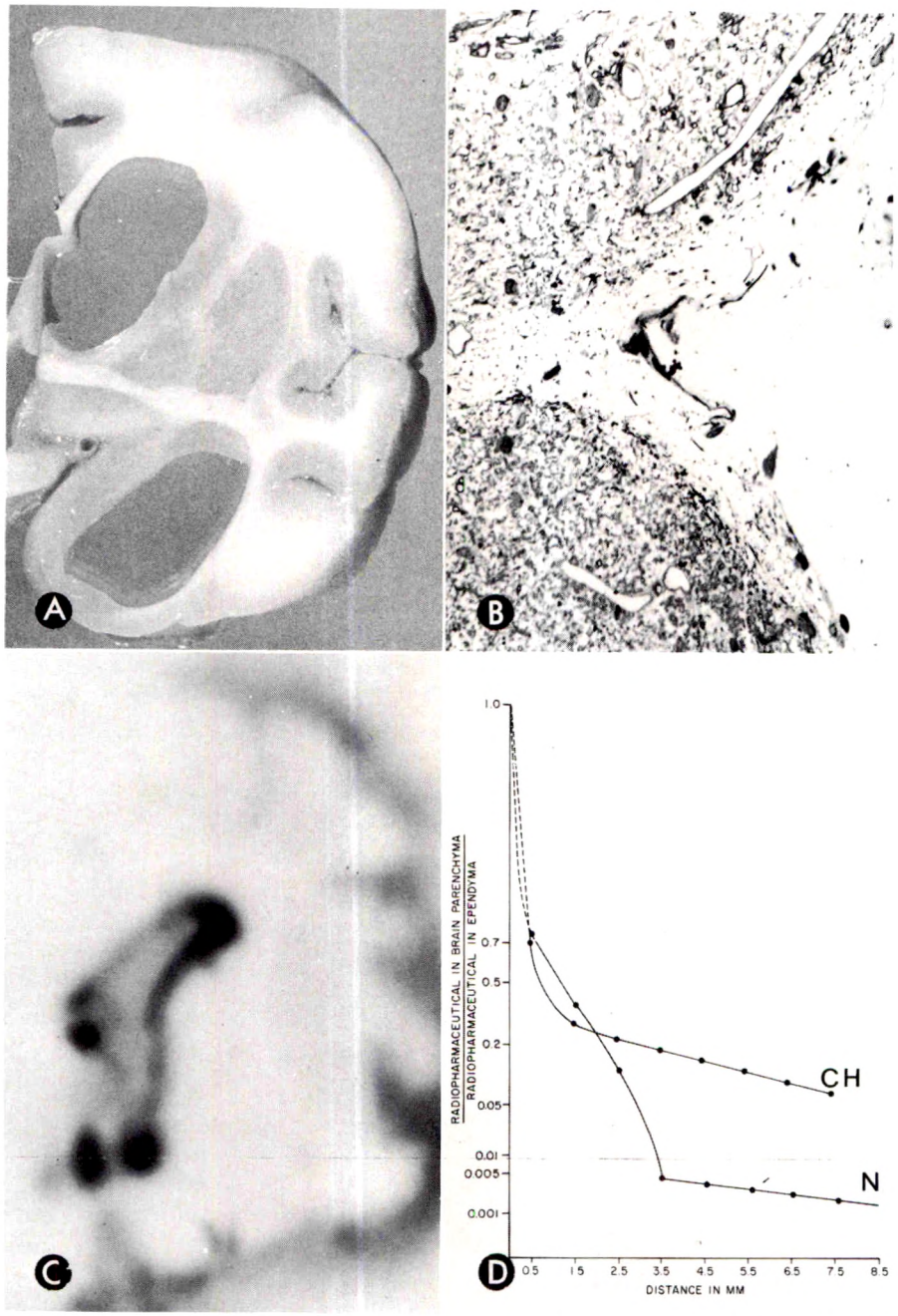


FIG. 1. (*A*) Coronal section of primate (Macca mulatta) brain 83 days after silastic injection into the peripheral subarachnoid space. Cisternograms revealed ventricular entry and stasis. Marked lateral ventricular enlargement is present, mainly at the expense of white matter. The most dramatic changes have occurred in the area of the ventricular angles. (*B*) Histologic appearance of the ventricular angle, Azure II and methylene blue stain, 550×. The ventricle is on the right. The upper left portion of the field is the corpus callosum and the lower left is the caudate nucleus. Ventricular ependyma, which is normally cuboidal, is flattened and, in the area of the angle, denuded. Changes of widened periventricular intercellular spaces and perivascular edema are present. (*C*) Gross autoradiograph, coronal section made after intraventricular placement of 100 uc of I^{131} serum albumin and sacrifice by vascular perfusion 1 hour later. Radioactivity is seen in the periventricular area, especially at the ventricular angles. No radioactivity is seen in the ventricle due to clearing during the fixation process. (*D*) Curves of quantitation of radioactivity per millimeter

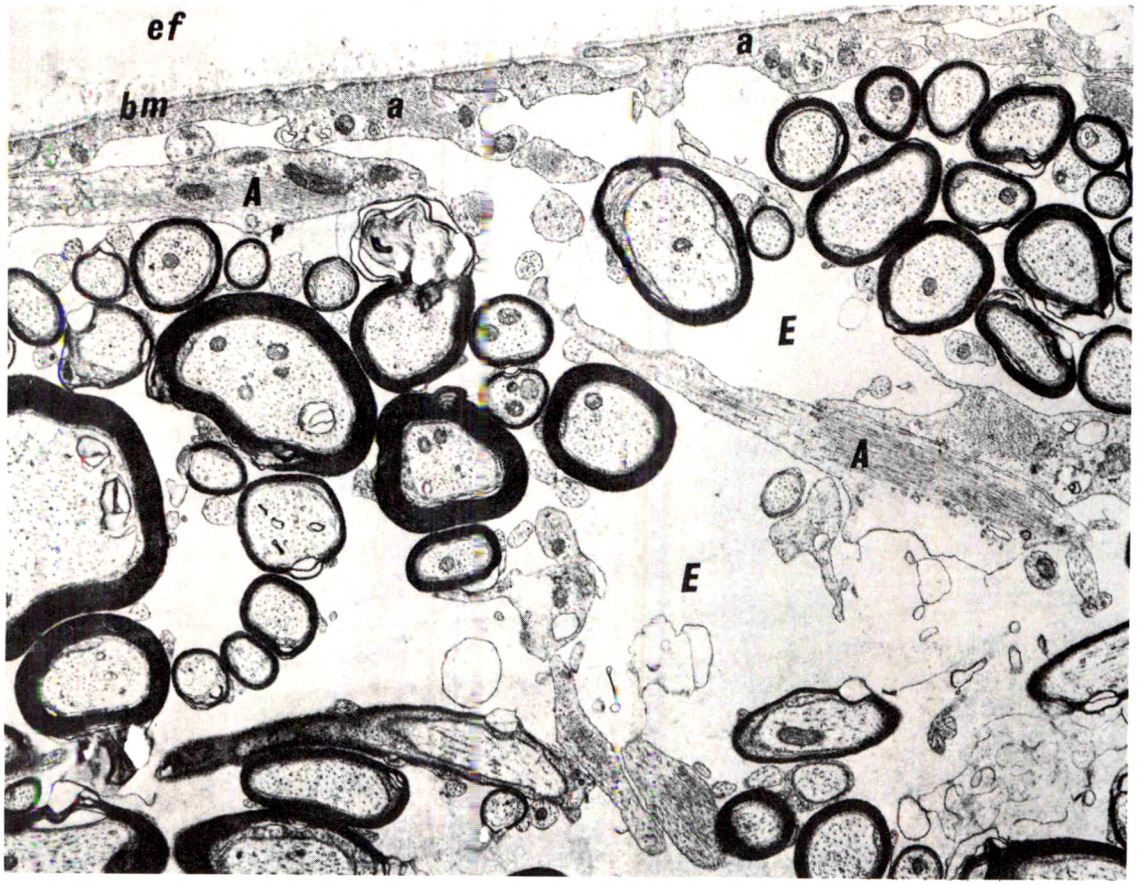


Fig. 2. Ultrastructural appearance of the periventricular area (7,874×) following perfusion with Karanofsky's formaldehyde-glutaraldehyde fixative, postfixation in osmium tetroxide, and embedding in Epon 812. In this area of the corpus callosum, intercellular edema fluid (E) and reactive astrocyte processes (A) containing numerous glial filaments are present. The regular arrangement of perivascular astrocyte end feet (a) is preserved in this figure despite the presence of perivascular edema fluid (ef) dissecting through the basement membrane (bm).

ventricular ependyma and is imaged in this enlarged CSF space for prolonged periods of time (stasis). The radiopharmaceutical does not flow out over the cerebral cortex because the normal route to CSF absorption at the parasagittal arachnoid villi has been occluded or restricted. These explanations are supported by gross and microscopic autoradiographs showing progressive penetration of labeled albumin in the brain parenchyma from the ventricular surface outward in an animal with chronic communicating hydrocephalus[16,18] (Fig. 3). In patients with ventricular "stasis" in delayed views, "apparent" ventricular enlargement occurs on the images, probably reflecting transependymal migration in the clinical circumstance (Fig. 4, *A–C*). It is

distance from the ventricular ependyma (plotted on probability paper). In normal animals the curve is almost straight and deflected slightly downward. After a distance of 3.5 mm. from the ependymal surface, the amount of radioactivity approaches background. In animals with chronic communicating hydrocephalus the curve is deflected upward, suggesting facilitated diffusion into the periventricular area. Even at a distance of 7.5 mm. from the ventricular ependyma, significant radioactivity is present reflecting penetration into the periventricular area.

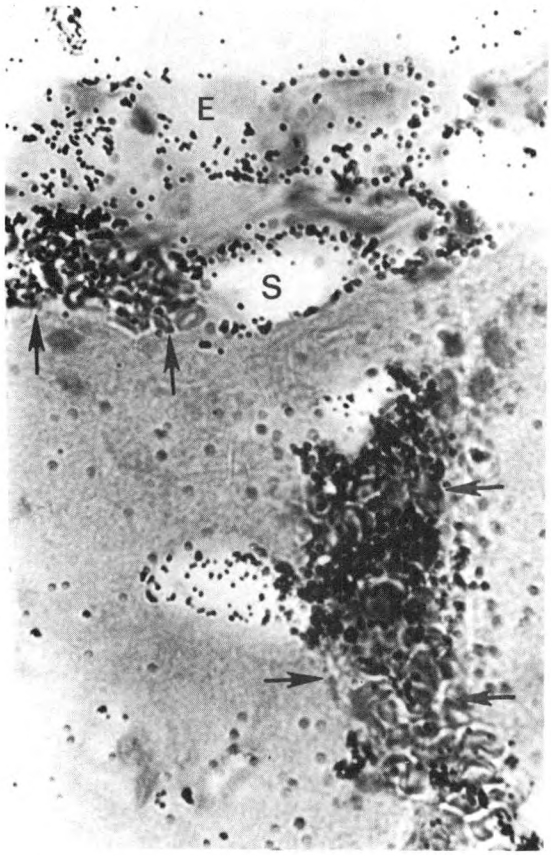


FIG. 3. Histologic autoradiograph of lateral ventricular ependymal and periventricular area in animal with chronic communicating hydrocephalus following injection of I^{131} serum albumin (100 uc) into the cisterna magna. The animal was sacrificed 1 hour later by intravascular perfusion at physiologic pressures. Normal cuboidal ependymal cells are flattened (E); acellular areas (S) containing and surrounded by Ag granules are present. Small cerebral vessels (arrows) in the periventricular area contain radioactivity as reflected by the Ag grains.

recognized, however, that alternative explanations for the apparent increase in the area of radioactivity include radiopharmaceutical decay and disassociation of the label.

CONCLUSION

Thus, the CSF image patterns reflect the pathophysiologic changes in CSF flow and reabsorption which probably occur in chronic communicating hydrocephalus. Several other established methods might be utilized to test this hypothesis including ventriculo-cisternal perfusion and ventricular infusion of histochemical markers. Ventriculo-cisternal perfusion will give accurate information regarding over-all CSF production and absorption, but will not delineate where these alterations are occurring anatomically. Histochemical markers are excel-

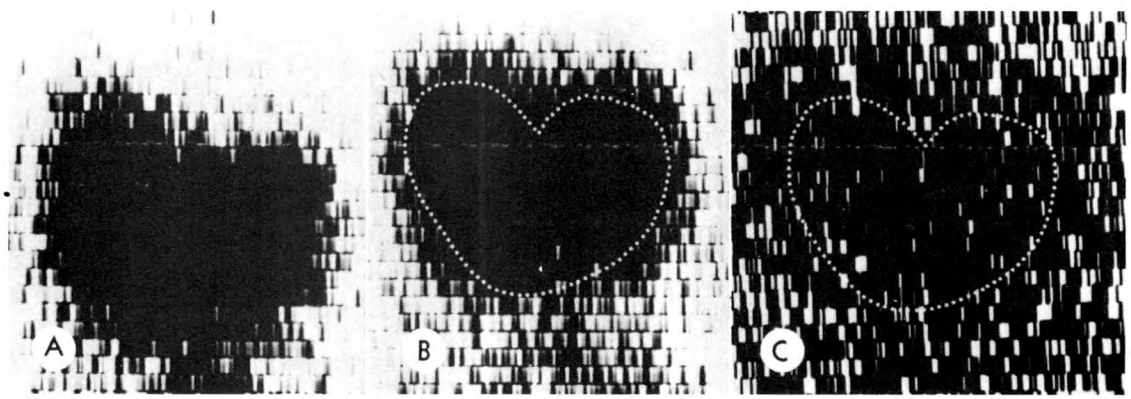


FIG. 4. (*A–C*) Composite of anterior views at 2, 6 and 24 hours in patient with chronic communicating hydrocephalus of the "normal pressure" type. (Same instrument and technical factors utilized following injection of 1 mc Yb^{169} DTPA.) The area of radioactivity in the lateral ventricle at 2 hours was traced and superimposed over (white lines) radioactivity in the same area at 6 and 24 hours. (The same appearance has been noted with I^{131} HSA, In^{111} DTPA and Tc^{99m} HSA.) The change in area can be accounted for by: (a) increased photon scatter; (b) dissociation of radionuclide and chelate; and (c) transependymal migration of the radiopharmaceutical. Experimental data accumulated in our laboratory suggest that (c) applies in this circumstance.

lent means of determining the integrity of endothelial cell junctions and physiologic barriers, but their molecular size is different from albumin and no *in vivo* image can be obtained for correlation. For these reasons, we utilized cisternographic images, and correlated them with gross and semiquantitative autoradiography for these determinations. Further extension of these and other studies utilizing this or another model for chronic communicating hydrocephalus seems indicated to elucidate the basic mechanisms of CSF production and absorption in normal and abnormal circumstances.

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REFERENCES

1. Bering, E. A., Jr., and Sato, O. Hydrocephalus: changes in formation and absorption of cerebrospinal fluid within cerebral ventricles. *J. Neurosurg.*, 1963, *20*, 1050–1063.
2. Davson, H. Physiology of the Cerebrospinal Fluid. J. & A. Churchill Ltd., London, 1970, p. 348.
3. DeLand, F. Unpublished data. Presented at Society of Nuclear Medicine, Los Angeles, 1972.
4. Di Chiro, G. Cisternography: from early tribulations to useful diagnostic procedure. The Gould Lecture. *Johns Hopkins Med. J.*, 1973, *133*, 1–15.
5. Epstein, F. Personal communication.
6. Hakim, S. Biomechanics of hydrocephalus. In: Cisternography and Hydrocephalus. Chapter III. Charles C Thomas, Publisher, Springfield, Ill., 1972, pp. 25–55.
7. James, A. E., and Strecker, E. P. Use of silastic to produce communicating hydrocephalus. *Invest. Radiol.*, 1973, *8*, 105–110.
8. James, A. E., Jr., New, P. F. J., Heinz, E. R., Hodges, F. J., III, and DeLand, F. H. Cisternographic classification of hydrocephalus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 39–49.
9. James, A. E., Strecker, E. P., Novak, G., and Burns, B. Correlation of serial cisternograms and cerebrospinal fluid pressure measurements in experimental communicating hydrocephalus. *Neurology*, 1973, *23*, 1226–1233.
10. James, A. E., Strecker, E. P., Sperber, E., Flor, W. J., Merz, T., and Burns, B. Alternative pathway of cerebrospinal fluid absorption in communicating hydrocephalus. *Radiology*, 1974, *111*, 143–146.
11. Levin, V. A., Milhorat, T. H., Fenstermacher, J. D., Hammock, M. K., and Rall, D. P. Physiological studies on development of obstructive hydrocephalus in monkey. *Neurology*, 1971, *21*, 238–246.
12. Lorenzo, A. V., Page, L. K., and Watters, G. V. Relationship between cerebrospinal fluid formation, absorption and pressure in human hydrocephalus. *Brain*, 1970, *93*, 679–692.
13. Rall, D. P., Oppelt, W. W., and Patlak, C. S. Extracellular space of brain as determined by diffusion of inulin from ventricular system. *Life Sc.*, 1962, *1*, 43–48.
14. Sahar, A., Hochwald, G. M., and Ransohoff, J. Alternative pathway for cerebrospinal fluid absorption in animals with experimental obstructive hydrocephalus. *Exper. Neurol.*, 1969, *25*, 200–206.
15. Shurr, R. H., McLaurin, R. L., and Ingraham, F. D. Experimental studies on circulation of cerebrospinal fluid and methods of producing communicating hydrocephalus in dog. *J. Neurosurg.*, 1953, *10*, 515–525.
16. Strecker, E. P., James, A. E., Konigsmark, B., and Merz, T. Albumin flow and absorption in experimental communication hydrocephalus: autoradiographic observations. *Neurology*, 1974, *22*, 192–197.
17. Strecker, E. P., James, A. E., Kelley, J. E. T., and Merz, T. Semiquantitative studies of transependymal albumin movement in communicating hydrocephalus. *Radiology*, 1974, *111*, 341–346.
18. Strecker, E. P., Scheffel, E., Kelley, J., and James, A. E. CSF absorption in communicating hydrocephalus: evaluation of transfer of radioactive albumin from subarachnoid space to plasma. *Neurology*, 1973, *23*, 854–864.

MALPOSITION OF THE ATTACHED PORTION OF THE FALX CEREBRI AND THE SUPERIOR SAGITTAL SINUS*

AN INDICATOR OF SEVERE CEREBRAL MALDEVELOPMENT

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THE attached portion of the falx cerebri and the superior sagittal sinus contained within it are normally fixed midline structures. Although the importance of the position of the tentorium and of the configuration of the transverse and straight sinuses has been documented,[14] there is minimal information in the literature referable to anomalous location of the falx and superior sagittal sinus.

Only 1 previous case report of displacement of these structures was found in the literature.[11] We have identified malposition of the attached portion of the falx and superior sagittal sinus in 28 patients.

The purpose of this communication is to define the embryologic basis and prognostic implication of this malposition, to show which conditions result in this change and to give identifying features that can be demonstrated by various forms of roentgenologic examination (plain skull roentgenography, pneumography, ventriculography, angiography and isotope encephalography).

MATERIAL AND METHOD

This study is based upon the review of roentgen studies of all patients with hydranencephaly (18 cases), congenital porencephaly (19 cases), absence of the corpus callosum with interhemispheric cyst (5 cases), and congenital asymmetric cerebral hypoplasia (10 cases), that have been studied at Primary Children's Medical Center between 1962 and June, 1973. The congenital asymmetric cerebral hypoplasia group includes 5 cases each with unilateral hypoplasia and bilateral hypoplasia with asymmetric involvement.

Each of these 52 reviewed cases has been studied with routine skull roentgenography and intracranial pneumography (nitrous oxide encephalography or air ventriculography). In addition, 2 cases have been studied by cerebral angiography and 3 patients by isotope encephalography. Postmortem examination was performed in 1 case.

We have not identified displacement of the attached portion of the falx and the superior sagittal sinus in other brain anomalies.

RESULTS

Malposition of the attached portion of the falx cerebri and the superior sagittal sinus was identified in 28 of the 52 reviewed cases. The roentgen changes are summarized in Table 1.

Routine skull roentgenograms may demonstrate the site of an anomalously positioned superior sagittal sinus or the site of the attachment of the falx cerebri to the overlying calvarium. There may be: (1) localized thickening of the calvarium; (2) a groove indenting the inner surface of the calvarium; or (3) flattening of a portion of the calvarial configuration.

The calvarial thickening is triangular in configuration with its apex directed centrally (Fig. 1; 3*A*; and 4*B*). This localized internal thickening indicating the site of the attachment of the falx occurred in 6 of 18 cases of hydranencephaly, 5 of 19 cases of congenital porencephaly and 1 of 10

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TABLE I

ROENTGEN CHANGES IN MALPOSITION OF THE ATTACHED PORTION OF THE FALX CEREBRI AND THE SUPERIOR SAGITTAL SINUS

	Total	Hydranencephaly	Congenital Porencephaly	Congenital Asymmetric Cerebral Hypoplasia	Absent Corpus Callosum with Interhemispheric Cyst
No. of Cases	52	18	19	10	5
I. Plain skull roentgenographic findings					
A. Internal groove	7/52	0/18	3/19	2/10	2/5
B. Internal thickening	12/52	6/18	5/19	1/10	0/5
C. External flattening	12/52	8/18	1/19	2/10	1/5
II. Correlation of plain roentgenograms and gas studies					
A. Plain skull evidence of falx displacement	23/52	10/18	7/19	3/10	3/5
B. Falx off midline on gas studies	28/52	12/18	9/19	3/10	4/5

cases of congenital asymmetric hypoplasia.

The internal groove in the calvarium, demarcating the superior sagittal sinus anomalously positioned off the midline, was identified less frequently (Fig. 2; and 5, *A* and *B*). A groove was identified in 3 cases of congenital porencephaly, 2 cases of congenital asymmetric hypoplasia and 2 of 5 cases with absence of the corpus callosum with interhemispheric cyst. A groove was not identified in cases of hydranencephaly.

Often a more diffuse alteration in the configuration of the parietal and occipital bones denoted the side of the malpositioned falx. Flattening or slight concavity of the external margin of the calvarium occurred rather than the normal convexity (Fig. 1; and 3*A*). This finding occurred in 8 of the cases of hydranencephaly, 2 patients with congenital unilateral hypoplasia, and 1 case each with congenital porencephaly and

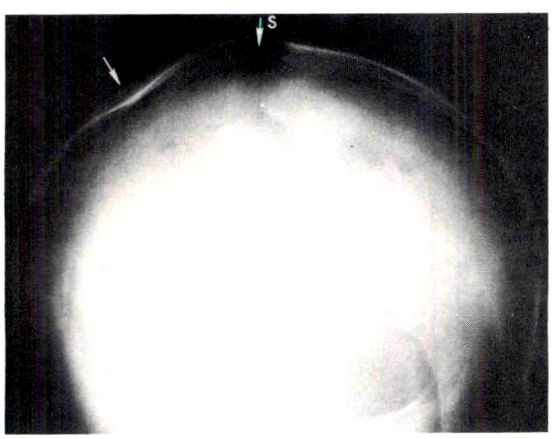


FIG. 1. Focal thickening of right parietal bone (arrow) indicates the site of attachment of the anomalously positioned falx. There is considerable displacement from the sagittal suture (S). There is also mild flattening of the configuration of the calvarium in the region of the falx attachment site.

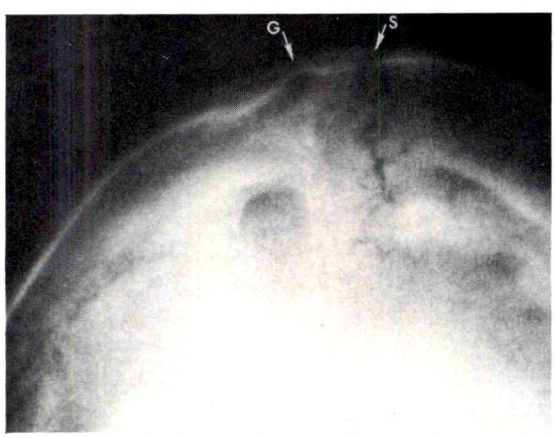


FIG. 2. The vascular groove (G) denotes the malpositioned superior sagittal sinus in relationship to the sagittal suture (S) in this Towne's projection.

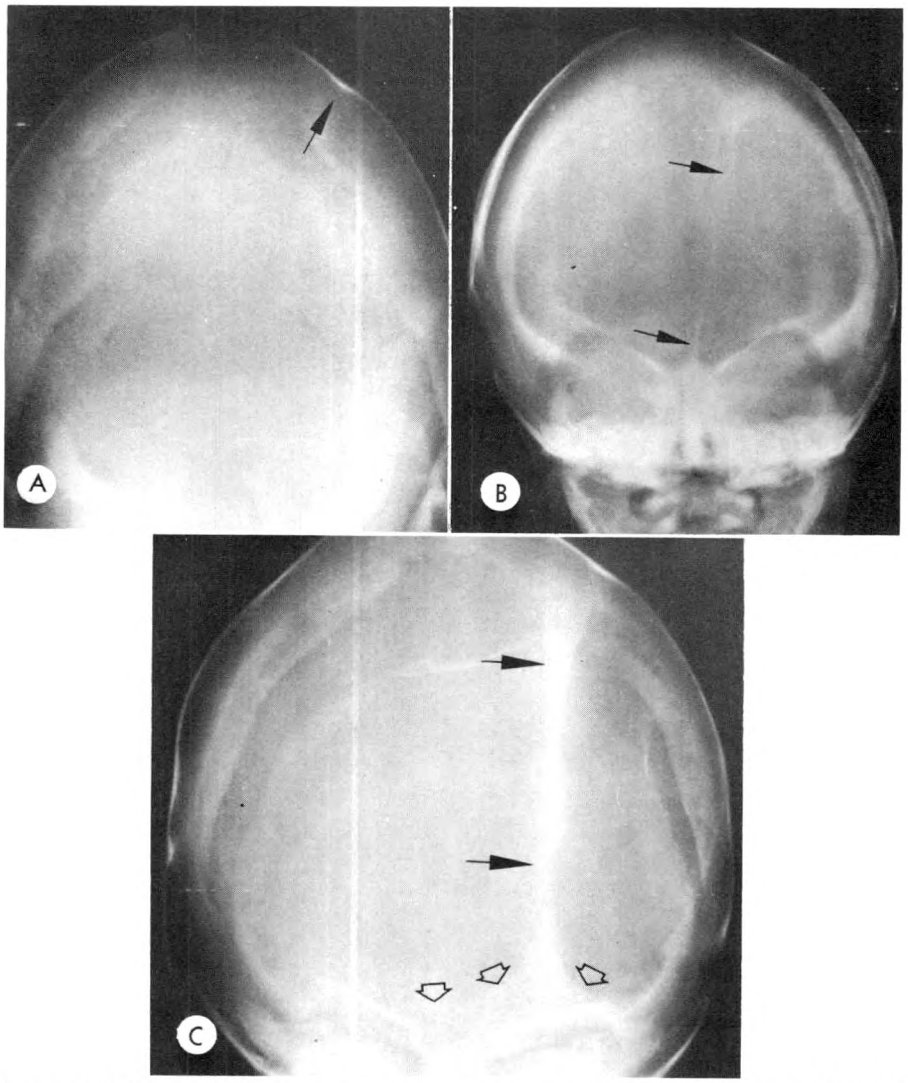


FIG. 3. Hydranencephaly in an infant. (*A*) Towne's position roentgenogram shows focal calvarial thickening and flattening (arrow). (*B*) Anteroposterior brow-up pneumogram shows a normal attachment site of the anteroinferior portion of the falx (lower arrow) to the crista galli. There is progressive displacement of the falx posteriorly (upper arrow). (*C*) Posteroanterior brow-down pneumogram demonstrates continued increase in the degree of falx (black arrows) displacement in its more posterior portion. The falx is seen extending laterally into the tentorium (open arrows).

absence of the corpus callosum with interhemispheric cyst.

Although the falx attachment site could usually be identified on both anteroposterior and Towne's projection roentgenograms, the findings were sometimes more prominent in the Towne's view (Fig. 3, *B* and *C*). Occasionally, the malposition could only be identified in this projection. In only 5 patients did plain skull roentgenograms fail to give evidence of malposition of the falx. This occurred in 2 patients with hydranencephaly, 2 patients with congenital porencephaly, 1 patient with absence of the corpus callosum with interhemispheric cyst, and 1 patient with asymmetric cerebral hypoplasia.

Displacement of the attached portion of the falx cerebri and superior sagittal sinus was confirmed with gas studies in all cases

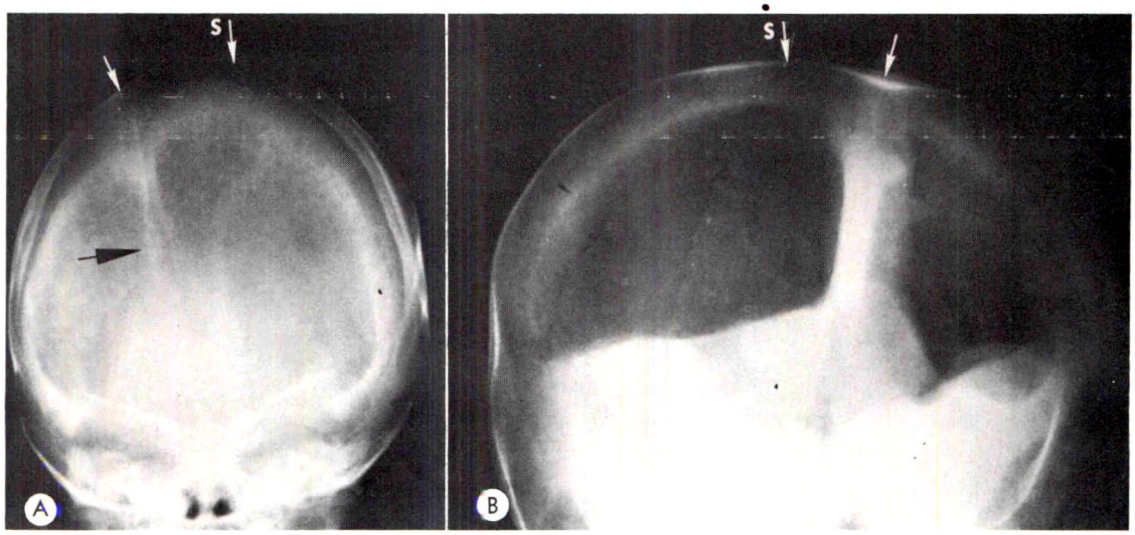


Fig. 4. Pneumographic demonstration of malposition of the falx. (*A*) In this case of hydranencephaly the basilar attachment is midline, but the more posterior portions (arrows) are malpositioned away from the sagittal suture (S). (*B*) Mild displacement of the falx from the sagittal suture (S). Focal thickening of the calvarium at the falx attachment site (arrow).

(Fig. 3, *B* and *C*; and 4, *A* and *B*). Demonstration of displacement of the superior sagittal sinus was also demonstrated by cerebral angiography in 2 cases (Fig. 5, *A* and *B*) and isotope encephalography in 3 cases (Fig. 6, *A* and *B*). The location of the attached portion of the falx and superior sagittal sinus, as identified on plain film skull roentgenograms, correlated well with the contrast and isotope studies. Malposition of the superior sagittal sinus was demonstrated with transillumination in a few cases (Fig. 7).

The degree of displacement of the super-

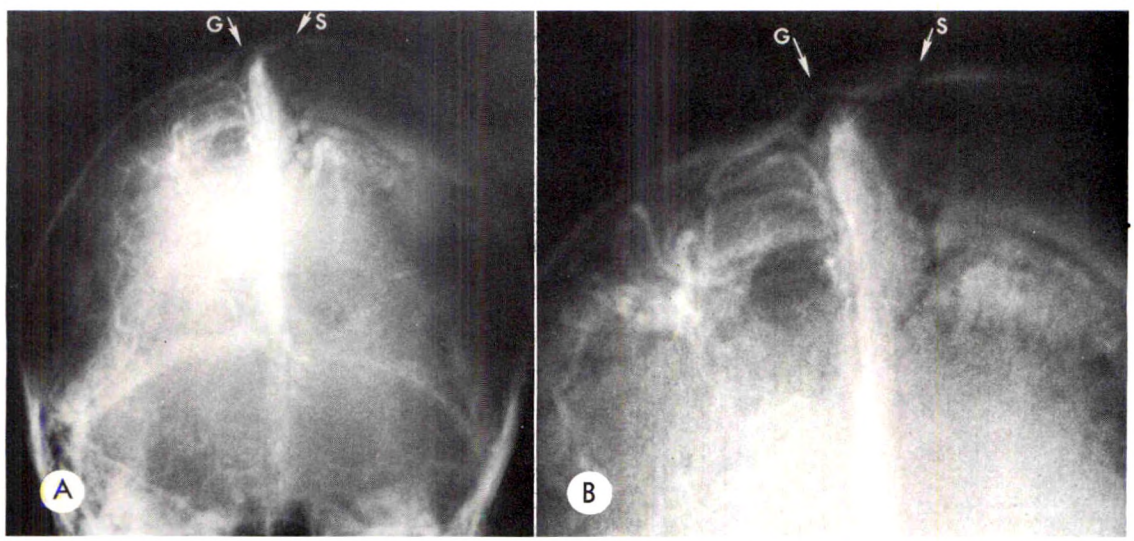


Fig. 5. (*A*) Venous phase of cerebral angiography showing anomalous position of the parietal portion of the superior sagittal sinus (G) from the sagittal suture (S). A groove denotes the sinus location. (*B*) Close-up of the same case.

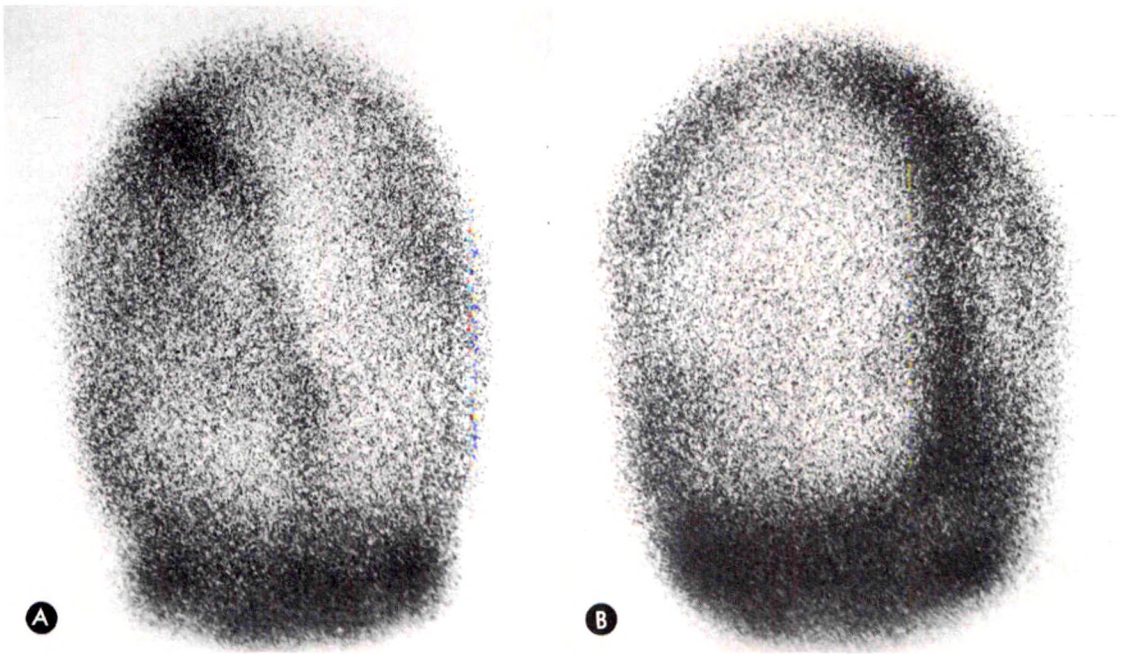


FIG. 6. Isotope encephalograms demonstrating malposition of the falx and superior sinus. (*A*) The anterior view demonstrates the basilar portion of the falx to be normal in position. Progressive shift occurs posteriorly. (*B*) The posterior view shows marked shift of the posterior falx and its asymmetric connection with the tentorium. (Courtesy of Dr. Philip R. Frederick.)

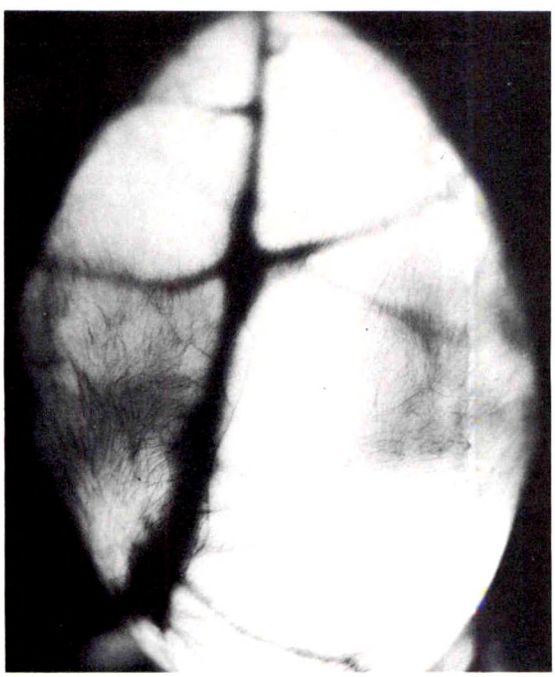


FIG. 7. Transillumination demonstrating malposition of the superior sagittal sinus. The shift is greater posteriorly (lower portion of photograph).

ior sagittal sinus and attached falx from the sagittal suture varied from 0.5 cm. to 5.0 cm. The displacement was less than 2 cm. in most cases (Table II).

Postmortem examination confirmed the anomalous location of the attached portion of the falx and the superior sagittal sinus in 1 case (Fig. 8). The degree of shift in this autopsied case was the least (0.5 cm.) of any reported in this series. Skull roentgenograms were normal in this case. The malposition demonstrated on pneumography correlated well with the autopsy findings.

EMBRYOLOGY

The development of the superior sagittal sinus in the human fetus was elucidated by Mall[8] and Streeter[12] in the early 1900s. Their studies indicate that as the primitive telencephalon differentiates into the cerebral hemispheres, a plexus of veins forms on its dorsal surface. At the 21 mm. stage (8 weeks),[1] a venous pattern similar to that seen in the adult is recognizable, with a

TABLE II

DEGREE OF DISPLACEMENT OF THE ATTACHED PORTION OF THE FALX AND THE SUPERIOR SAGITTAL SINUS IN 28 CASES

Distance Displaced	Total (28 cases)	Hydranencephaly	Congenital Porencephaly	Congenital Asymmetric Cerebral Hypoplasia	Absent Corpus Callosum with Interhemispheric Cyst
0.5–1 cm.	11	5	3	2	1
1–2 cm.	14	6	5	1	2
2–3 cm.	1	0	1	0	0
over 3 cm.	2	1	0	0	1

longitudinal channel present which becomes the anterior portion of the superior sagittal sinus. The developing cerebral hemispheres become separated by mesenchymal tissue which forms the falx cerebri.[5] The posterior portion of the superior sagittal sinus develops through elongation as the tentorium descends and the developing cerebral hemispheres extend posteriorly. By the 50 mm. stage (11 weeks), the superior sagittal sinus is relatively well defined and its position is similar to the adult pattern. It is implied that attachment between the falx cerebri and developing calvarium occurs at approximately this same time.[3]

The roentgen demonstration of displacement of the attached portion of the falx cerebri indicates that the primary disease process occurred very early during fetal life. This process can be either unilateral or bilateral with initial asymmetric involvement. After the dura becomes attached to the overlying bony precursor structures, the relationship of the attached portion of the falx and the calvarium remains stable. The embryologic studies noted above suggest that this occurs prior to the twelfth week of intrauterine development. Since the posterior portion of the falx attaches last, this portion could be expected to be displaced more frequently and to a greater extent than the anterior portion. This has been noted in our cases.

Other anomalies of the cerebrum and meninges have been common in the absent corpus callosum with interhemispheric cyst[13] and porencephaly[10] groups of patients. There is experimental and embryologic evidence that the stage of fetal devel-

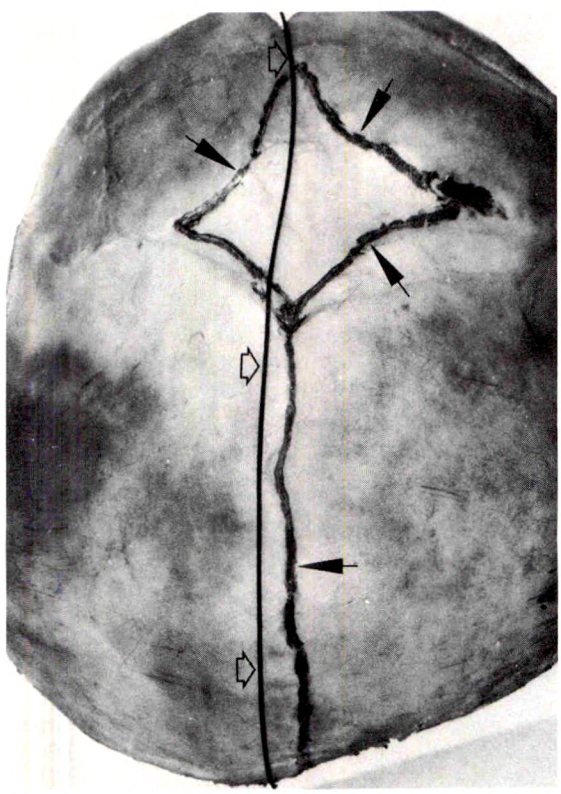


FIG. 8. A portion of the calvarium of an autopsy case in which the superior sagittal sinus and attached portion of the falx were mildly shifted. The relationship of the sagittal suture and anterior fontanelle (black arrows) to the falx attachment location (open arrows) is demonstrated. The malposition is greater posteriorly.

opment during which many of these malformations (absence of the corpus callosum, heterotopic gray matter, aqueduct stenosis, ventricular dilatation, interhemispheric cyst, porencephaly and failure of tentorium descent) occur is similar.[2,6,7,10,13,14] These anomalies probably all develop between the eighth and sixteenth weeks of intrauterine development. The association of malposition of the falx with these other anomalies suggests that they all develop at approximately the same time.

DISCUSSION

The plain film roentgenographic appearance at the site of the falx attachment is, in part, related to the volume of brain tissue present. In hydranencephaly and also in severe cases of porencephaly and other lesions with markedly decreased brain tissue, a groove formed by the superior sagittal sinus would be unlikely because of the small size of this vascular structure in cases which have a small volume of cerebral tissue. Localized calvarial thickening and flattening or concavity of the margin of the calvarium may be secondary to a tethering effect by the falx and disruption of normally directed forces in the "calvarium-skull base-dura mater complex."[9,15]

The roentgen pattern of malposition of the attached portion of the falx cerebri and superior sagittal sinus should not be confused with the roentgen findings of the so-called Davidoff-Dyke[4] syndrome of acquired unilateral cerebral atrophy. In those cases the disease process is atrophic rather than hypoplastic or dysplastic and develops during the latter portions of fetal life, infancy or early childhood. The position of the attached portion of the falx, of course, will be normal in these cases, since asymmetric development of the brain will not alter the relationship of this structure with the calvarium after they become conjoined.

The roentgen pattern described herein should also not be confused with deviation of the most posterior portion of the superior sagittal sinus which may deviate from the midline as a normal variant. This deviation occurs to the side of the dominant transverse sinus and is limited to the segment of the sinus attached to the occipital bone.

The clinical pattern of the cases reported in this paper would certainly be consistent with a major insult occurring early in the development of the central nervous system. Gross mental and motor abnormalities were present in all our cases, in which the attached portion of the falx was malpositioned.

The major importance in the roentgen demonstration of malposition of the falx cerebri and superior sagittal sinus is its prognostic implication of a severe underlying cerebral malformation. This should be of great aid to the clinician in management of the patient and counseling the patient's parents. Definite plain film roentgenographic findings in the proper clinical setting may make further diagnostic studies unnecessary.

SUMMARY

Malposition of the superior sagittal sinus and attached portion of the falx cerebri is a reliable indicator of a severe cerebral malformation which develops in the first trimester of fetal development. This malposition has been identified in approximately one-half of our patients with hydranencephaly, congenital porencephaly, absence of the corpus callosum with interhemispheric cyst and congenital asymmetric cerebral hypoplasia.

Plain skull roentgenography usually demonstrates changes indicating the abnormal location of these structures.

Intracranial pneumography, cerebral angiography, isotope encephalography and transillumination are methods which can confirm displacement of these structures.

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REFERENCES

1. Arey, L. B. Developmental Anatomy: A Textbook and Laboratory Manual of Embryology. Seventh edition. W. B. Saunders Company, Philadelphia, 1965, pp. 106–107.
2. Bergeron, T. R. Pneumographic demonstration of subependymal heterotopic cortical gray matter in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 168–177.
3. Blechochmidt, E. The Stages of Human Development Before Birth: An Introduction to Human Embryology. W. B. Saunders Company, Philadelphia, 1961, p. 261.
4. Dyke, G. G., Davidoff, L. M., and Masson, C. B. Cerebral hemiatrophy with homolateral hypertrophy of skull and sinuses. Am. J. Roentgenol. & Rad. Therapy, 1933, *57*, 588–600.
5. Hamilton, W. J., and Boyd, J. D. Human Embryology. Williams & Wilkins Company, Baltimore, 1962, pp. 111–332.
6. Hicks, S. P. Developmental malformations produced by radiation: timetable of their development. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *69*, 272–293.
7. Loeser, J. D., and Alvord, E. C., Jr. Agenesis of corpus callosum. *Brain*, 1968, *91*, 553–570.
8. Mall, F. P. On development of blood vessels of brain in human embryo. *Am. J. Anat.*, 1905, *4*, 1–18.
9. Moss, M. L. Pathogenesis of premature cranial synostosis in man. *Acta anat.*, 1959, *37*, 351–370.
10. Nixon, G. W., Johns, R. E., Jr., and Myers, G. G. Congenital porencephaly. To be published.
11. Smith, D. R., Kempe, L. G., and Dunaway, R. Prenatal agenesis of one cerebral hemisphere with displacement of sagittal sinus. *J. Neurosurg.*, 1969, *30*, 80–82.
12. Streeter, G. L. Development of venous sinuses of dura mater in human embryo. *Am. J. Anat.*, 1915, *18*, 145–173.
13. Swett, H. A., and Nixon, G. W. Agenesis of corpus callosum with interhemispheric cyst. To be published.
14. Wolpert, S. M. Dural sinus configuration: measure of congenital disease. *Radiology*, 1969, *92*, 1511–1516.
15. Young, R. W. Influence of cranial content on postnatal growth of skull in rat. *Am. J. Anat.*, 1959, *105*, 383–415.

CEREBRAL CALCIFICATION IN CHILDHOOD LEUKEMIA MIMICKING STURGE-WEBER SYNDROME*

REPORT OF TWO CASES

By PATRICIA F. BORNS, M.D.,† *and* LEE F. RANCIER, M.D.‡
PHILADELPHIA, PENNSYLVANIA

THE increasingly successful treatment of acute (lymphocytic) leukemia (ALL), particularly with chemotherapy,[3] brings the patient in long-term remission to the radiologist's attention more and more frequently. When these patients suffer leukemic relapse, the central nervous system is often the region of involvement and roentgenograms of the skull are obtained.

The following 2 cases are of particular interest, since they present a unique pattern of brain calcification, mimicking that seen in Sturge-Weber syndrome, associated with long-term remission of ALL.

REPORT OF CASES

CASE I. D.G. was 4½ years old when ALL was diagnosed in September, 1965. Remission was induced with vincristine and prednisone, and was successfully maintained with methotrexate, given intramuscularly. In March and again in May, 1967 the patient suffered episodes of severe central nervous system (CNS) depression with unconsciousness, although he was in remission (by bone marrow aspiration). Plain skull roentgenograms were negative. Cerebrospinal fluid (CSF) contained 1,000 mononuclear cells during the first episode. CSF pressure was elevated. Spiking fevers were noted. He received intrathecal methotrexate and a total of 550 rads Co^{60} teletherapy to the midline of the skull. He responded partially, but continued to have severe and prolonged CNS complications, including dementia. Intrathecal and intramuscular methotrexate was continued.

For the first time in January, 1968 a skull series showed a serpiginous pattern of cerebral calcification (Fig. 1, *A* and *B*). Tomograms demonstrated the calcifications to be deep-seated as well as cortical (Fig. 2). The patient had not received further radiation therapy to that time. The calcification was progressive. The marrow remained in remission and leukemic cells in the CSF were not documented.

Brain scanning and electroencephalography showed no localized lesion during his course. Radiation therapy to the skull was given again in September, 1968 and August, 1969. Methotrexate was continued. The patient died in February, 1970. Permission for autopsy was refused.

CASE II. S.S., a $3\frac{5}{12}$ year old female, was diagnosed as having ALL in April, 1966. Induction and maintenance were achieved with methotrexate. Vincristine and prednisone were also used early in her course. The first CNS relapse occurred in September, 1967, characterized by seizures. Skull roentgenograms were negative. Intrathecal methotrexate and radiation therapy to the skull were given. A total of 3,400 rads, in 5 fractionated courses of treatment, was administered between September, 1967 and October, 1968 because of increasingly severe bouts of CNS difficulty, including hemiparesis and aphasia.

Left frontal craniotomy was performed to remove a localized lesion in December, 1968. No leukemic infiltrate was found, but severe cerebral degeneration with astrocytic gliosis was present.

Wavy lines of calcification in the brain were first noted on roentgenograms obtained in March, 1969 (Fig. 3). A branch of the posterior temporal division of the middle cerebral artery closely paralleled the calcific lines on angiography (Fig. 4).

In April, 1969 a right occipital craniotomy revealed several large thrombosed cortical veins. Cortical biopsy showed cortical capillary telangiectasia with calcification, and extreme subcortical astrocytosis. There were no leu-

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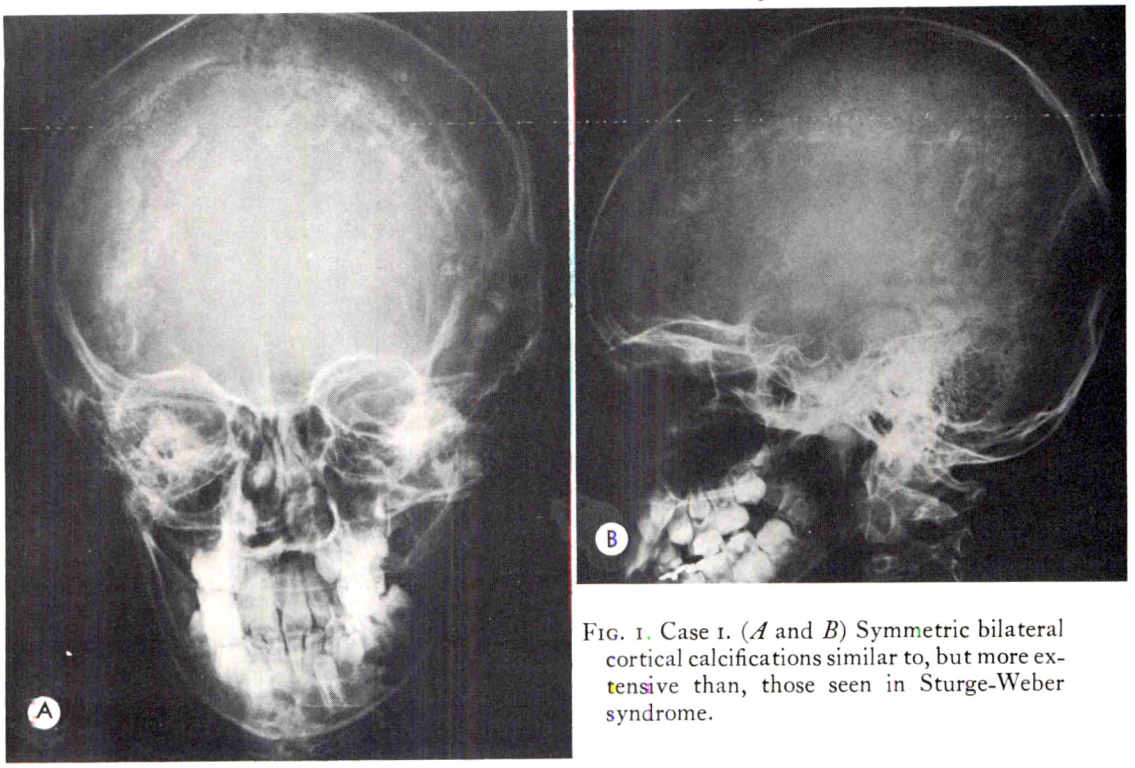


Fig. 1. Case I. (*A* and *B*) Symmetric bilateral cortical calcifications similar to, but more extensive than, those seen in Sturge-Weber syndrome.

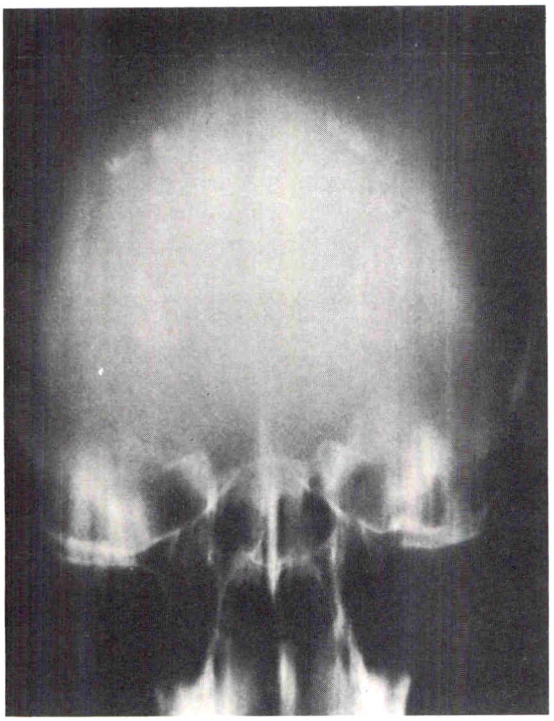


Fig. 2. Case I. Tomogram demonstrates that there are deep intracranial calcifications, probably paraventricular as well as cortical.

kemic cells. She remained in hematologic remission, and chemotherapy was discontinued in 1970. Remission has been maintained through October, 1972. Skull roentgenograms showed progression of the calcifications (Fig. 5). The child is severely retarded, although she was of normal intelligence prior to 1967.

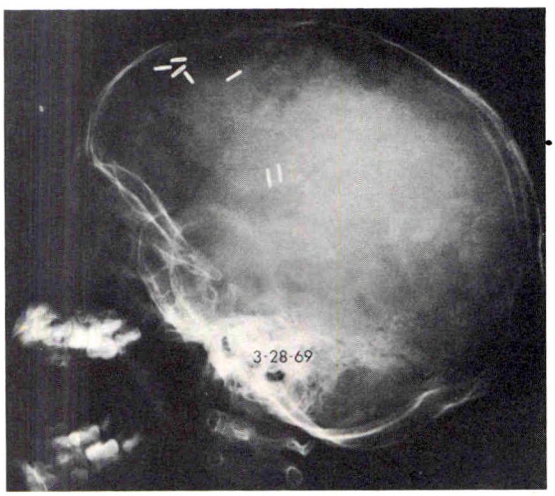


Fig. 3. Case II. First appearance of the characteristic parallel cortical calcifications.

DISCUSSION

The treatment of childhood leukemia is turning from a palliative objective towards one of cure through vigorous combined chemotherapy.[3] Both of the cases presented developed a striking pattern of cerebral calcification during treatment for ALL. Each had received radiation therapy (one received only 550 rads) and chemotherapy including intrathecal methotrexate, prior to the appearance of the calcification. Both remained in hematologic remission throughout their course.

The unique, gyriform pattern of brain calcification in these 2 patients forms the basis for this report. Sturge-Weber disease exhibits a similar pattern, although the cerebral calcification in that disease is virtually always unilateral. No portwine-colored facial nevus of Sturge-Weber was present in our 2 patients. Glioma may occasionally produce a serpiginous calcification in the brain,[2] but such a tumor was excluded by biopsy in one of the patients, and by the symmetric and extensive involvement in the other patient.

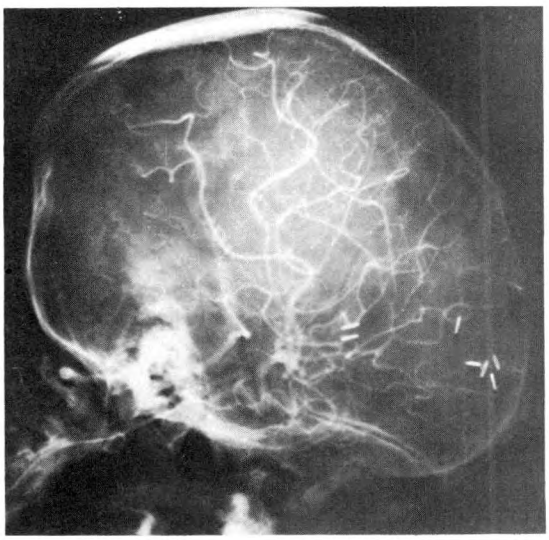


FIG. 4. Case II. Angiogram shows that a branch of the posterior temporal division of the middle cerebral artery closely parallels the calcific lines seen on the plain film roentgenogram.

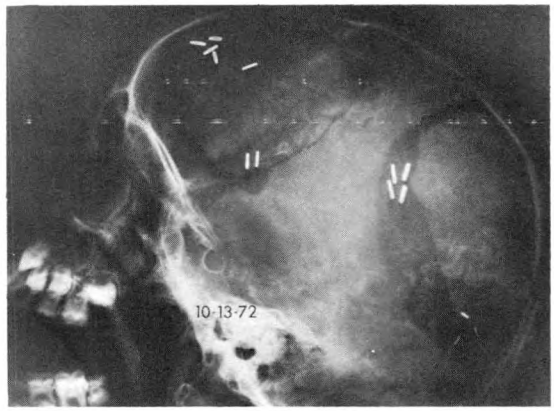


FIG. 5. Case II. The calcifications have become more extensive and more dense, although the child has remained free of leukemia and has received no further treatment.

A somewhat similar pattern of calcification was reported in a patient who had received roentgen therapy (dose unspecified) for tinea capitis.[4] The dose and fractionation of radiation therapy in these cases makes brain calcification from radiation necrosis unlikely.[1] The possibility that the chemotherapy received by these patients played a role in the brain changes remains speculative.

A careful review of the literature failed to reveal a report of similar cases. By personal communication from Dr. Noemi Cremer we have data on a similar child followed in Brussels, Belgium.[5] It is strongly suspected that such cases are occurring sporadically wherever leukemic patients are surviving for a significant length of time. We also suspect that dementia is consistently associated with this form of cortical damage.

SUMMARY

Two cases of ALL (acute lymphocytic leukemia), both in long-term remission, developed a gyriform pattern of brain calcification during treatment for CNS (central nervous system) complications, on a treatment regimen which included: prednisone, vincristine, methotrexate and irradiation.

Cerebral angiography and brain biopsy

were obtained in one. The calcium was closely related to vascular structures.

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REFERENCES

1. Bouchard, J. Radiation Therapy of Tumors and Diseases of the Nervous System. Lea & Febiger, Philadelphia, 1966.
2. DiChiro, G., and Lindgren, E. Radiographic findings in 14 cases of Sturge-Weber syndrome. *Acta radiol.*, 1951, *35*, 387–399.
3. Pinkel, D. Drug dosage and remission duration in childhood lymphocytic leukemia. *Cancer*, 1971, *27*, 247–258.
4. Valentino, V. A proposito degli effetti biologici necrotizzanti dell'irradiazione roentgen sul tessuto nervoso; riporto di un caso. Osp. Riun., Napoli. *Rif. Med.*, 1959, *73*, 28.
5. Cremer, N. Personal communication, 1973.

ROENTGEN FINDINGS OF PSEUDO-HURLER POLYDYSTROPHY IN THE ADULT, WITH A NOTE ON CEPHALOMETRIC CHANGES

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PSEUDO-HURLER polydystrophy has been recently described in detail.[5,6,13] This syndrome, which was mentioned by Spranger and his co-workers under the name of mucolipidosis III[9,10] shows multiple skeletal defects. Of the 16 cases reported in the literature to date,[5–10,13] 1 was an adult,[5] and in the addendum to the paper by Melhem *et al.*,[8] a 25 year old man with pseudo-Hurler polydystrophy is reported in detail, along with accompanying roentgenograms; all the others were children or adolescents.

The roentgen findings described in this syndrome are variable[5,6,9,13] and are considered not to be sufficiently characteristic to permit the establishment of the diagnosis on such grounds alone.[8]

The 2 patients reported here, a brother and sister, are adults in their twenties. They have the clinical and roentgen features previously described with this syndrome and, in addition, several other findings that have hitherto not been described.

REPORT OF CASES

CASE 1. This patient was a 28 year old, heavy set, 146 cm. tall male, married and father of 4 normal children. He was brought to our attention because of severe low back pain, a painful gait, and progressive limitation of motion in the right hip joint. He was of normal intelligence. He had coarse facies and thickened skin folds.

The heart sounds were normal with a blowing systolic murmur Grade I/IV at the apex and over the anterior axillary line. The heart was not enlarged and the electrocardiogram was normal.

The abdomen was soft; a small umbilical hernia with a wide neck, as well as surgical scars from previous repairs of bilateral inguinal hernias, were found. The liver, which was palpated 2 cm. below the costal margin, was firm, smooth and not tender. The spleen was not palpable.

The musculoskeletal system revealed numerous abnormalities. The neck was short and the hands were broad, with flexion contractures of all the distal interphalangeal joints. Both wrists were in positions of 15° ulnar deviation and had a range of palmar flexion of only 25°. The muscles of the thenar eminence showed extensive wasting and the distal interphalangeal joints had a range of movement limited between 15° and 50°. Both elbows had a full range of flexion and extension, but the supination/pronation range of movement of the forearm was restricted. The range of movement in both hips was restricted and painful. The right hip had a 25° flexion adduction contracture which caused a 2.5 cm. shortening of the right lower limb.

Although the detailed clinical and biochemical findings were reported elsewhere,[1,12] it should be briefly mentioned that the urinary mucopolysaccharides were not increased.

On slit lamp examination, a diffuse corneal clouding was observed, without gross corneal opacity.

The roentgen examinations revealed as follows:

Spine. There was loss of the physiologic lordosis in both thoracic and lumbar spine, with wedging of the lower thoracic and upper lumbar vertebrae. There were irregularities of the end plates of the vertebral bodies, while the intervertebral spaces were well preserved (Fig. 1*A*). A slight scoliosis to the left of the upper lumbar spine was noted. The oar-shaped

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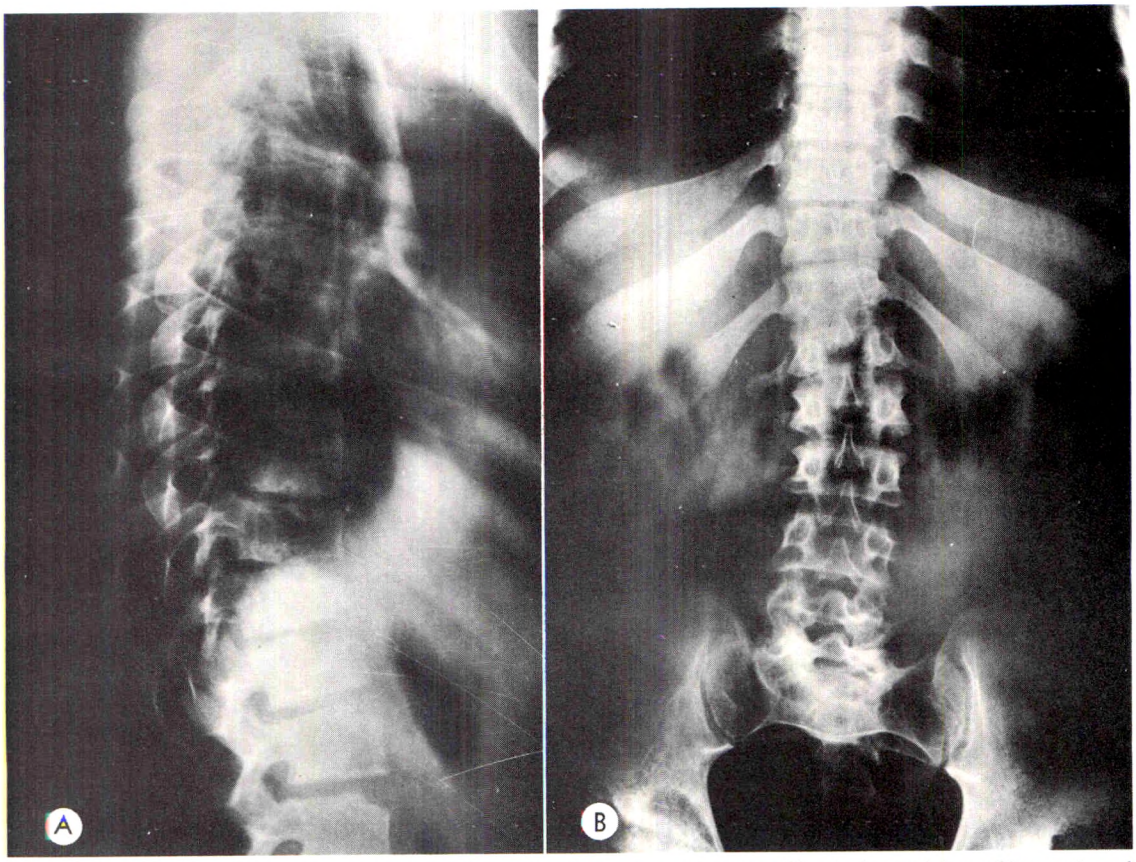


Fig. 1. Case 1. (*A*) Lateral roentgenogram of dorsolumbar spine. Absence of lordosis, wedging of lower dorsal and upper lumbar vertebrae, and irregularities of the end plates. The intervertebral space is well preserved. (*B*) Anteroposterior roentgenogram. Slight scoliosis of the upper lumbar spine to the left. Characteristic oar-shaped ribs.

form of the lower ribs was prominent (Fig. 1*B*). The dens was absent causing atlanto-axial subluxation (Fig. 2).

Pelvis. There was a definite flaring of the iliac wings with prominence of the anterior inferior iliac spines. The right femoral head was flattened, irregular with increased density, and there was irregularity of the acetabulum. The left hip was dysplastic with a shallow acetabulum. These features indicate probably osteoarthritis (Fig. 3).

Long bones. The humeral head was small, flattened with a shortened neck. The corticalis was thickened. The proximal end of the tibiae had thickened borders with cyst-like defects. The left medial femoral condyle showed signs of osteochondritis dissecans (Fig. 4, *A* and *B*).

Hands. Claw hands with irregular defects of the distal ulna on the right were found. There was a nonunion of the styloid of the radius bilaterally, and short metacarpals were present, with narrowing of their distal ends. The proximal phalanges were similarly short and cone shaped. The scaphoid and lunate were hypoplastic. Radial deviation at the wrists and ulnar deviation at the metacarpo-phalangeal joints were present (Fig. 5, *A* and *B*).

Chest. On the posteroanterior roentgenogram the heart and lungs appeared normal. The clavicles were short and widened with distortion of the typical S-shape appearance (Fig. 6). The inferior margin of the acromion was concave (no arthritic changes in the shoulders). On barium swallow study, a typical imprint on the esophagus, indicating a left aberrant subclavian artery, was demonstrated (Fig. 7, *A* and *B*). *The skull* was normal.

Case II. This patient is the 20 year old sister of the male described above. She was 148 cm.

tall, heavy set, with coarse facies and thickened skin folds similar to her brother's, but to a lesser degree. She was single, of normal intelligence and had no clinical complaints.

The heart examination revealed normal sounds with a systolic murmur Grade I/IV over the precordium and at the apex, where it was most pronounced. The electrocardiogram was normal. The abdomen was soft; the liver and spleen were not palpable. A small umbilical hernia was found.

Musculoskeletal examination showed no impairment of joint motion. Nevertheless, careful inspection of the upper extremities revealed diffuse enlargement of the distal radius and ulna with a slight ulnar deviation of the wrist, wasting of the thenar eminence (less than in the brother) and diffuse thickening of the soft tissues around the distal interphalangeal joints of the fingers.

The detailed clinical and biochemical findings were reported elsewhere.[1,12]

The roentgen examinations revealed as follows:

Spine. There was scoliosis to the right in the lumbar region (Grade 1 spondylolisthesis L5-S1), with irregularities of the vertebral end plates (Fig. 8, *A* and *B*). Oar-shaped ribs were prominent. The dens was absent without atlanto-axial subluxation or dislocation (Fig. 9).

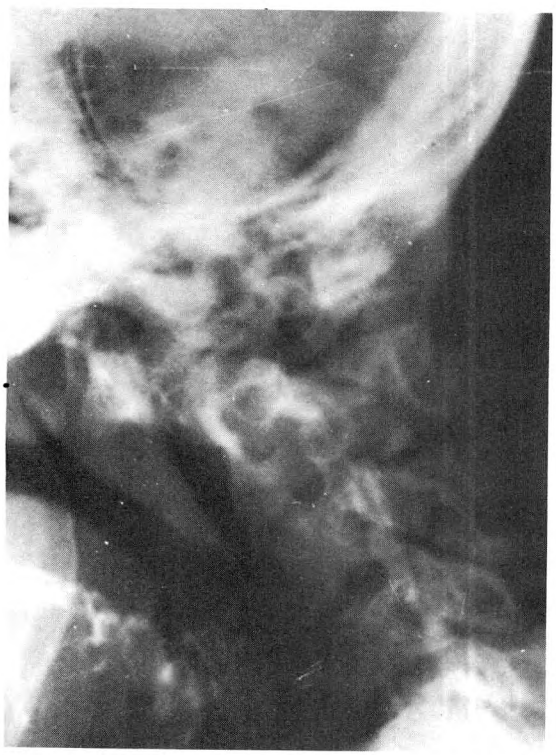


FIG. 2. Lateral roentgenograms of upper cervical spine. Atlanto-axial subluxation.



FIG. 3. Roentgenogram of the pelvis. Flared iliac wings with prominence of the anterior inferior iliac spines. The right femoral head is flattened and irregular with increased density. There is irregularity of the acetabulum. The left hip is dysplastic with shallow acetabulum.

Pelvis. Cyst-like defects were detected both in the ischial bones and in the femoral heads. The hip joints were normal (Fig. 10).

Long bones. Small and flattened humeral

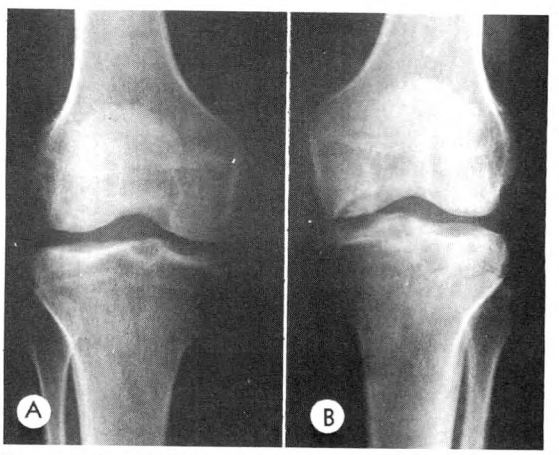


FIG. 4. (*A* and *B*) Anteroposterior roentgenograms of the knees. There are cyst-like defects with thickened borders in the proximal ends of both tibiae. The articular space and surface are normal. There are beginning bilateral degenerative changes of the lateral aspect of the distal femora.

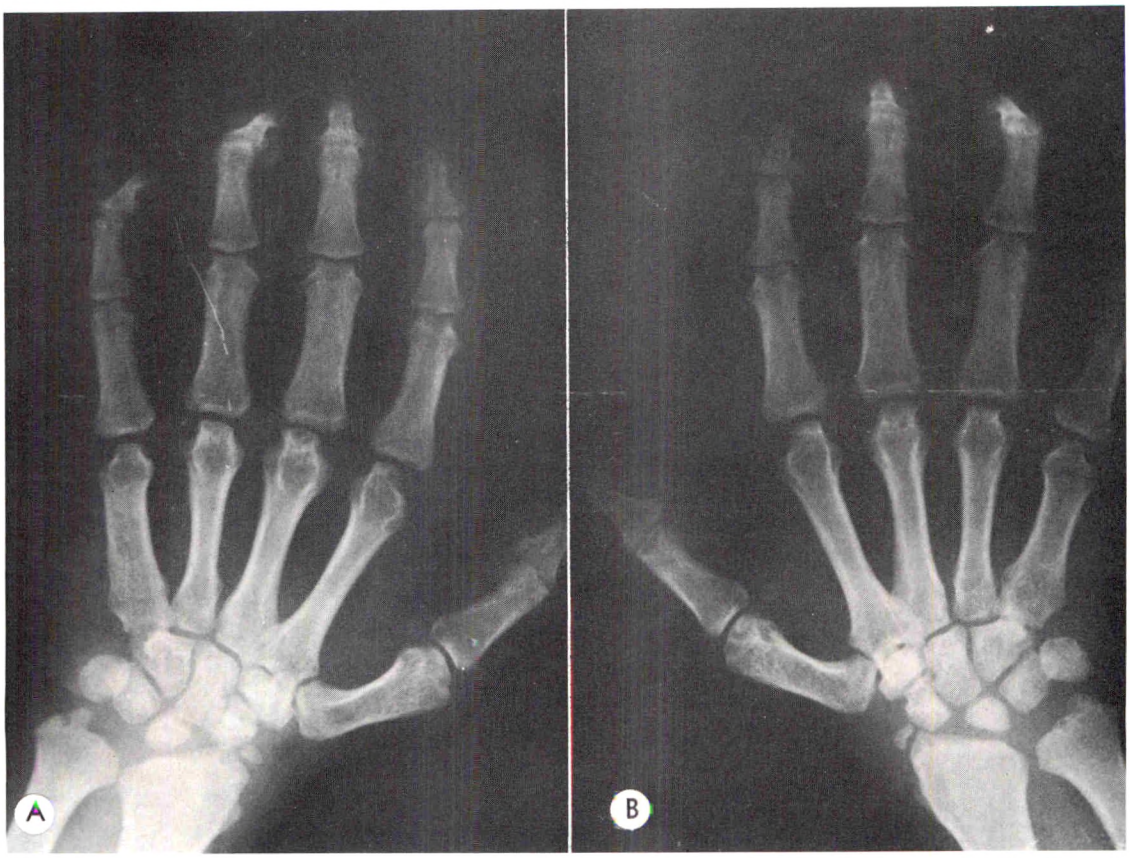


Fig. 5. (*A* and *B*) Anteroposterior roentgenograms of the hands and wrists. The distal end of the forearms is short and plump. There are irregularities with cyst-like defects in the distal end of the right ulna. The scaphoid and lunate are hypoplastic, with increased distance between them. There is radial deviation at the wrists and ulnar deviation at the metacarpophalangeal joints. There is bilateral nonunion of the styloid of the radius. The metacarpals are short, with cone-like narrowing of their distal ends. There is a similar appearance of the proximal phalanges.

heads were present (Fig. 11). The tibiae and femora showed no roentgen abnormalities.

Hands and feet. There was loss of normal contour of the distal forearms, and cyst-like deformities in the carpal bones, more prominent on the left, were noted. The first metacarpals were disproportionally short. There was cone-shaped narrowing of the distal end of the phalanges (Fig. 12, *A* and *B*). In the feet, the size and shape of the metatarsal bones and phalanges appeared normal.

Chest. On the posteroanterior roentgenogram the heart and lungs appeared normal. The clavicles were short and widened with distortion of the typical S-shape appearance. *The skull* was normal.

FULL MOUTH ROENTGENOGRAMS

Full mouth roentgenograms were obtained of both siblings. The cephalometric roentgenogram may show the harmony and balance of the component parts of the face; *i.e.*, the skeletal framework, the teeth, and the overlying musculature.[2] For the cephalometric roentgenograms the Broadbent-Bolton Roentgenographic Cephalometer was used, and measurements were made from acetate tracings of the headplates.

The full mouth intraoral roentgenograms of both siblings showed no particular signs of metabolic disturbance.[11] The cortical plates were of normal thickness, the borders of the maxillary sinuses were visible, as was the lamina dura of the tooth sockets. The films of the sister showed congenitally

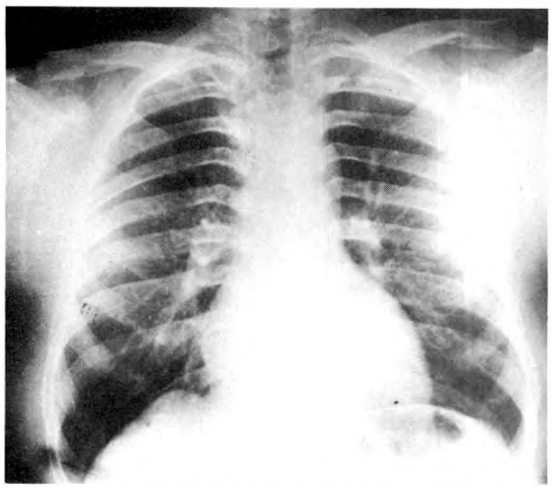


FIG. 6. Posteroanterior roentgenogram of the chest. Both clavicles are short and widened, with loss of the typical S-shape appearance. The inferior margin of the acromion is concave. The heart and lungs are normal. (Note the oar-shaped appearance of ribs.)

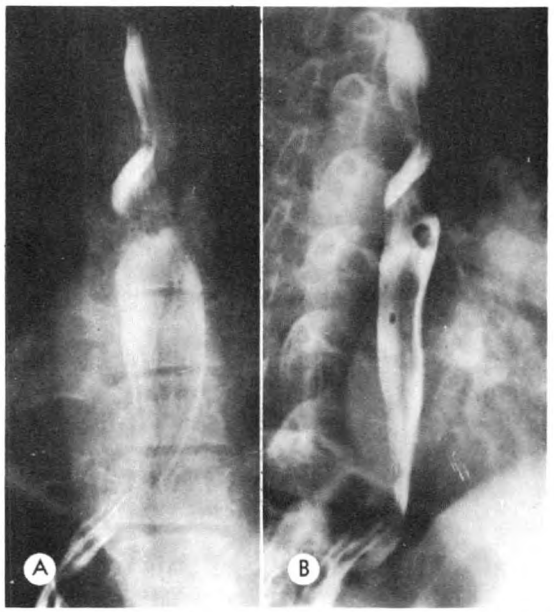


FIG. 7 (*A* and *B*) Barium swallow study of the esophagus. There is a definite imprint upon the upper thoracic esophagus which is consistent with a left aberrant subclavian artery.

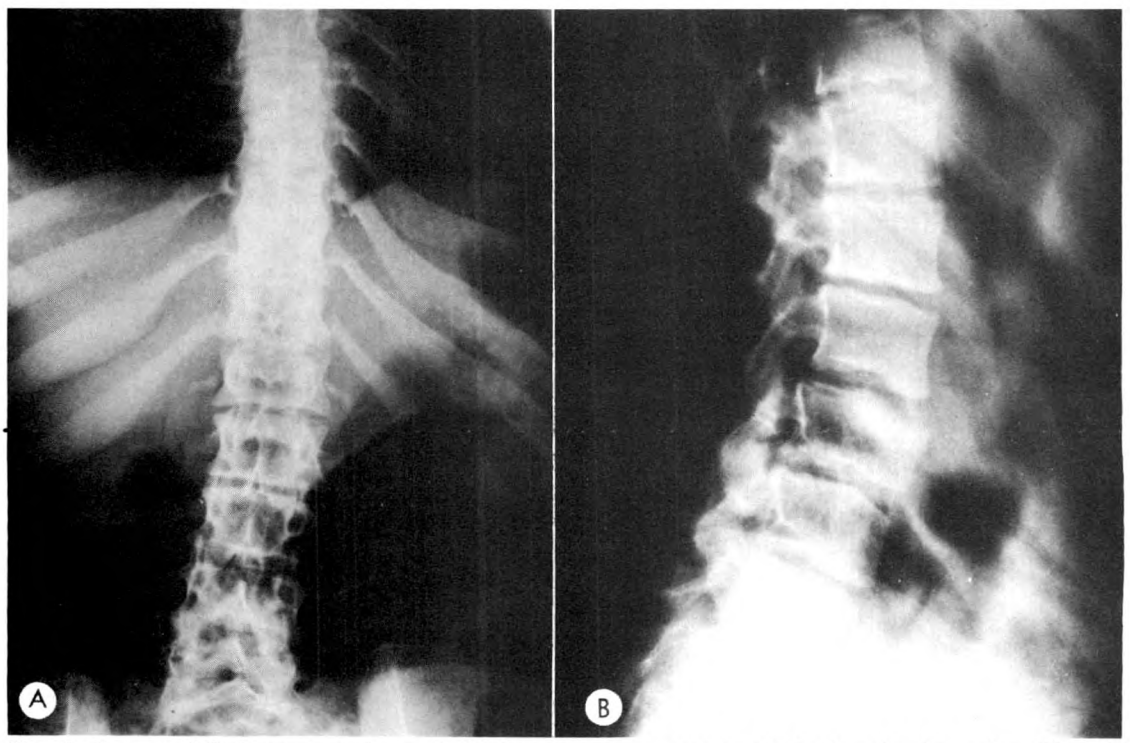


FIG. 8. Case II. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the dorsolumbar spine. There is scoliosis to the right in the lower lumbar spine and spondylolisthesis of L5–S1. There are slight irregularities of the end plates, with absence of anterior superior epiphysis in the lower lumbar vertebrae. (Note oar-shaped lower ribs.)

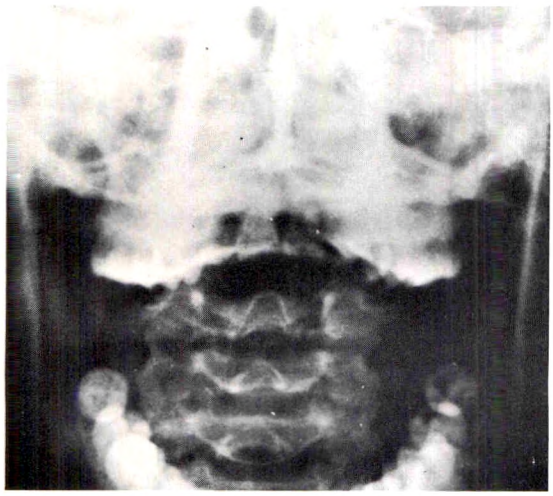


Fig. 9. Anteroposterior roentgenogram of the upper cervical spine through the open mouth. There is absence of the dens.

missing maxillary third molars ($\frac{8}{\perp 8}$) only.

Figures 13 and 14 are the acetate tracings made from the headplates. The tracings of the brother (Fig. 13) show a facial and dental mandibular prognathism. This condition is not very clear in the tracing of the sister (Fig. 14), due to the open bite that extends forward from the second mandibular molar on each side. The atypical folds of the skin of both siblings are demonstrated on the tracings.

Comparison between the cephalometric data of the brother and sister showed that these were very similar in both sibs. These measurements did not present a fixed pattern, but varied within the extreme range of the normal limits. This is represented in a graphic way, using 10 measurements of Down's analysis, plotted on the polygon proposed by Vorhies and Adams (Fig. 15).

In the brother, although the mandible is protruded, the chin is only slightly developed, as can be seen from the "chin button" measurements. The latter measures only 2 mm., while the 'mean' normal is 3 mm. The reverse would have been expected here (Fig. 13).

The chin button is the distance between Pg and the N-B line (Fig. 13).

DISCUSSION

The roentgen findings of the 2 described siblings have many features similar to the syndrome described by Maroteaux and Lamy[6] as pseudo-Hurler polydystrophy and summarized by Spranger and Wiedemann as mucolipidosis III.[10]

Of the 16 patients with this syndrome described in the medical literature, only 1 was an adult,[5] while the others were either children or adolescents. The roentgen find-

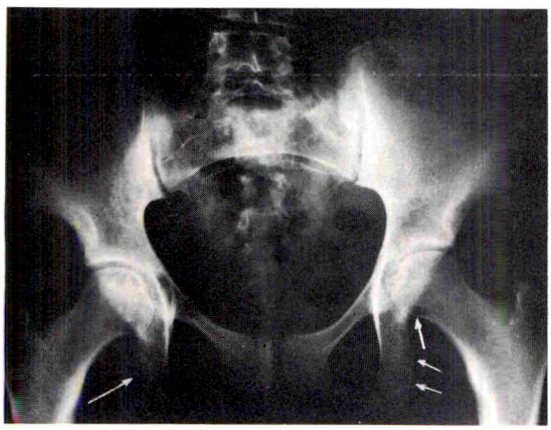


Fig. 10. Roentgenogram of the pelvis. There are cyst-like defects in the head of the femur and both ischia (arrows). The articular spaces and surfaces are normal.

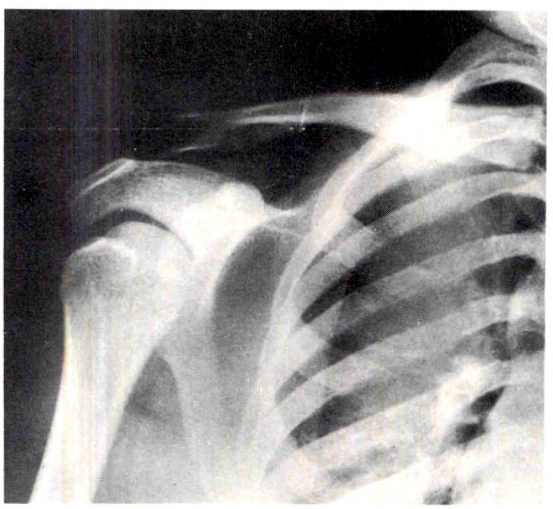


Fig. 11. Roentgenogram of the right shoulder. The head of the humerus is small and flattened. The clavicle is short and widened.

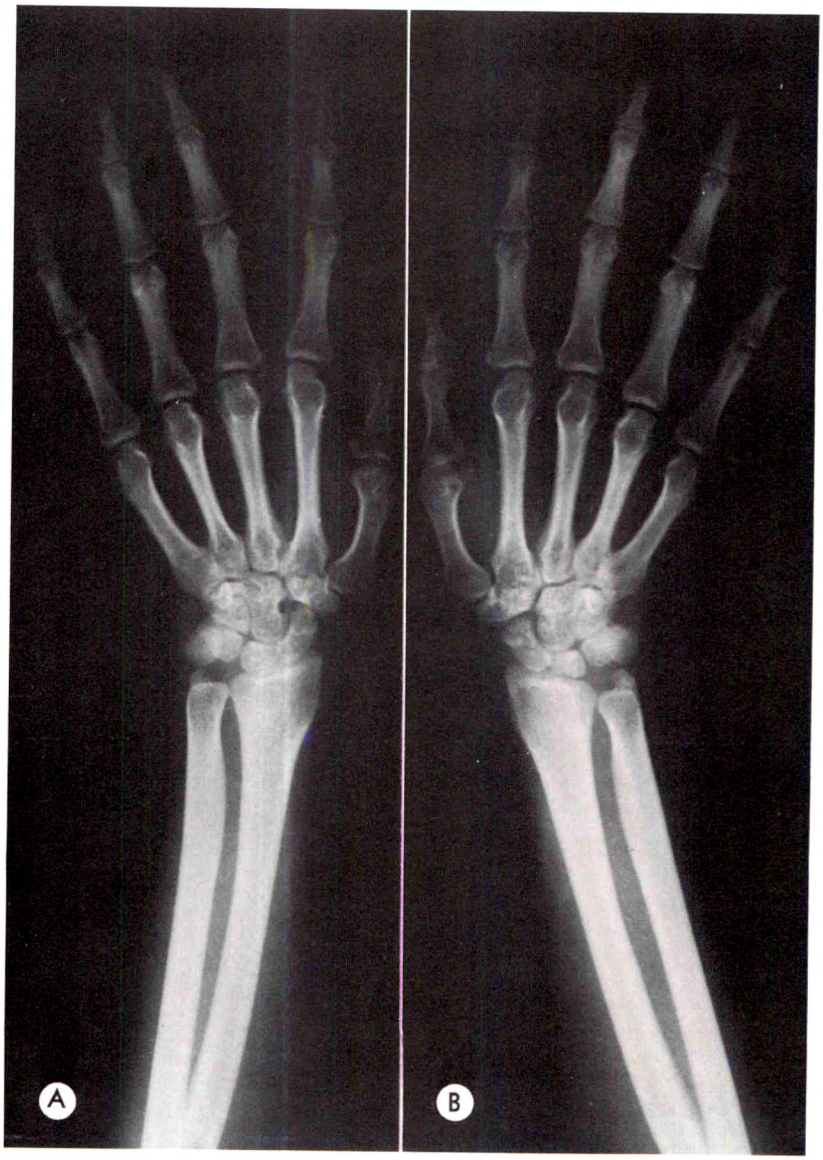


FIG. 12. (*A* and *B*) Roentgenograms of the upper extremities. There is loss of normal contour of the distal forearm. There are cyst-like deformities in the carpal bones, more prominent on the left. There is bilateral osteoporosis of the carpal bones. There are short metacarpals and phalanges, with beginning of narrowing of their distal ends.

ings in children affected by this syndrome have recently been described by Melhem *et al.*[8] as being variable and not sufficiently characteristic to permit diagnosis on roentgenologic grounds alone.

It is possible that some of the skeletal findings detected in our patients, and which were not described by others, are related to their adult age. These findings include lordosis of the upper lumbar spine, short and plump tubular bones with cyst-like defects in their distal ends, as well as cyst-like defects found in some of the carpal bones. These cyst-like defects may be related to the degenerative changes found in the synovial joints.

The odontoid process was absent in both patients, the clinical and roentgen

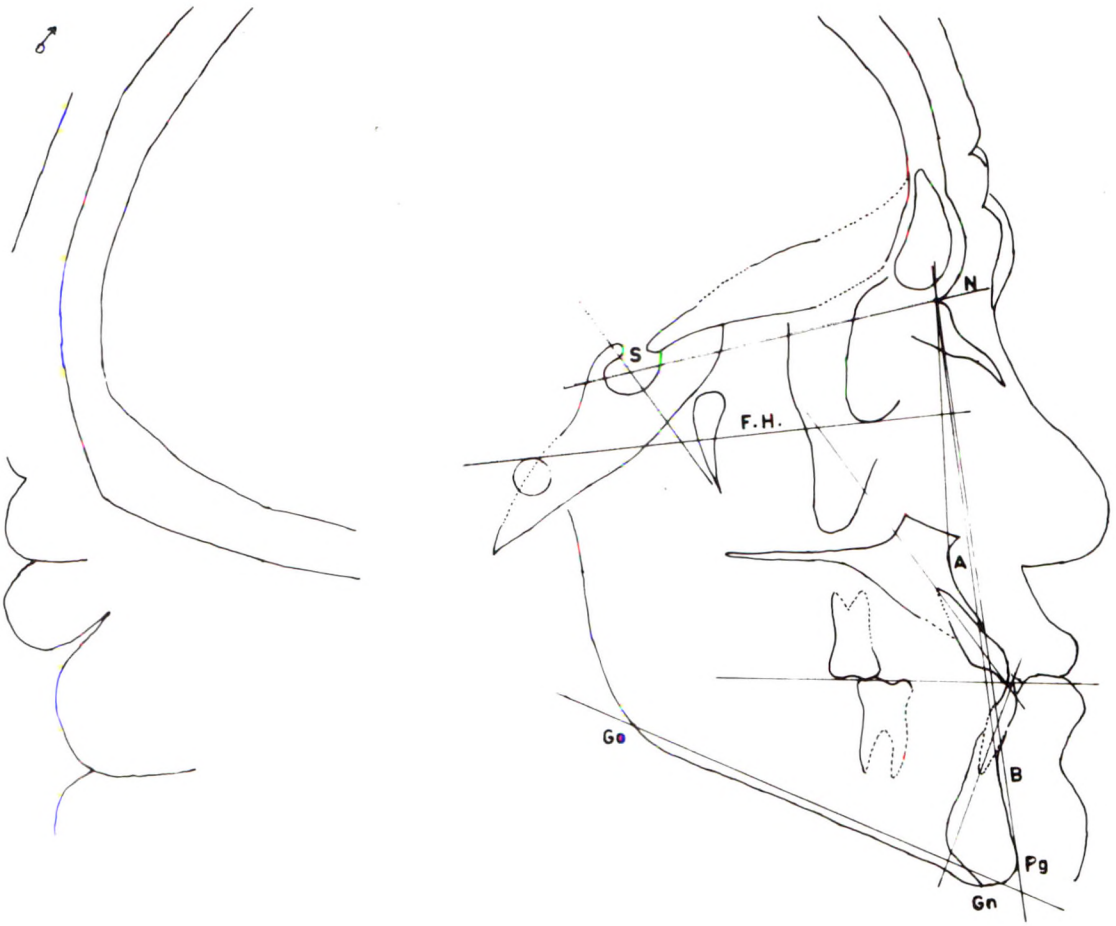


Fig. 13. Cephalometric tracing of Case 1 showing mandibular prognathism.

signs of C1–C2 subluxation becoming evident in the brother only.

The barium swallow was administered as part of the cardiac study and revealed a definite posterior imprint upon the upper thoracic esophagus, indicating a left aberrant subclavian artery. Whether or not this finding is a part of this syndrome remains to be established. The presence of an additional valvular lesion as described by Steinbach *et al.*[13] can only be ascertained by cardiac catheterization and angiocardiographic studies.

The shape and size of the skull of both brother and sister were normal.

The clinical impression of mandibular prognathism was substantiated by the cephalometric studies. The cephalometric roentgenogram has the potentiality to show the harmony and balance of the component parts of the face; *i.e.*, the skeletal framework, the teeth, and the overlying musculature.[2] The development of this method was made possible by the extensive research work done by tracing, measuring and comparing dozens of planes, angles and dimensions on standardized head roentgenograms. From these observations a concept gradually evolved of what constitutes acceptable balance and harmony of the component parts of the face.[3]

The most frequently used cephalometric analyses rely on measurements. When a facial deformity is clearly apparent clinically, it is to be expected that at least some cephalometric values will differ from aver-

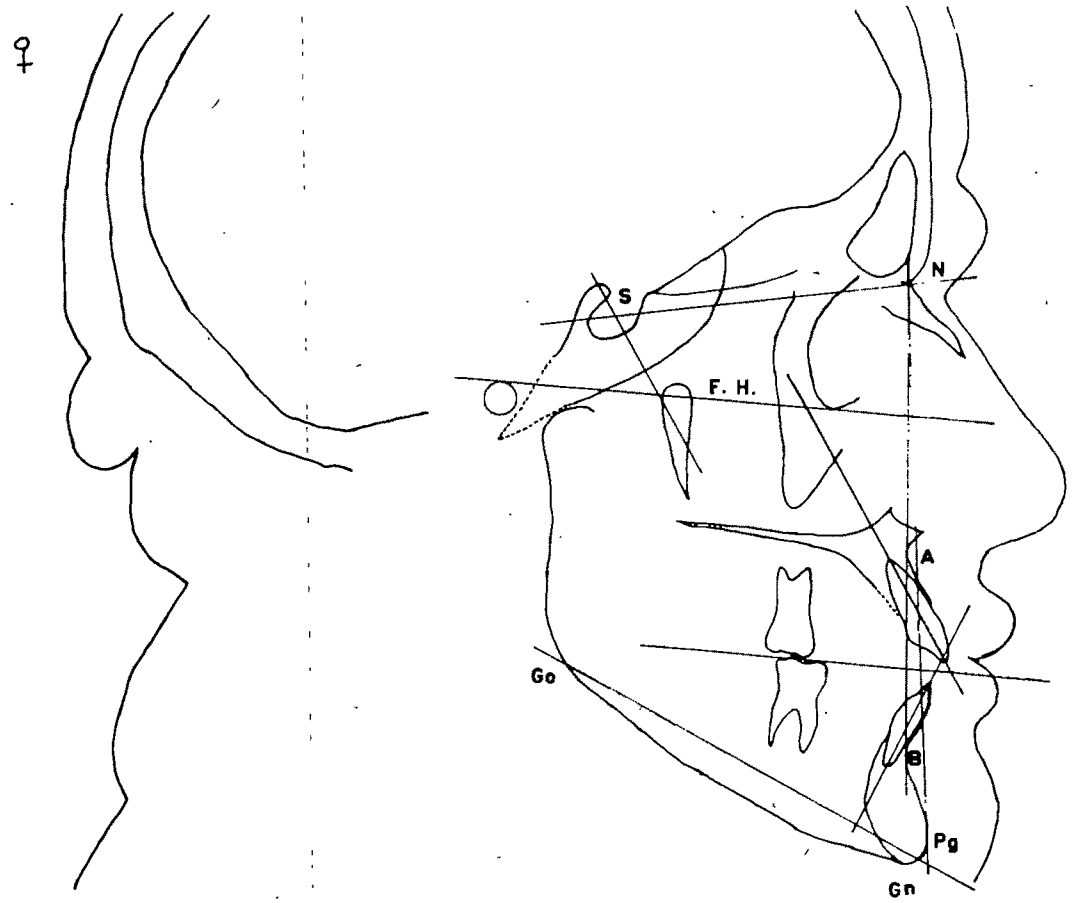


FIG. 14. Cephalometric tracing of Case II showing the anterior open-bite and the tendency to mandibular prognathism.

age "norms." Mandibular prognathism is a facial disfigurement in which the lower portion of the face appears unduly prominent and the teeth are in a Class II (Angle) malocclusion. Horowitz[4] pointed out that, when each cephalometric measurement is evaluated independently, it becomes difficult to determine the relative importance of any particular variable in separating the deformity group from the control group.

Almost all measurements of both siblings fall within the ranges of normal subjects, but by analyzing combinations of measurements, mandibular prognathism (especially of the brother) is evident.

Some of the discrepancies in measurements can be ascribed to the fact that the sister exhibited an anterior open-bite. Had this not been present, the measurements would have conformed more closely to those of the brother.

The effects of a disease in which skeletal involvement may be a feature, as in metabolic disturbances, may be manifested by accelerated or retarded development and rate of eruption of teeth, structural defects, changes in trabecular pattern, and formation of lesions of the supporting bone.[11] Changes that may be seen in full-mouth periapical roentgenograms are:

1. Alteration in the thickness of the cortical plates.

2. Disappearance of the narrow radiopaque lines that represent the borders of the maxillary sinuses and nasal fossae.

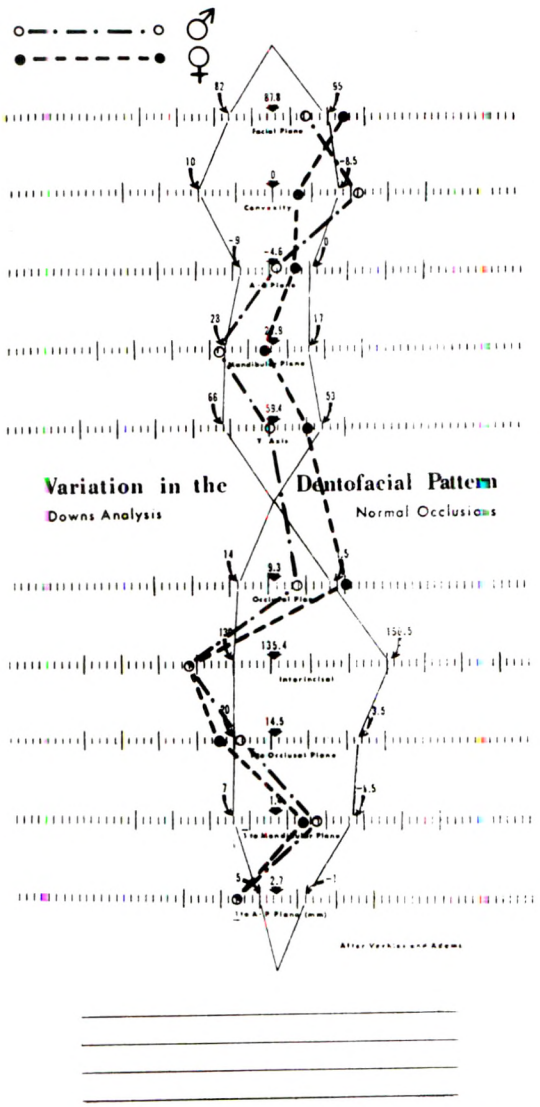


Fig. 15. Ten cephalometric measurements of both siblings plotted on Down's polygon, presenting the similar atypical facial pattern of both patients.

3. Disappearance of the lamina dura that delineates the normal tooth sockets.[11]

It is noteworthy that in the here described 2 patients, affected by a storage disease of which the basic enzymatic defect still awaits elucidation, the full-mouth roentgenograms did not reveal any signs of metabolic disturbances.

Although the roentgen findings in pseudo-Hurler polydystrophy are variable, from the skeletal changes described by different authors regarding this syndrome, a pattern emerges that may permit the differentiation of mucolipidosis III from other mucolipidoses and certain mucopolysaccharidoses on roentgenologic grounds. Nevertheless, for a definite diagnosis the correlation of the clinical, laboratory and roentgen findings is imperative.

SUMMARY

The roentgen findings of 2 adult siblings affected by pseudo-Hurler polydystrophy (mucolipidosis III) are described.

These include: mandibular prognathism; absence of dens; short and wide clavicles; short and plump tubular bones with cyst-like defects in their distal ends; claw hands with nonunion of the styloid of the radius; and oar-shaped ribs. The findings in the spine include irregularities of the vertebral end plates and slight scoliosis.

Although a pattern of the roentgen findings in this syndrome emerges from the literature and the here published data, laboratory and roentgen findings are necessary for a specific diagnosis.

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REFERENCES

1. Berman, E. R., Yatziv, S., and Stein, H. Pseudo-Hurler polydystrophy (mucolipidosis III). 2. Biochemical and tissue culture studies. To be published.
2. Down, W. B. Variations in facial relationships: their significance in treatment and prognosis. *Am. J. Orthodontics*, 1948, *34*, 812–840.
3. Down, W. B. Analysis of dentofacial profile. *Angle Orthodontics*, 1956, *26*, 191–212.
4. Horowitz, S. L. Challenge of facial deformity. *Dent. Pract.*, 1972, *22*, 191–202.
5. Langer, L. O., Jr., Kronenberg, R. S., and Gorlin, R. J. Case simulating Hurler syndrome of unusual longevity, without abnormal mucopolysaccharidiuria. *Am. J. Med.*, 1966, *40*, 448–457.
6. Maroteaux, P., and Lamy, M. La pseudopolydystrophie de Hurler. *Presse méd.*, 1966, *74*, 2889–2892.
7. Mckusick, V. A. Mendelian Inheritance in Man. Third edition. Johns Hopkins Press, Baltimore, 1971, p. 458.

8. MELHEM, R., DORST, P. J., SCOTT, C. I., and MCKUSICK, V. A. Roentgen findings in mucolipidosis III. *Radiology*, 1973, *106*, 153–160.
9. SPRANGER, J., WIEDEMANN, H-R., TOLKSDORF, M., GRAUCOB, E., and CAESAR, R. Lipomucopolysaccharidose: Eine neue Speicherkrankheit. *Ztschr. Kinderh.*, 1968, *103*, 285–306.
10. SPRANGER, J. W., and WIEDEMANN, H-R. Genetic mucolipidoses. *Humangenetik*, 1970, *9*, 113–139.
11. STAFNE, E. C. Oral Roentgenographic Diagnosis. Second edition Chapter 18. W. B. Saunders Company, Philadelphia, 1967.
12. STEIN, H., BERMAN, E. R., MERIN, S., SCHESKIN, Y., and COHEN, T. Pseudo-Hurler polydystrophy (mucolipidosis III). 1. Clinical study of two adult siblings. To be published.
13. STEINBACH, H. L., PREGER, L., WILLIAMS, H. E., and COHEN, P. Hurler syndrome without abnormal mucopolysaccharidiuria. *Radiology*, 1968, *90*, 472–478.

VOL. 122, NO. 1

THE FEASIBILITY OF THE PA PROJECTION FOR TOMOGRAPHY OF THE PETROUS BONE*

SIGNIFICANT REDUCTION IN RADIATION DOSE TO THE LENS OF THE EYE

By PAUL BERGER, M.D., SUSAN GILDERSLEEVE, R.T., *and* ANDREW POZNANSKI, M.D.
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THE possibility of cataract formation makes reduction of dose to the lens of the eyes of children worthwhile. The purpose of this study is to determine how much the eye dose can be reduced by using the PA (posteroanterior) rather than the AP (anteroposterior) projection in linear tomography of the petrous bone, and to what extent this projection is feasible when examing children.

METHOD

Lithium fluoride thermoluminescent dosimeters were placed as close to the lower eyelid as possible in 2 groups of patients. Both petrous bones were examined on each cut. Roentgenographic factors were: 200 mas.; 62 to 67 kv.; Par Speed screens and RPL Kodak film; 2 mm. Al filtration; and 40 inch focal film distance. The field size was 12 cm.×5 cm. and a 12:1 Bucky grid was used.

Group I consisted of 3 children, each of whom had 6 linear tomographic cuts in the AP projection, as well as a single Towne view, and a right and left Law projection.

Group II consisted of 14 children, 13 of whom had linear tomograms made in the PA projection.

The first group all had 6 tomographic cuts and the second group 3 to 9 cuts; 8 of the 13 children had had 6 exposures each (Table 1). One child in Group II was 4 months of age and due to lack of cooperation was examined in the AP projection. Because of the size, 100 mas. was used rather than 200.

The lithium fluoride dosimeters were calibrated, based on an exposure of controls to cesium 137, corrected to an effective energy of 25 kev. The precision was approximately plus or minus 5 per cent.

Prior to beginning tomography the technician emphasizes to the children the importance of lying still during the procedure. The child is then placed in the prone position with forehead and nose touching the table. The head is immobilized between 2 weighted blocks. Occasionally a foam rubber padding is placed beneath the forehead, but most often this was unnecessary. The child is given encouragement during the procedure and the importance of lying still is re-emphasized. Tomographic cuts are then obtained as for the AP projection.

RESULTS

We have found that most children can and will cooperate very well during tomography. We find that in about 80 per cent of children seen at our hospital the PA projection can be easily used.

Dosages to the eye following AP projection are listed in Table 1 and range from 3.6 to 11.2 rads. Case 6 was the 4 month old infant, in whom a lower exposure was needed. The reason for the relatively lower dose in Case 1 is not known, but it may have been that the dosimeter was at the edge of the field.

The dose to the eye in children examined in the PA projection was markedly lower than in the AP, ranging from 50 millirads to a maximum of 340 millirads with most readings between 100 and 300 millirads. From these data it appears that the eye dose is 30–50 millirads per linear tomographic exposure in the PA projection.

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TABLE I

DOSAGE TO THE LENS FOLLOWING AP AND PA LINEAR TOMOGRAPHY

Case	Direction of Beam AP or PA	No. Tomographic Cuts	Dose (mrad)
Group 1			
1	AP	6	3,650
2	AP	6	11,200
3	AP	6	10,600
Group 2			
4	PA	6	180
5	PA	4	50
*6	AP	6	4,000
7	PA	6	140
8	PA	6	220
9	PA	6	140
10	PA	6	240
11	PA	6	290
12	PA	7	310
13	PA	6	240
14	PA	5	130
15	PA	6	140
16	PA	9	340
17	PA	3	110

* Exposure factors of 4 month old child 58 kvp. and 100 mas. AP = anteroposterior; PA = posteroanterior.

DISCUSSION

Linear tomography of the petrous bone is part of the routine evaluation of suspected abnormalities of the middle and inner ear at the C. S. Mott Children's Hospital. Although tomography with hypocycloidal or other complex motion provides best delineation of the petrous bone, it is not readily available in many hospitals. We have found that linear tomography can provide satisfactory detail, particularly in children.

It has been shown that lens opacities may occur in animals from a single dose of 15 rads,[5] although the cataractogenic dose in humans is probably higher.[4] The dose to the lens with AP hypocycloidal tomography can be significantly reduced with eye shields. Chin *et al.*[1] showed a drop from 10 r to 1 r, while Krohmer[3] for 3 tomographic cuts showed a 6 fold drop from 4.4 r. Unfortunately, eye shields cannot be used in linear tomography since they produce ghost shadows which obscure the region of the middle and inner ear.

A much simpler method in reduction of dosage is by the use of the PA rather than the AP projection. This concept is rather obvious and was remarked by others,[1,2,6] although PA tomography has rarely been used because of inconvenience. However, we have found that most children can and will cooperate very well during tomography and in about 80 per cent of children seen in a children's hospital this projection can easily be used.

The total dose to the lens following the entire PA tomographic examination was usually between 100–300 millirads or a reduction by a factor of at least 20–30× when compared to similar examination when done in the AP projection. This radiation dose is less than the dose to the lens from a single AP roentgenogram of the skull.[6]

There was no difference in the quality of the film obtained using PA *versus* AP projection. The 20–30 fold reduction in dosage to the lens certainly makes the PA projection worthwhile. In the few children who are uncooperative and who will not tolerate this position, sedation may be indicated.

SUMMARY

PA view during linear tomography of the petrous bone can easily be used in about 80 per cent of children.

This results in a 20–30 fold reduction in dosage to the lens when compared to the AP view. Dose to the lens with 6 cuts in the PA projection is less than from a single AP roentgenogram of the skull.

It is strongly recommended that the PA projection be used during linear tomography of the petrous temporal bone in all children and adults whenever possible.

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REFERENCES

1. Chin, F. K., Anderson, W. B., and Gilbertson, J. D. Radiation dose to critical organs during petrous tomography. *Radiology*, 1970, *94*, 623–627.
2. Dobrin, R., Becker, M. H., and Geniesser, N. B. Radiation protection of cornea and lens during petrous-bone tomography. *Radiology*, 1973, *109*, 201–203.
3. Krohmer, J. S. Patient dose distribution during hypocycloidal tomography. *Radiology*, 1972, *103*, 447–450.
4. Merriam, G. R., Jr., and Focht, E. F. Clinical study of radiation cataracts and relationship to dose. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *77*, 759–785.
5. Upton, A. C., Christenberry, K. W., and Furth, J. Comparison of local and systemic exposures in production of radiation cataract. *A.M.A. Arch. Ophth.*, 1953, *49*, 164–167.
6. Van Tuinen, R., Arnold, W., Elson, H., Keene, J., and Kereiakes, J. Radiation dose using thermoluminescent dosimetry. *App. Radiol.*, 1973, 27–29.

DÉJERINE-SOTTAS SYNDROME IN CHILDREN*

(HYPERTROPHIC INTERSTITIAL POLYNEURITIS)

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HYPERTROPHIC interstitial polyneuritis (H.I.P.), otherwise known as Déjerine-Sottas syndrome, is an uncommon familial condition. It is characterized by thickening of the affected nerves due to hyperplasia of the nerve sheath, and may involve one or more of the peripheral nerves, spinal nerves or the cauda equina. If the peripheral nerves are not involved, diagnosis can be difficult. Myelography, performed when the condition is suspected, can prevent an erroneous diagnosis of Friedreich's ataxia or Charcot-Marie-Tooth myopathy.

Although the condition starts in childhood and is progressive, the clinical manifestations are usually not apparent until later in life. The original case reports by Déjerine,[4] however, were of 2 patients aged 12 and 15 years. Clinically, there is progressive motor and sensory neural involvement with periods of apparent remission.

Our report is of 2 cases of H.I.P. diagnosed in childhood by myelography; the roentgenographic findings of H.I.P. in this age group are presented.

REPORT OF CASES

CASE I. O.J., a 14 year old Caucasian girl, had full-term gestation with a normal neonatal period. At 14 months of age her gait became abnormal, being described as stiff, stamping and high-stepping. This remained so until she was 12 years old, when ataxia and associated sensory loss also developed. At 14 years of age she was referred to The Hospital for Sick Children, Toronto, for neurologic evaluation.

Her family history gave no other cases of possible H.I.P. The main neurologic findings were sensory loss, ataxia and nystagmus.

Roentgenograms of the spine demonstrated mild scoliosis in the thoracolumbar region, with some widening of the upper lumbar interpedicular distances and flattening of the pedicles at L1–L2.

Lumbar myelography was attempted twice. It was difficult to get good cerebrospinal fluid flow through the needle, and a small amount of contrast material introduced was thought to have gone subdural because of the very slow flow seen fluoroscopically. Repeat myelography *via* cisternal puncture demonstrated generalized thickening of the lumbar and sacral nerve roots and very slow flow of the contrast medium (Fig. 1, *A–D*), both characteristic of H.I.P., as well as a widened lumbar dural sac. H.I.P. was diagnosed, although arachnoiditis or a diffuse tumor were considered possible alternatives.

Complete laminectomy from L1 to L5 was performed. The dura was tense and non-pulsatile. On opening the dural sac, many large, swollen herniated nerve roots were seen. Each of the anterior and posterior nerve roots was approximately 4 times normal size, again characteristic of H.I.P. A biopsy showed proliferating interstitial tissue outside the myelin sheath with thickened individual nerve fibers, confirming the diagnosis of H.I.P.

The patient has been followed for the last 5 years without any significant clinical change.

CASE II. A.S., a 12 year old Caucasian girl, had a history of "walking difficulties" and poor balance since infancy. She was slow in her milestones, having walked at 2½ years. Bilateral genu varum deformities were treated with braces, and later with multiple orthopedic devices, but no improvement resulted. Gait and balance deteriorated slowly during the 2 years prior to admission to this Hospital.

An uncle of the patient, confined to a wheel chair at 19 years of age, died at the age of 34, but further medical information could not be obtained. Another uncle was alleged to have had "multiple sclerosis."

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Decreased power in the lower limbs, absent reflexes and equivocal plantar responses were present on examination. Sensation was decreased to light touch in both lower limbs. Her hands became increasingly clumsy; her gait was ataxic. Initial diagnosis was Friedreich's ataxia. Lumbar puncture demonstrated slow flow of cerebrospinal fluid, with an abnormally raised protein content of 120 mg./100 ml. A high cervical lesion was suspected.

Spinal roentgenograms showed mild posterior scalloping of the lower thoracic and lumbar vertebral bodies (Fig. 2, *A* and *B*). The interpediculate distances and the pedicles were normal.

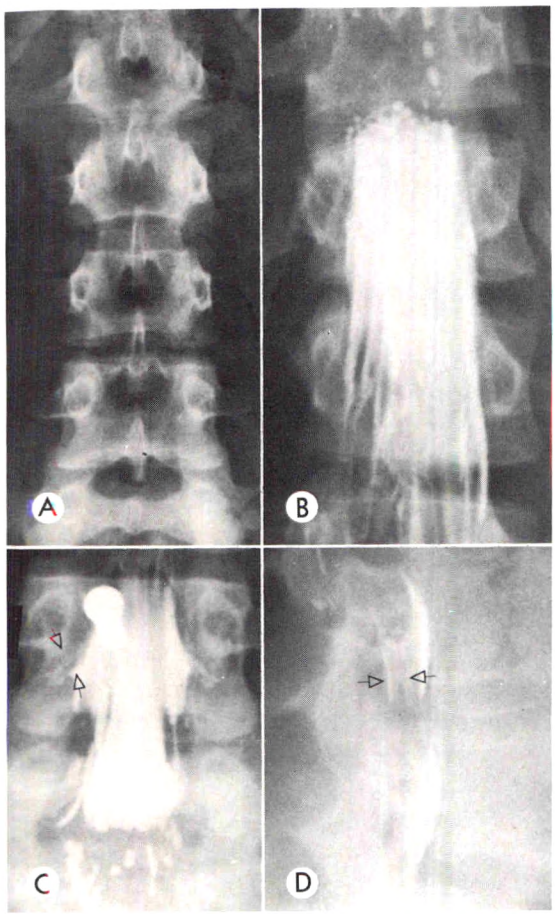


Fig. 1. Case 1. (*A*) Plain film roentgenogram, anteroposterior view. There is slight widening of the upper lumbar interpediculate distance and flattening of the pedicles at L1 and L2. (*B*) Myelogram shows thickened nerve roots outlined by adjacent contrast substance in the cauda equina region. (*C*) a thickened nerve root (arrows) as demonstrated at the level of T12-L1. (*D*) Prone lateral view after cisternal myelography reveals scalloping of the vertebral bodies with contrast material pooling and a thick nerve root (arrows).

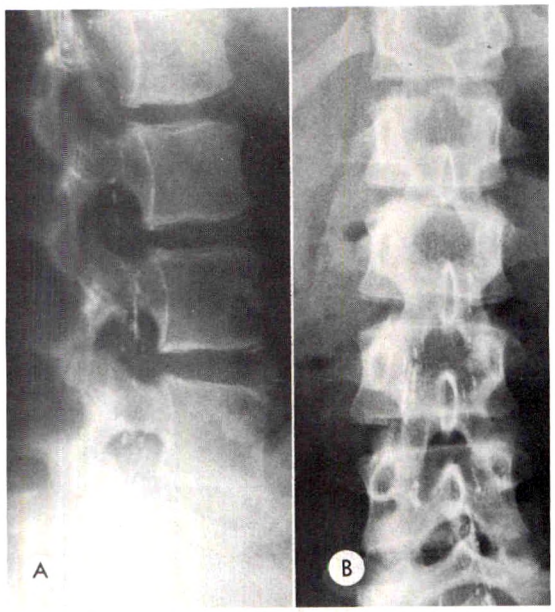


Fig. 2. Case 11. (*A* and *B*) Plain film roentgenograms show mild posterior scalloping of the lower thoracic and lumbar vertebral bodies. The pedicles are probably normal.

Myelography demonstrated a very slow flow of pantopaque in the subarachnoid space. Peculiar linear intradural extramedullary defects, mainly in the lumbosacral region, were noted (Fig. 2, *C–F*). The pantopaque column narrowed at the lumbar disk levels, while the distance between the pedicles and the pantopaque column was increased. H.I.P. was diagnosed and she was treated with steroids. Surgery has not yet been performed and thus the myelographic findings remain unverified.

DISCUSSION

Since the original description by Déjerine and Sottas in 1893, 46 proven cases, mostly adults, have been reported in the English language literature.[1–4,6,7,11,12] Of these, 19 were diagnosed by peripheral nerve biopsy, 21 were found at autopsy and 7 were discovered by myelography. The reported myelographic findings in these 7 cases are shown in Table 1.

Associated bone changes in the vertebral column occur,[3] apparently due to pressure

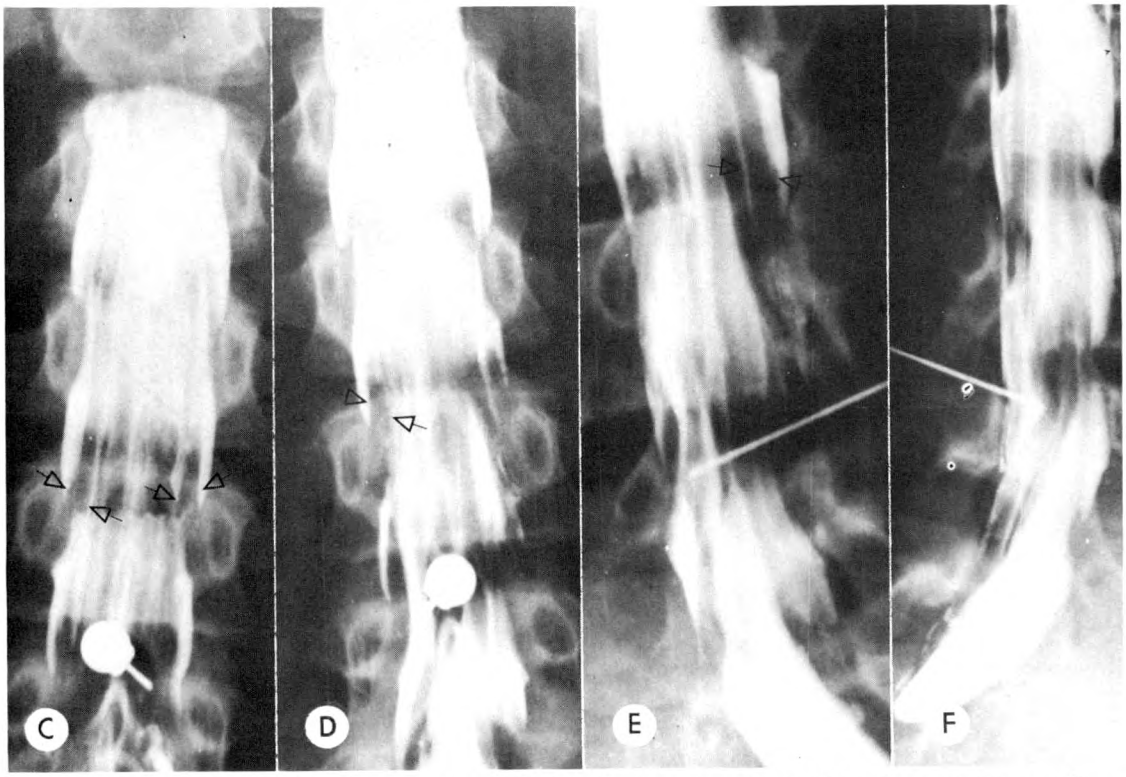


FIG. 2. Case II. (*C* and *D*) Myelograms reveal thickened nerve roots outlined by the pantopaque column adjacent to it in both prone (*C*) and supine (*D*) views, which is not commonly seen. Transverse bar defects are also present. There is increased distance between the pedicle and the pantopaque column due to thickened nerve roots (arrows). (*E* and *F*) The thickened nerve roots are well visualized in the oblique views as they exit (arrows).

from the enlarged nerve roots or from the entrapped cerebrospinal fluid. These bone changes are: (1) posterior scalloping of the vertebral bodies;[9] (2) increase in the interpediculate distances; (3) triangulation of the pedicles with flattening of the superior aspect of the pedicle at the level of the hypertrophied nerve roots and erosion of the medial aspect of the pedicle, all due to pressure from these roots; and (4) diffuse enlargement of the spinal canal, especially in the lumbar region. By themselves, each of these findings do not necessarily indicate H.I.P. and may not be present in childhood. Conversely, all are present in adults.[3] In the 2 children here reported, not all the above bone changes were present.

MYELOGRAPHY

The principal myelographic findings are: (1) linear parallel radiolucencies due to thickened nerve roots; (2) displacement of the pantopaque column medially, thus increasing the distance between the oil column and the medial aspect of the pedicle; and (3) bizarre pooling of the contrast medium in gutters between large hypertrophied nerves. At lumbar puncture, good cerebrospinal fluid flow is difficult to obtain and subsequent recovery of pantopaque is hindered by contrast material being entrapped between the hypertrophied nerve roots. The flow of pantopaque is slow in the subarachnoid space and is occasionally blocked.

These myelographic appearances are characteristic enough to diagnose H.I.P. without the necessity of surgical biopsy. Redundant nerve roots, however, may occasionally produce a clinical picture resembling H.I.P. Similar myelographic findings due to large serpiginous nerve roots

TABLE I

MYELOGRAPHIC FINDINGS IN 7 PREVIOUSLY REPORTED CASES OF HYPERTROPHIC INTERSTITIAL POLYNEURITIS (H.I.P.)

	Sex	Age at Diagnosis	Clinical Findings	Method of Diagnosis
Andermann *et al.*[1]*	F	30	Weakness of distal muscle group since age of 25; nystagmus since age of 22	Myelography showed distorted spinal nerve roots and sleeves Biopsy of hypertrophied nerves
Bellon *et al.*[3]	M	42	Progressive muscular weakness since age of 22	Plain film roentgenograms showed bone changes Myelography demonstrated hypertrophied nerve roots which were proven by biopsy
Hinck and Sachdev[6]	M	63	Intermittent but progressive muscle weakness and neurosensory loss since age of 38	Myelography gave evidence of thickened nerve roots with blocked CSF flow; there was also increased CSF protein content and slow flow
Lewtas and Dimant[7]	M	26	Limping for 3 years; lower limb weakness and muscle wastage; high protein content in cerebrospinal fluid (CSF); thickened peripheral nerves	Plain film roentgenograms were normal Myelography demonstrated slow flow and partial block Biopsy distinctive of H.I.P.
Lewtas and Dimant[7]	M	62	Lower abdomen and leg pain since age of 56; weakness in lower limbs	Cisternal myelography showed block at lower lumbar region near cauda equina Biopsy confirmation of H.I.P.
Symonds and Blackwood[11]	F	55	Lower limb weakness since age of 35	Myelography demonstrated thickened nerve roots with blocked flow at C_3 level
Tenner[12]	—	—	—	Myelography

* Andermann *et al.* reported 3 relatives of this patient: 1 was diagnosed by autopsy; the other 2 by presence of clinical signs and familial association.
— Not given.

have been reported, and some of these have had an associated block.[5,8] These may represent a variation of H.I.P. with isolated nerve root involvement, but this is uncertain since no biopsies were taken.

SUMMARY

The occurrence of hypertrophic interstitial polyneuritis (H.I.P.) in 2 children, each of whom had characteristic roentgenographic features, is presented.

H.I.P. rarely presents in childhood.

Myelography is recommended for all children having bizarre and progressive gait abnormalities, deficits in motor or sensory abilities, or both. These abnormalities often resemble those caused by Friedreich's ataxia or peripheral neuropathy. However, we feel that the myelographic findings, taken together with the minor spinal changes, are quite characteristic of H.I.P. in this age group.

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REFERENCES

1. Andermann, F., Lloyd-Smith, D. L., Mavor, H., and Mathieson, G. Observations on hypertrophic neuropathy of Déjerine and Sottas. *Neurology*, 1962, *12*, 712–724.
2. Austin, J. H. Observations on syndrome of hypertrophic neuritis: hypertrophic interstitial radiculo-neuropathies. *Medicine*, 1956, *35*, 187–237.

3. BELLON, E. M., KAUFMAN, B., and TUCKER, M. E. Neuropathy: plain film and myelographic changes. *Radiology*, 1972, *103*, 319–322.
4. BLACKWOOD, W., MCMENEMEY, W. H., MEYER, A., NORMAN, R. M., and RUSSELL, D. S. Greenfield's Pathology. Second edition. Edward Arnold & Company, London, 1963, pp. 643–646.
5. GULATI, D. R., and ROUT, D. Myelographic block caused by redundant lumbar nerve root: case report. *J. Neurosurg.*, 1973, *38*, 504–505.
6. HINCK, V. C., and SACHDEV, N. S. Myelographic findings in hypertrophic interstitial neuritis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 947–948.
7. LEWTAS, N. A., and DIMANT, S. Diagnosis of hypertrophic interstitial polyneuritis by myelography. *J. Fac. Radiologists*, 1957, *8*, 276–279.
8. LOMBARDI, V. Redundant nerve root of cauda equina: case report. *Neurology*, 1969, *19*, 1223–1224.
9. MITCHELL, G. E., LOURIE, H., and BERNE, A. S. Various causes of scalloped vertebrae with notes on their pathogenesis. *Radiology*, 1967, *89*, 67–74.
10. SHAPIRO, R. Myelography. Second edition. Year Book Medical Publishers, Inc., Chicago, 1968, pp. 385–387.
11. SYMONDS, C. P., and BLACKWOOD, W. Spinal cord compression in hypertrophic neuritis. *Brain*, 1962, *85*, 251–260.
12. TENNER, M. S. Myelography of nonmass lesions in spinal canal. *Seminars in Roentgenol.*, 1972, *7*, 277–296.

VOL. 122, NO. 1

RECENT EXPERIENCE WITH INTRATHECAL RADIOGOLD FOR MEDULLOBLASTOMA AND EPENDYMOBLASTOMA: A PROGRESS REPORT*

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THE cure of patients with medulloblastoma following surgery and total neural axis irradiation remains disappointingly elusive for this very radioresponsive tumor. Five year survival rates of 25–40 per cent have been reported.[1,4,7]

Recurrent tumor at the primary site is the most frequent cause of treatment failure, but a number of patients also develop disease outside of the posterior fossa.[6] The spinal leptomeninges often are involved in spite of initial "prophylactic" irradiation of the neural axis to relatively high doses. Retreatment for recurrent medulloblastoma seldom results in cure, so that complete ablation of the tumor at the time of initial treatment is of paramount importance.

Increasing the dose of external irradiation runs the risk of causing central nervous system damage. An attempt to increase the dose delivered to the leptomeninges without running this risk has been made by supplementing external irradiation with colloidal radiogold (Au^{198}) instilled intrathecally. The rationale, technical details, and early results have been reported elsewhere.[2,3,5] Those communications detailed the development of the cauda equina syndrome in some of the early cases treated by this method. Additional experience in 6 patients using intrathecal radiogold (I.T.R.G.) is here reported. Innovations in both the technique used and the dose of the radionuclide instilled have been made in the attempt to avoid the cauda equina syndrome.

MATERIAL AND METHOD

From September 1968 to May 1973, 9 patients, 8 with medulloblastoma and 1 with ependymoblastoma, received I.T.R.G. Each patient had a craniotomy for decompression and gross tumor removal, followed by postoperative total neuraxis external beam (telecobalt 60) irradiation. In the 6 patients treated initially by us the neural axis received 3,000–4,000 rads in 4 to 4.5 weeks. The posterior fossa was supplemented with an additional 500–1,000 rads. I.T.R.G. was instilled 6 weeks thereafter. This was repeated, usually twice, at 6 weekly intervals. Myelographic examination preceded each instillation of radiogold.

The technique employed is as follows:

(1) The procedure is performed in the Diagnostic Radiology Department with the patient on a 0°–90° tilt table. Radiogold is instilled following myelography, with the patient's head tilted 15–20° down (Fig. 1). Immediately after the dose is administered, the table tilt is increased so that the patient is sharply head down.

(2) Each patient is comfortably suspended by the feet in a specially con-

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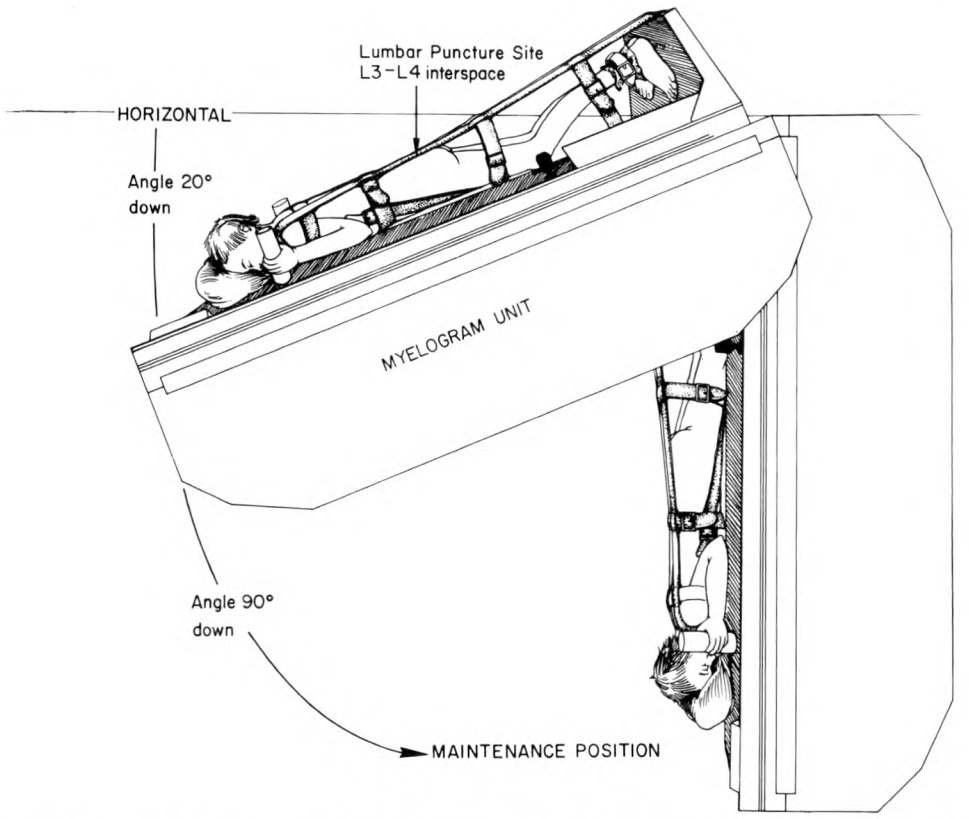


FIG. 1. Diagrammatic representation of patient in position for the intrathecal radiogold (I.T.R.G.) instillation, followed by the 90° head down position.

structed body harness to facilitate a 90° head down position (Fig. 2).

(3) The 90° head down position is maintained for most of 60–70 minutes following the instillation, with suitable rest periods during which the patient is returned to the horizontal position.

(4) External monitoring by hand-held dosimeters during this period is used to ensure even distribution of the radionuclide throughout the subarachnoid space.

(5) When external monitoring indicates even distribution throughout the spinal canal and the cranium, the patient is returned to the horizontal position. Thereafter, the patient lies in bed, but changes position every ½ hour from the supine to the left decubitus, right decubitus and prone positions. This is continued over a 5–6 hour post-instillation period.

(6) External scanning of the neuraxis 24 hours after the instillation is used to check distribution of the I.T.R.G. (Fig. 3).

(7) The scanning is repeated 3–4 days later to check for any residual "hot spots" (Fig. 4, *A* and *B*). These findings are used when planning subsequent I.T.R.G. instillations.

RESULTS

The patients in the study can be divided into 2 groups:

A. Patients treated initially according to the full protocol; *i.e.*, craniotomy, total neuraxis irradiation, and I.T.R.G. There were 6 such patients, 4 females and 2 males, with ages ranging from 2–14 years. The average age was 6 years. Five patients are

alive and well, 42, 36, 32, 13, and 12 months after treatment (Table 1). The 6th, a girl, died of widely disseminated systemic metastases as well as recurrent intracerebral and intraspinal disease. She survived 38 months following the initial diagnosis. One of the survivors developed right-sided hemiparesis after occlusion of the left internal carotid artery. This occurred 14 months after the second of 2 instillations of I.T.R.G. At last follow-up, he had improved and had only slight left-sided weakness and mild aphasia, and has no evidence of tumor.

B. Patients with recurrent disease after external therapy. These patients were retreated using both external sources and I.T.R.G. There were 3 such patients treated palliatively: 2 are known to be dead and the third has been lost to follow-up (Table II).



FIG. 2. A patient in the 90° head down position following instillation of I.T.R.G

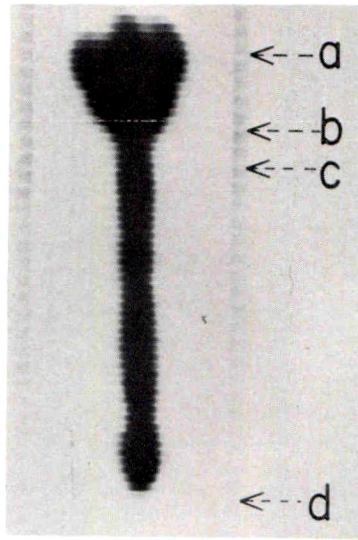


FIG. 3. Anterior total neural axis scan 24 hours following therapeutic I.T.R.G. showing satisfactory distribution of the radiocolloid .(a) Intracranial subarachnoidal distribution; (b) basilar cisterns; (c) cervical subarachnoid; (d) inferior extent of spinal subarachnoid space.

DISCUSSION

The cauda equina syndrome was a troublesome complication in the early cases treated with intrathecal radiogold. No instances of this complication have been noted in 9 patients treated according to the method described here. One of the key features is the head down position maintained throughout most of the period during and after the instillation of the radionuclide. This promotes early cephalad flow of the radiogold away from the caudal sac. Because the colloidal preparation fixes rapidly to the parieties of the spinal

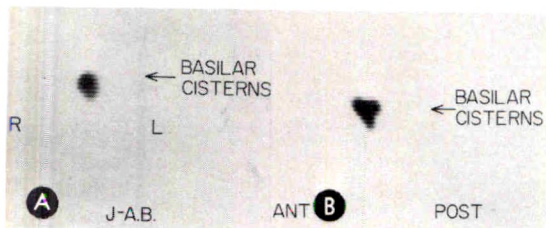


FIG. 4. (*A* and *B*) Total neural axis scan at 96 hours following I.T.R.G. and showing a residual "hot spot" in the area of the basilar cisterns.

TABLE I

INITIAL TREATMENT WITH INTRATHECAL RADIOGOLD (I.T.R.G.)—MEDULLOBLASTOMA AND EPENDYMOBLASTOMA

Patient and Sex	Age (yr.)	Date of Dx.	No. I.T.R.G. Instillations (10 mc)	Side Effects	Status (Last FU)	Survival Dx. to Last FU
1. F	2	11/69	3	F	NED	42 mo.
2. F	11	11/69	3	F, HA, N & V, P	Dead	38 mo.
3. M	4	5/70	2	F	NED	36 mo.
4. M*	2	12/70	2	F	NED	32 mo.
5. F	14	8/72	3	P, HA, F	NED	13 mo.
6. F	5	9/72	3	F	NED	12 mo.

* Diagnosis of ependymoblastoma.
F = fever; HA = headache; N&V = nausea and vomiting; P = pain.
NED = no evidence of disease.
FU = follow-up.

canal, it is essential that the radionuclide be prevented from pooling in the lumbar hollow, thus giving high dose to the strands of the cauda equina. An additional feature is a reduction in the amount of the radionuclide given on each occasion. Past experience indicated that 2 or 3 instillations of 10 mc of Au^{198} were well tolerated, and this dose has become a standard. Also, in very young children, 2 rather than 3 instillations have been employed when the follow-up scans suggested a "hot spot" anywhere along the cranial-spinal axis.

The reason for occlusion of the left internal carotid artery in one of our survivors is unknown. No similar complication was encountered in any of the children treated with I.T.R.G. in this series, or in those in the earlier study. It is possible that pooling of the radionuclide within the basilar cisterns may have resulted in heavy irradiation of the vessel, resulting in thrombosis, but the exact cause for the complication remains unclear.

Survival data for the 10 medulloblastoma patients treated at the University of Minnesota using combined external irradiation and I.T.R.G. are herein included and are based on the latest follow-up data by Smith *et al.*[8] Life table analysis of the survival in those patients, combined with the 5 medulloblastoma patients here re-

TABLE II

RECURRENT MEDULLOBLASTOMA PATIENTS—RETREATMENT WITH EXTERNAL BEAM IRRADIATION AND I.T.R.G.

Patient Data		No. I.T.R.G. Instillations (10 mc ea.)	Elapsed Time (mo.)		Total Survival Time	Side Effects
No. and Sex	Age		1st Dx. to 1st Recurrence	I.T.R.G. to Last FU		
7. M	11	1 (12 mc)	26	52 (dead)	78	+
8. M	9	3	21	9 (lost)	30	+
9. M	6	3	39	19 (dead)	58	+

I.T.R.G. = intrathecal radiogold.
+ = Local pain, nausea and vomiting, fever to 102° F. and headache.

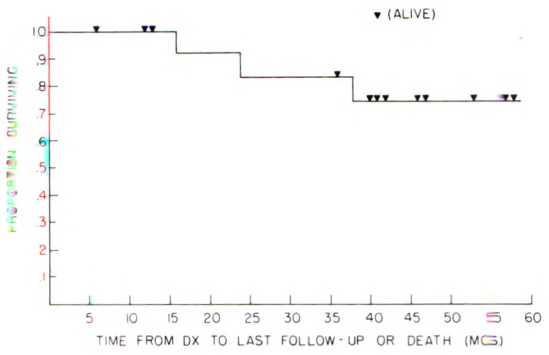


FIG. 5. Actuarial survival curve (15 patients). Eight of 12 are alive 36 months or more. (Ten patients University of Minnesota Series; 5 patients Memorial Sloan-Kettering Cancer Center Series.)

ported shows a 75 per cent projected long-term survival of 3 years or more (Fig. 5). These results are encouraging, especially because the cauda equina syndrome appears avoidable by use of the method here detailed.

SUMMARY

Nine patients, 8 with medulloblastoma and 1 with ependymoblastoma, were treated with intrathecal colloidal Au^{198}. Six of these patients were treated for cure using a combination of surgery, total neuraxis irradiation from external sources, and the intrathecal radionuclide. Five of these patients are alive and free of disease for periods ranging from 12 to 42 months (32 months median).

The cauda equina syndrome, a complication encountered in some patients treated earlier with intrathecal radiogold, appears to have been avoided with the use of a new technique. This calls for the use of smaller doses of radionuclide, and prompt cephalad flow of the radioactive material by tilting the head down immediately after instillation of the isotope.

A projected long-term survival (3 years or more) of 75 per cent in a collected group of patients is encouraging.

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REFERENCES

1. BLOOM, H. J. G., WALLACE, E. N. K., and HENK, J. M. Treatment and prognosis of medulloblastoma in children: study of 82 verified cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *105*, 43–62.
2. D'ANGIO, G. J., FRENCH, L. A., STADLAN, E. M., and KIEFFER, S. A. Intrathecal radioisotopes for treatment of brain tumors. *Clin. Neurosurg.*, 1968, *15*, 288–300.
3. GOLD, L. H., KIEFFER, S. A., D'ANGIO, G. J., FALLON, V. T., and LONG, D. M. Current status of intrathecal radiogold in treatment of medulloblastoma. *Acta radiol.* (Therapy), 1972, *11*, 329–340.
4. JENKIN, R. D. T. Radiation therapy of medulloblastoma in childhood. *Canad. M. A. J.*, 1969, *100*, 51–53.
5. KIEFFER, S. A., D'ANGIO, G. J., and NOWAK, T. J. Laboratory studies of intrathecal radiogold with new rationale for its use. *Radiology*, 1966, *87*, 1120–1121.
6. MCFARLAND, D. R., HORWITZ, H., SAENGER, E. K., and BAHR, G. K. Medulloblastoma: review of prognosis and survival. *Brit. J. Radiol.*, 1969, *42*, 198–214.
7. PEARSON, D. Medulloblastoma. In: The Treatment of Malignant Disease by Radiotherapy. Second edition. Edited by R. Paterson. Williams & Wilkins Company, Baltimore, 1963, p. 453.
8. SMITH, C. E., LONG, D. M., JONES, T. K., JR., and LEVITT, S. H. Medulloblastoma: analysis of time-dose relationships and recurrence patterns. *Cancer*, 1973, *32*, 722–728.

AN ELUSIVE AND OFTEN UNSUSPECTED CAUSE OF STRIDOR OR PNEUMONIA (THE ESOPHAGEAL FOREIGN BODY)*

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ESOPHAGEAL foreign bodies are not uncommon in young children and the usual presenting symptoms of excessive drooling, poor feeding, and dysphagia are familiar to most generalists, pediatricians and radiologists. Less well appreciated are those children whose primary presenting complaints are predominantly respiratory. In these infants, symptoms of stridor, wheezing, or chronic pneumonias cause one to focus initial attention on the respiratory tract. Esophageal symptoms, if present at all, may be completely overshadowed by the severe degree of respiratory difficulty, and, in fact, may completely elude the initial examiner. Failure to consider this possibility as a cause of respiratory difficulty can lead to serious complications, and in some cases, the unnecessary employment of potentially hazardous diagnostic procedures, such as tracheoscopy and bronchoscopy. This latter point is especially pertinent, as in most cases diagnosis can be made with simple and relatively innocuous radiologic examinations.

REPORT OF CASES

The case reports are summarized in Table 1. Following is a more detailed account.

CASE 1. E.C. This 15 month old white male was well until November 11, 1971, when he had an episode of fever, cough and wheezing. He was seen, at that time, in an outlying community and was treated with antibiotics. However, he never became completely symptom-free, and indeed, continued to have a cough, mild substernal and intercostal retractions, and noisy stridorous breathing. His family also related that he had been vomiting solid food since that time, but somehow this portion of the history was lost in the preoccupation with the respiratory problems. In addition, and interestingly enough, a chest roentgenogram was not obtained at this time.

On admission to our hospital, 5 months after the initial episode, physical examination revealed bilateral, diffuse ronchi, and mild inspiratory stridor. Suprasternal and intercostal retractions were again present. The admitting diagnosis was a vascular ring, although an airway foreign body was also a consideration. The possibility of an esophageal foreign body producing these symptoms was still not included in the list of diagnostic possibilities.

Roentgenographic examination showed a normal chest roentgenogram except that the mid portion of the tracheal air column was poorly defined, and perhaps somewhat displaced to the left (Fig. 1). A lateral view of the neck in deep inspiration showed overdistention of the pharynx, hypopharynx, and upper trachea. Below the level of the manubrium, the tracheal air column again appeared obliterated (Fig. 2*A*). A lesion around the mid-trachea was suspected, but it was not until a forcibly extended view of the neck was obtained (this is most important) that a plastic button was identified in the esophagus (Fig. 2*B*). Localized soft tissue swelling around this area was clearly producing narrowing of the adjacent tracheal airway. Subsequent barium swallow confirmed the presence of the button in the esophagus, but, in addition, showed a deep anterior ulceration (Fig. 3). This deep ulcer, and its surround-

* Presented at The Seventy-fourth Annual Meeting of the American Roentgen Ray Society, Montreal, P. Q., Canada, September 25–28, 1973. A Scientific Exhibit on the same subject was awarded the Gold Medal (see Vol. *119*, p. 639).

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TABLE I

SUMMARY OF CASES

Case No.	Age	Presenting Symptoms	Duration of Symptoms	Esophageal Symptoms	Type of Foreign Body	Diagnosis Established
I.	15 mo.	Persistent cough and stridor	5 mo.	Vomited solid foods	Plastic button	With lateral neck roentgenogram
II.	9 mo.	Stridor	2 da.	Poor feeding	Plastic toy	With lateral neck roentgenogram and barium swallow
III.	21 mo.	Stridor	2 hr.	None	2 pennies	With lateral neck roentgenogram
IV.	4 yr.	Pneumonia	4 mo.	None	Key chain snap	With chest roentgenogram
V.	13 mo.	Recurrent pneumonia	3–4 mo.	None	Metal clip	With chest roentgenogram

ing inflammatory reaction, was located in the area of tracheal narrowing.

Esophagoscopy was subsequently performed and the button was found at the level of the cricopharyngeus muscle. It was removed, the paraesophageal inflammation subsided, and the patient made an uneventful recovery. Two months after this episode, repeat barium swallow showed a persistent ulcer, but the patient was asymptomatic.

CASE II. M.J.S. This 9 month old white female was well until 10 A.M. on the day of her admission to John Sealy Hospital. At that time she was playing on the floor and developed acute respiratory distress with stridorous breathing. Physical examination in the Emergency Room revealed marked high pitched inspiratory stridor without an expiratory component. She was admitted with the tentative diagnosis of upper airway obstruction secondary to a laryngeal foreign body and underwent emergency laryngoscopy and bronchoscopy. At this time no intraluminal tracheal or laryngeal foreign body was demonstrated, but it was the laryngoscopist's opinion that the trachea was deviated to the right and that a mediastinal mass was present. Chest roentgenograms were obtained prior to laryngoscopy, but were interpreted by nonradiologists. Although abnormal, they were interpreted as being normal (Fig. 4).

Two days later the infant again developed severe inspiratory stridor and a radiologic consultation was obtained. The chest roentgenogram illustrated in Figure 4 was reviewed, and it was determined that the mediastinum was wider than normal and that the trachea was indeed displaced to the right. In addition a

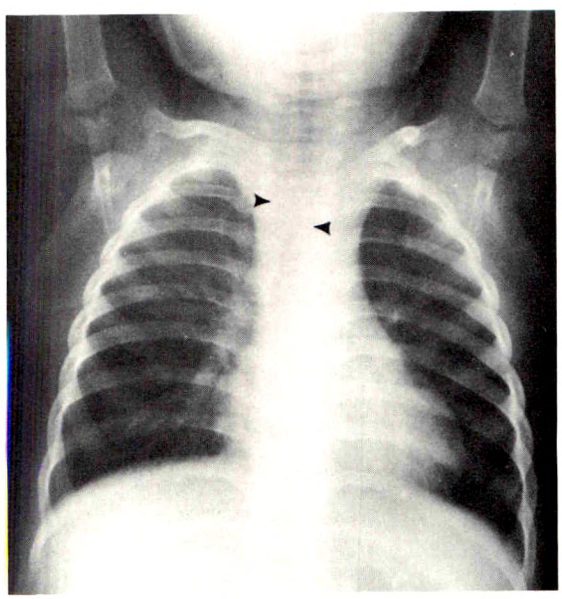


FIG. 1. Case 1. Chest roentgenogram is near normal, except that the tracheal air column is obliterated midway between the larynx and the carina (arrows). This finding is quite subtle and could be overlooked.

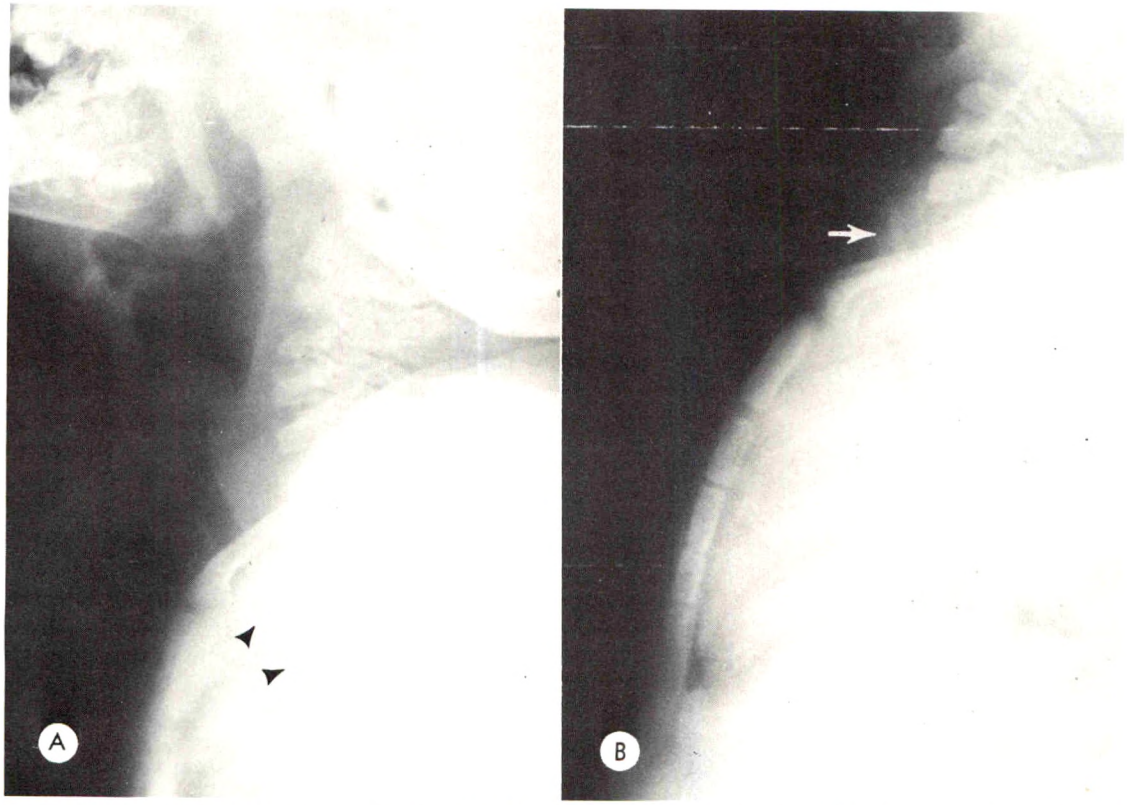


FIG. 2. Case I. (*A*) Lateral neck study with deep inspiration and moderate extension shows overdistention of the pharynx, hypopharynx and upper trachea. The tracheal air column is again obliterated at the level of the manubrium (arrows). If a laryngeal obstructing lesion was present, the upper trachea would probably not be distended. The fact that the upper trachea was distended suggested an obstruction at the level of the manubrium. (*B*) More pronounced extension reveals an oval, radiopaque disk, lodged in the upper esophagus (arrow). This subsequently was demonstrated to be a button. Such hyperextension of the neck is most important in the demonstration of high esophageal foreign bodies. Also note surrounding paraesophagitis which is leading to further compression.

lateral view of the neck, with pronounced extension and deep inspiration, was obtained. The prevertebral soft tissues were judged to be wider than normal (Fig. 5), and in view of the fact that the air column was entirely normal, it was decided to proceed with a barium swallow examination.

Before giving the infant some barium to drink, it was inquired of the mother as to whether she had noticed any feeding pattern changes in the infant. This question was asked because the infant seemed to be drooling. The mother volunteered that the infant desired to drink only chocolate milk since the first episode of stridor, and it is interesting to note that this bit of historical data was not elicited until this time. With the first swallow or two of barium (the infant would swallow only small quantities), it became readily apparent that an upper esophageal obstruction was present. A radiolucent defect in the esophagus was outlined (Fig. 6) and the diagnosis of esophageal foreign body was made.

Following the esophagography, esophagoscopy was performed and a small plastic toy was removed from the esophagus. Subsequent to this the patient did well, and it was notable that not only did the respiratory and esophageal symptoms subside rapidly, but that the initial mediastinal widening (mediastinal edema and inflammation) showed concomitant disappearance (Fig. 7). Two days later the infant was discharged, completely asymptomatic.

CASE III. N.K. This 21 month old female was playing with her mother's coin purse and

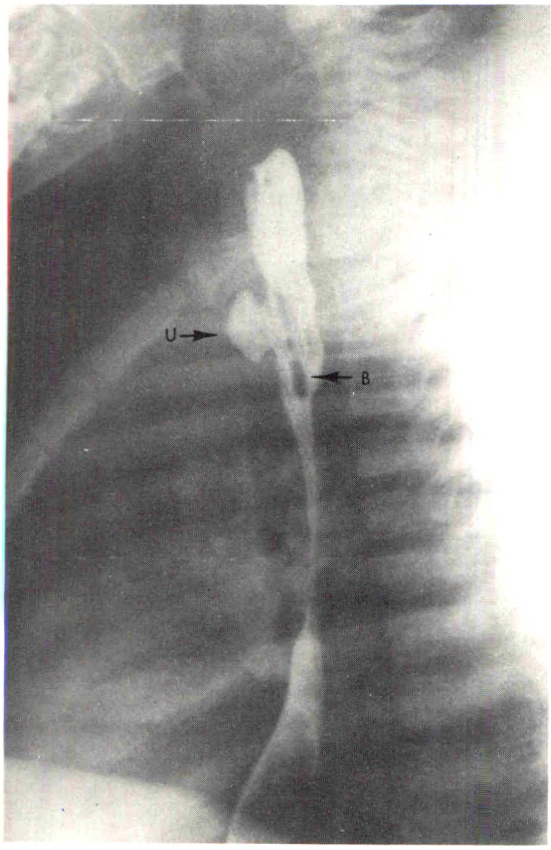


Fig. 3. Case 1. Barium swallow confirms the presence of the button (B), and in addition shows a deep anterior, collar button ulcer of the esophagus (U). It is readily apparent how this ulcer, and the adjacent paraesophageal inflammation produced compression of the adjacent trachea.

shortly after developed respiratory stridor which subsided just prior to her arrival in the Emergency Room. The child was turned upside down and spit up two pennies. However, prior to this maneuver a lateral neck roentgenogram was obtained and the 2 pennies were readily visualized (Fig. 8).

There was no difficulty in making the diag-

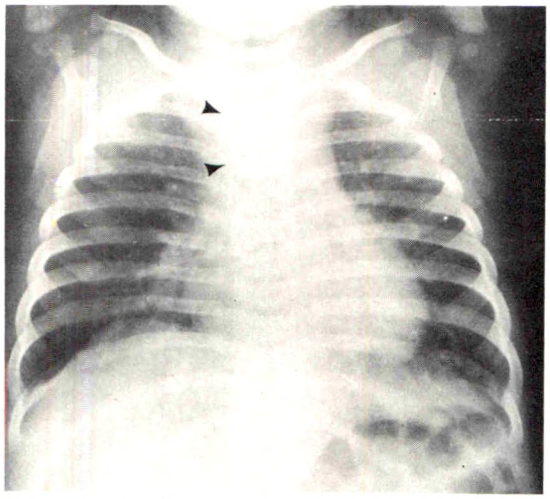


Fig. 4. Case 11. Chest roentgenogram shows moderate mediastinal widening and a little more than normal shift and deviation of the trachea to the right (arrows). None of these findings are strikingly abnormal (*i.e.*, superior mediastinal widening could be due to normal thymic tissue, while simple right-sided tracheal deviation is a common normal finding in infants).

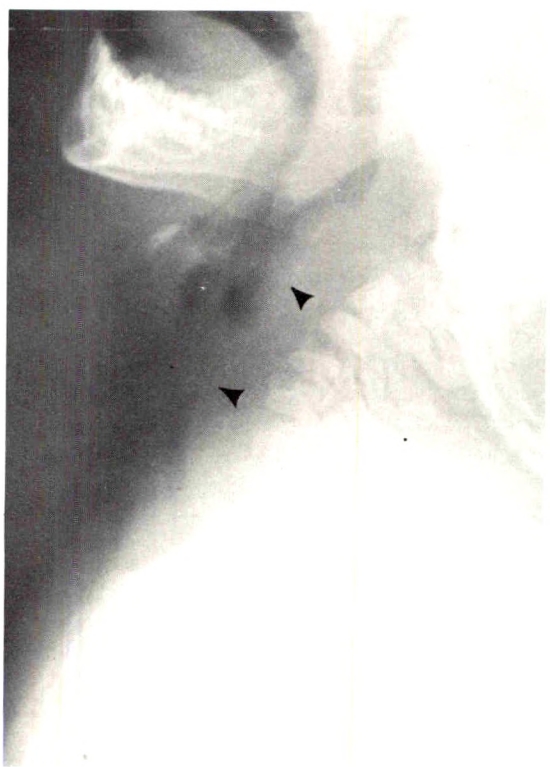


Fig. 5. Case 11. Lateral neck roentgenogram with moderate inspiration, but pronounced extension shows some thickening of the prevertebral soft tissues (arrows). Over-all, the findings are not very striking, and yet the prevertebral soft tissues do appear thickened, especially in the retropharyngeal region.

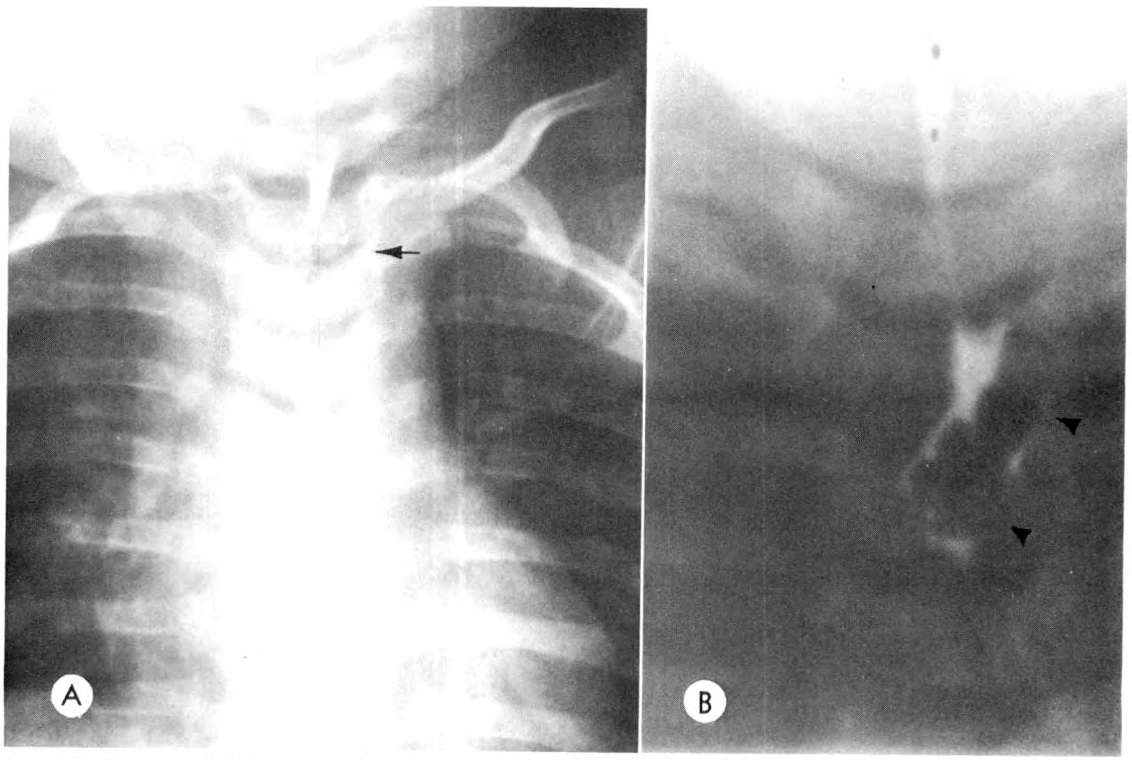


FIG. 6. Case II. (*A*) Barium swallow shows radiolucent filling defect (plastic toy) in the upper esophagus (arrow). The infant was unable to swallow much of the barium, and thus the foreign body is only partially outlined. (*B*) The findings were more striking during fluoroscopic examination, as shown on this spot roentgenogram (arrows).

nosis in this case, but it is included because the initial symptoms were those of stridor.

CASE IV. C.A. This 4 year old female presented in January, 1969, with fever, chills, and a productive cough. Actually she had had a chronic cough for a number of weeks. Physical findings were those of a right upper lobe consolidation, a finding confirmed with chest roentgenography (Fig. 9, *A* and *B*). However, the chest roentgenogram also revealed a metallic (key chain) foreign body in the lower esophagus. Review of a previous roentgenogram obtained because of an injury to the clavicle in this infant revealed that the foreign body was present, but not appreciated, 4 months earlier (Fig. 10). Indeed, the presence of the partially obstructing esophageal foreign body totally eluded clinical diagnosis at that time. On the present admission barium swallow confirmed the position of the foreign body (Fig. 11), and after this procedure esophagoscopy was performed. A metallic key chain was retrieved and after its removal the patient's course was uneventful. The right upper lobe pneumonia with which this infant presented on the current admission was believed to be secondary to aspiration, resulting from partial esophageal obstruction.

CASE V. C.M.C. This 13 month old female presented with a history of recurrent pneumonias of several months' duration. She was treated on numerous occasions for these pneumonias, and was actually referred for a work-up of failure to thrive, possible immunologic deficit, or some other form of chronic pulmonary disease.

Initial chest roentgenograms revealed indistinct parahilar peribronchial infiltrates, but in addition, an opaque foreign body in the upper esophagus (Fig. 12*A*). The presence of this foreign body was not suspected on clinical grounds and was a totally unexpected finding. It was also visible on the lateral view, wherein marked anterior displacement of the upper

trachea, resulting from extensive inflammatory reaction around the foreign body, was also seen (Fig. 12*B*). It was interesting that this infant had so accommodated to this foreign body that if any symptoms of dysphagia were present, they were completely obscured. The admitting problem was that of recurrent pneumonias.

Esophagoscopy was performed after the chest roentgenograms were obtained and a metallic clip was retrieved from the upper esophagus. Subsequent to this, the inflammatory reaction subsided and the infant pursued an uneventful course.

DISCUSSION

Esophageal foreign body, as a cause of respiratory distress in children, has been well documented in the otolaryngology literature.[2,4,6–8,10,12,13] Unfortunately, this problem has received far less attention in the radiologic and pediatric literature,[1,3,5,9] and while it is true that the otolaryngologist is frequently eventually called upon to treat these infants, it still remains that the majority of these children are initially seen by pediatricians, general practitioners, and

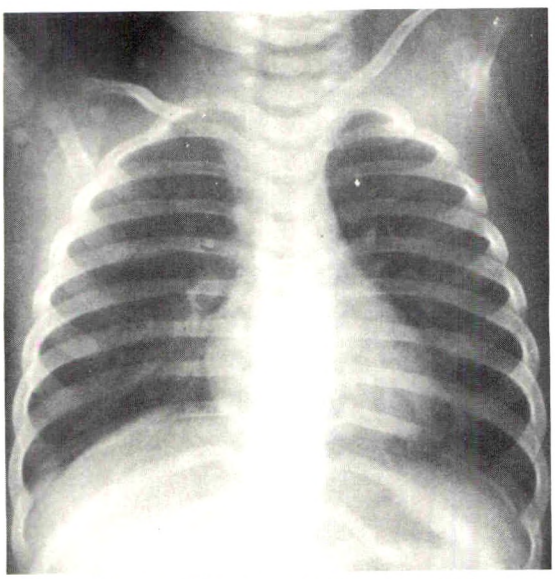


Fig. 7. Case II. Chest roentgenogram obtained 24 hours after removal of foreign body. Note that the superior mediastinal widening has regressed. The trachea also has returned to a more midline position. The decrease in mediastinal width was probably due to subsiding paraesophageal inflammation.

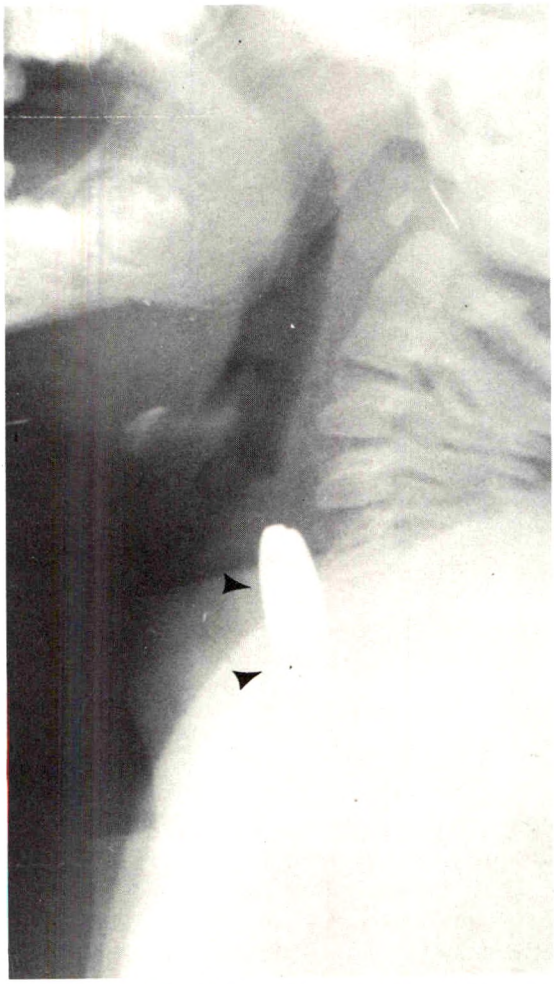


Fig. 8. Case III. Note 2 coins lodged in the upper esophagus (arrows). The initial symptoms in this patient were those of acute respiratory distress and stridor.

their radiologic colleagues. Consequently, it is most important that all of these individuals be completely aware of the fact that esophageal foreign bodies can cause a variety of respiratory symptoms, and that at times these may overshadow any esophageal problems. Further complicating the problem is the fact that potentially hazardous procedures such as tracheoscopy and bronchoscopy are often performed before more simple (and usually diagnostic) studies such as lateral neck roentgenograms and barium swallow roentgenograms are obtained. One can just imagine

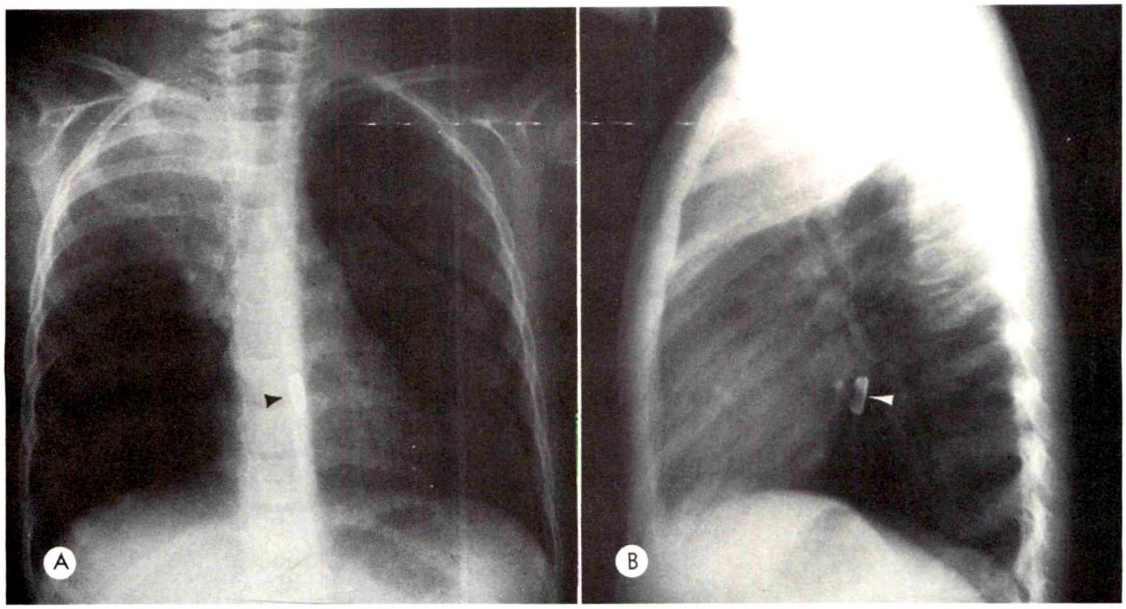


FIG. 9. Case IV. (*A*) Chest roentgenogram showing right upper lobe pneumonia and an opaque foreign body visible through the cardiac silhouette (arrow). (*B*) Lateral view again demonstrates the opaque foreign body (arrow), and the position would suggest that it is in the esophagus. The right upper lobe consolidation is again visible.

perforating the trachea in a case such as illustrated in Figure 2; the esophageal ulcer in this case was so near the tracheal wall that it is quite conceivable that a tracheoscope or bronchoscope could have shaved off the cap and created a tracheal perforation.

Jackson and Jackson,[10] in 1950, on reviewing over 1,400 cases of foreign bodies in the air and food passages found that a significant number had been overlooked for periods ranging from 1 month to 4 years. They attributed failure of the clinician to consider the diagnostic possibility of a foreign body as the primary reason for this delay in diagnosis. This experience was echoed by Glass and Goodman,[7] who in 1966 reported 9 instances of esophageal foreign body producing respiratory difficulty. Eight of their 9 patients had been seen and treated by pediatricians and general practitioners for periods ranging from 3 weeks to 1 year for such conditions as asthma, allergy, croup, and acute or recurrent pneumonias. In none of these cases was esophageal foreign body considered by the referring physicians. Our cases, as well as those in still other reports[2,6,7,11–13,15,17] reflect a similar delay in establishing the

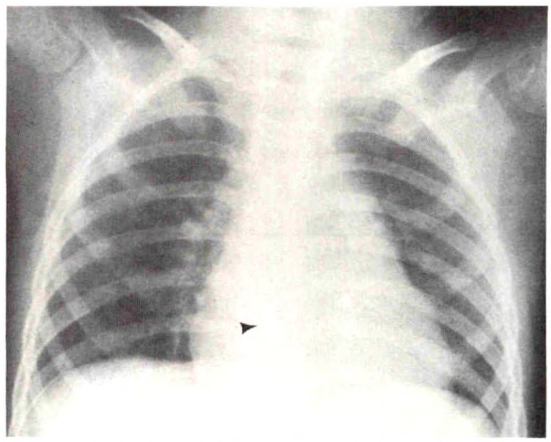


FIG. 10. Case IV. Chest roentgenogram obtained in another institution 4 months prior to that illustrated in Figure 9, *A* and *B*. This chest roentgenogram was obtained because of a suspected clavicular fracture, but note that the opaque foreign body, just barely visible through the cardiac silhouette (arrow) was present at this time. However, it was completely overlooked, both clinically and roentgenographically.

correct diagnosis. Principally, this was due to a failure to consider esophageal foreign body as a possible cause for the respiratory difficulty.

The most frequent site of impaction of foreign bodies in the esophagus is the cervical segment just below the cricopharyngeus muscle. Jackson and Jackson[10] postulated that large objects lodge here because of the weakness of peristalsis immediately below the cricopharyngeus constrictors and, to be sure, almost 90 per cent of esophageal foreign bodies will be found at this location.[8] Four of our cases showed the foreign body to be in this location. Other less frequent sites of lodgement include the area of physiologic narrowing caused by the left main stem bronchus as it crosses the esophagus and the gastroesophageal junction.[17]

Initial symptoms usually produced by esophageal foreign bodies include coughing, choking, excessive drooling, and inability to swallow. On a long-term basis failure to thrive may also be a problem.[1] It is interesting that many of the initial symptoms soon subside and are followed by a relatively

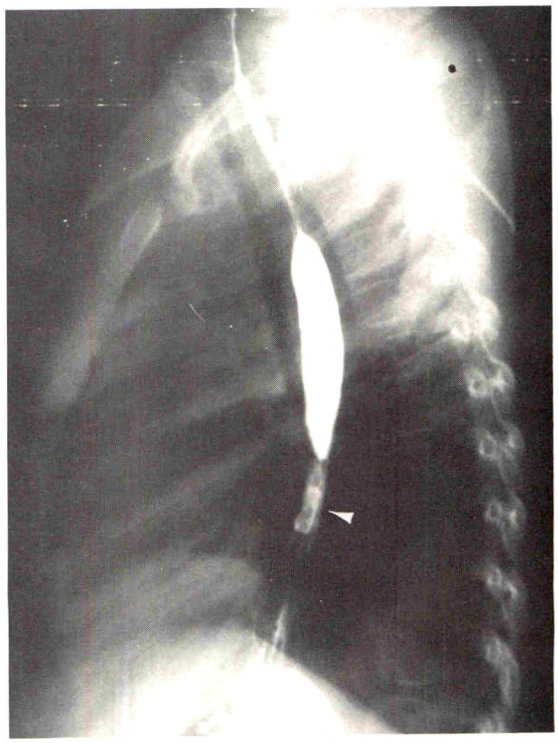


FIG. 11. Case IV. Lateral barium swallow confirms the presence of the foreign body in the lower esophagus (arrow). A metal key chain subsequently was removed from this infant's esophagus.

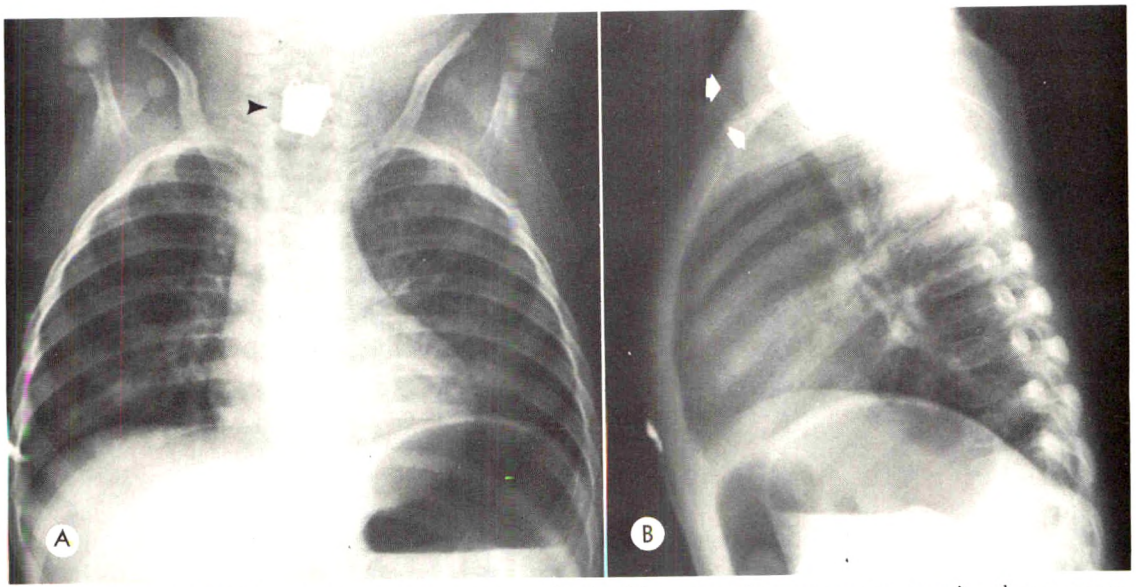


FIG. 12. Case V. (*A*) Frontal chest roentgenogram obtained because of recurrent pneumonias shows a completely unsuspected foreign body (arrow). (*B*) Lateral view shows the foreign body to lie in the upper esophagus. There is pronounced secondary paraesophageal inflammation which produced marked anterior displacement and compression of the adjacent trachea (arrows). The foreign body was a metal clip from a set of venetian blinds.

symptomless interval. Evidently the stimulus and irritation produced by the foreign body progressively decreases, and a point may be reached where no dysphagia at all is present.[10,11,14] The absence of dysphagia in these cases has been attributed to the diet of children in the age group most commonly affected by esophageal foreign bodies. Most cases involve children 2 years of age or younger, and in these children a normal diet is formed mainly of liquids or soft foods which can pass around incompletely obstructing foreign bodies. In addition, it is remarkable how rapidly and efficiently these infants can, by voluntary selection, alter their diets to accommodate the esophageal obstruction (*i.e.*, Cases II and V).

In those infants where respiratory symptoms predominate one may encounter stridor, wheezing, findings simulating asthma or airway foreign body, persistent cough, and in some cases symptoms of acute or chronic pneumonia. Stridor may result simply from direct compression of the trachea by large objects lodged in the postcricoid region, and in this regard it should also be noted that since the wall between the normal trachea and esophagus is thin, even smaller foreign bodies can produce significant tracheal compression. In other cases compression results from paraesophageal inflammation or frank abscess formation. In rare instances direct extension of the inflammatory process to the larynx and trachea can occur.

The close proximity of the upper trachea to the esophagus also predisposes to tracheal aspiration and pneumonia. This can result from the obstruction produced by the esophageal foreign body or from reactive spasm of the cricopharyngeus muscle. Less commonly, aspiration occurs after tracheoesophageal fistula formation resulting from deep ulceration of the esophagus.[10]

The diagnosis of radiopaque esophageal foreign body is usually easily secured by the use of plain roentgenograms of the chest and neck. If the foregin body is radiolucent, secondary soft tissue changes or focal overdistention of the upper airway may lead one to suspect its presence. In this regard it is most important that lateral roentgenograms of the neck be obtained in all cases and that they be exposed in inspiration with the neck hyperextended. If this rule is not adhered to, foreign bodies will be overlooked. Flat objects such as buttons, disks, coins, etc., always lie with their greatest diameter in the coronal plane of the esophagus, and thus are usually best seen on lateral neck or chest roentgenograms[10] (Cases I and III).

If plain film roentgenograms fail to provide definitive diagnosis, barium swallow examination may be necessary. However, it is usually unnecessary to employ a barium capsule or cotton soaked pledglet to locate the foreign body, for the findings of esophageal obstruction, deviation of the barium stream, or an actual filling defect, etc., adequately call attention to the problem. Following the removal of esophageal foreign body, barium swallow study should be performed to exclude the presence of a predisposing stricture[6] or esophageal ulceration resulting from the foreign body itself. This is not to say that an underlying and predisposing esophageal lesion is found in all cases (indeed, in our experience the reverse is true), but rather that the status of the esophagus should be assessed, if not before esophagoscopy, shortly thereafter.

SUMMARY AND CONCLUSIONS

Children with esophageal foreign bodies may present with a variety of respiratory symptoms and few, if any, symptoms of esophageal obstruction.

Awareness of this presentation by the clinician and radiologist should lead to early diagnosis. This is most important, for if the diagnosis is not secured at an early stage, complications such as esophageal ulceration, esophageal perforation, chronic aspiration pneumonia, and failure to thrive can develop.

Early roentgenographic examination of the neck (lateral view with extension and

inspiration) and chest is essential and usually all that is needed for the diagnosis.

Barium swallow examination may be required in some cases, but potentially hazardous diagnostic procedures such as tracheoscopy and bronchoscopy should be avoided.

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REFERENCES

1. Buckler, J. M. A., and Stool, S. E. Failure to thrive: exogenous cause. *A.M.A. Am. J. Dis. Child.*, 1967, *114*, 652–653.
2. Bunker, P. G. Foreign body complications. *Laryngoscope*, 1961, *71*, 903–917.
3. Capitanio, M. A., and Kirkpatrick, J. A. Upper respiratory tract obstruction in infants and children. *Radiol. Clin. North America*, 1968, *6*, 265–277.
4. Emerson, E. B. Foreign bodies in airways and esophagus. *Am. J. Surg.*, 1963, *105*, 522–523.
5. Fearon, B. Acute layrngo-tracheo bronchitis in infancy and childhood. *Pediat. Clin. North America*, 1962, *9*, 1095–1112.
6. Friedberg, S. A., and Blustone, C. D. Foreign body accidents involving air and food passages in children. *Otolaryngol. Clin. North America*, 1970, *3*, 395–403.
7. Glass, W. M., and Goodman, M. Unsuspected foreign bodies in young child's esophagus presenting with respiratory symptoms. *Laryngoscope*, 1966, *76*, 605–615.
8. Hoeksma, P. E., and Huizinga, E. On foreign bodies and perforations of esophagus. *Ann. Otol., Rhin. & Laryng.*, 1971, *80*, 36–41.
9. Hollinger, P. H., and Johnson, K. C. Infant with respiratory stridor. *Pediat. Clin. North America*, 1955, *2*, 403–411.
10. Jackson, C., and Jackson, C. L. Broncho-esophagology. W. B. Saunders Company, Philadelphia, 1950, pp. 13–33.
11. Levin, N. M. Foreign bodies in larynx, bronchi and esophagus. *South. M. J.*, 1972, *65*, 213–216.
12. Missal, S. C. Extrapleural tumor causing compression of lung. *Ann. Otol., Rhin. & Laryng.*, 1963, *72*, 970–973.
13. Naftzger, J. B., and Gittens, T. R. Foreign bodies in esophagus with respiratory symptoms complicating diagnosis: report of four cases. *Laryngoscope*, 1926, *36*, 370–376.
14. Ochsner, J. L., and LeJeune, F. E. Tracheal and esophageal obstructions in infants. *South. M. J.*, 1964, *57*, 1333–1339.
15. Schwalb, E., and Trucco, J. What wheezes isn't always asthma. *New York J. M.*, 1967, *67*, 1075–1076.
16. Sparger, C. F. Foreign bodies in tracheo-bronchial tree and esophagus. *Rocky Mountain M. J.*, 1968, *65*, 47–52.
17. Wishahy, A., Khalifa, A. S., Abdel Fattah, S., and El-Kahky, M. L. Esophageal foreign body in child resulting in repeated asthmatic attacks. *J. Egyptian M. A.*, 1968, *51*, 213–216.

FETAL ASPIRATION SYNDROME*

CLINICAL, ROENTGENOLOGIC AND PATHOLOGIC FEATURES

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PULMONARY disease is the most common cause of morbidity and mortality in the early newborn. Only hyaline membrane disease (HMD) exceeds fetal aspiration syndrome (FAS) as a cause for respiratory distress during the first week of life. Whereas HMD usually affects the premature infant, FAS primarily affects the full-term or postmature newborn. The distressed, anoxic fetus *in utero* develops FAS by aspirating amniotic debris consisting of epidermal squames, lanugo hair and meconium.

MATERIAL

During a 5 year period (1968–1972), FAS was recognized clinically in 89 newborns at Pennsylvania Hospital, a regional referral center for neonatal respiratory distress. The 61 cases with roentgenograms form the basis for this study. Follow-up roentgenograms were obtained in the majority of these cases, either for verification of clinical improvement or for guidance and assistance in therapy in the presence of clinical deterioration. The 4 fatal cases with autopsies form the basis of the pathologic study.

CLINICAL FEATURES

The typical infant with FAS is full-term or postmature, has suffered intrauterine distress, and is delivered with meconium stained amniotic fluid. The infant develops mild to moderate respiratory distress in the immediate postnatal period.

Forty-seven (77 per cent) of the 61 infants were delivered vaginally. Birth weights ranged from 1,620 to 4,990 grams, with an average birth weight of 3,819 grams. There were 37 males (61 per cent) and 24 females (39 per cent).

Frequently, the presenting clinical sign of FAS is air-trapping with an increased anteroposterior diameter of the chest. This increased anteroposterior diameter of the chest alone often prompted roentgenographic study because of its association with pneumomediastinum (PM) and pneumothorax (PT). The thoracic cage demonstrates diminished excursion and is occasionally fixed in inspiration. Auscultation of the chest may be normal, but may reveal nonspecific rales or rhonchi.

Although amniotic sac contents are usually sterile, prophylactic antibiotics are given to prevent secondary pulmonary infection. None of the infants in this series was intubated or treated with a positive pressure ventilator. Rapid improvement in 12 to 36 hours, despite a high frequency of PM and PT, is characteristic of FAS. Only 4 deaths occurred.

ROENTGENOLOGIC FEATURES

Review of the roentgenograms in the 61 cases indicated that hyperaeration was the most common finding, occurring in 40 cases (66 per cent) (Fig. 1, *A* and *B*). Patchy areas of atelectasis and consolidation, usually bilateral, were noted in 38 cases (62 per cent) (Fig. 2). Small pleural effusions, occasionally manifested only by thickening of the interlobar fissures, were evident in 10 cases (16 per cent) (Fig. 3). The roentgenograms of 15 cases (25 per cent) suggested intrapulmonary interstitial emphysema (IIE). Since pulmonary interstitial air will not be noted roentgenographically unless zones of atelectasis or

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consolidation are juxta-opposed to it, as in an air bronchogram, IIE is probably more common but not appreciated roentgenographically. IIE is easier to discern in HMD than in FAS because of the enhanced roentgenographic contrast between the hypoaerated lungs and the pulmonary interstitial air.

An impressive feature in this study was the high frequency of PM and PT, occurring in 27 cases (44 per cent) and 19 cases (31 per cent), respectively. Twelve of these cases had IIE as well. Eighteen cases of PT were right sided, 7 were left-sided, and 6 were bilateral. Fifteen cases (79 per cent) of PT occurred in males. No cases of tension pneumothorax were encountered. The roentgenographic findings of neonatal IIE, PM, and PT have been well described and illustrated previously[2,4,8,9] and, therefore, will not be repeated in this paper.

The initial roentgenograms, usually obtained within 3 to 6 hours after birth, always displayed the most striking abnormalities. Subsequent roentgenograms showed remarkable clearing of the parenchymal densities and significant resolution of PM and PT often at 12 to 24 hours of age (Fig. 4, *A* and *B*; and 5, *A* and *B*). Serial studies in several cases indicated that, although striking resolution occurs within the first day, the chest roentgenogram is usually not completely normal until 4 to 8 days.

PATHOLOGIC FEATURES

Postmortem examinations were performed on 1 male and 3 female neonates, whose birth weights ranged from 1,620 to 3,040 grams. Despite his low birth weight, the smallest infant had histologically mature tissues. The placenta in this case was

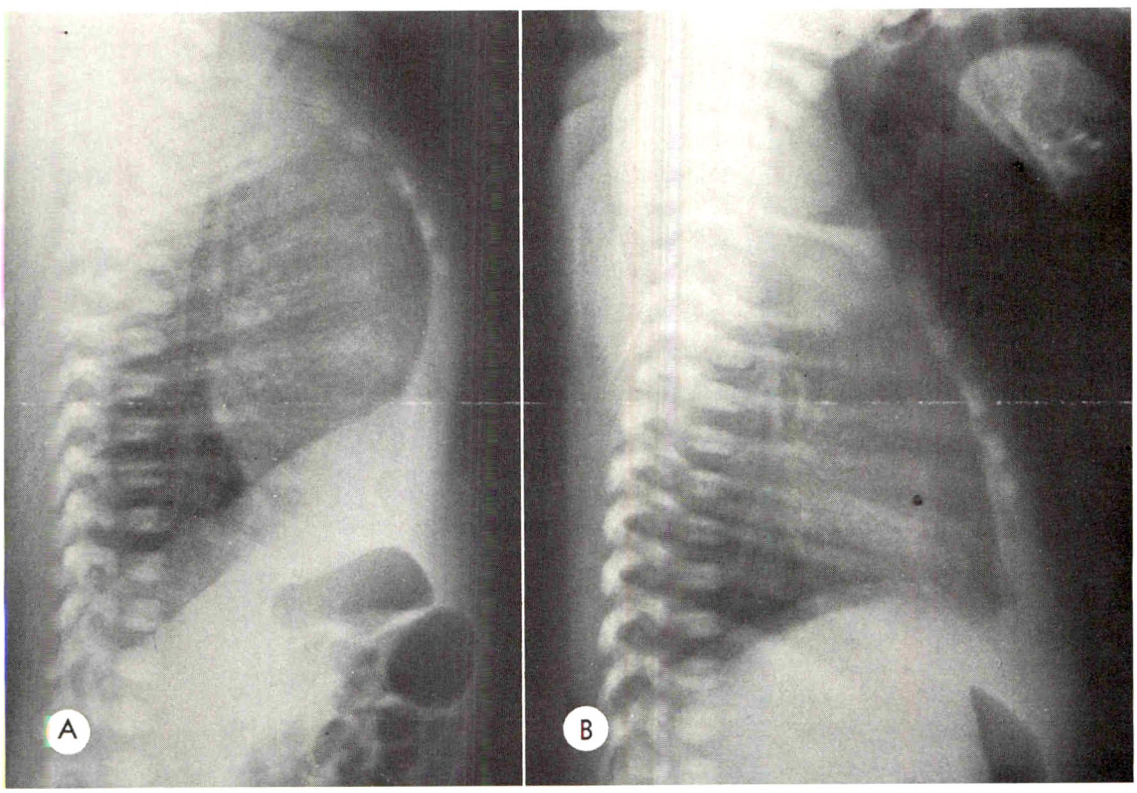


FIG. 1. (*A*) Lateral roentgenogram of a postmature newborn at 1 hour of age demonstrates hyperaeration by an increased anteroposterior diameter of the chest and flattened hemidiaphragms. (*B*) Repeat lateral roentgenogram 21 hours later reveals no sign of hyperaeration.

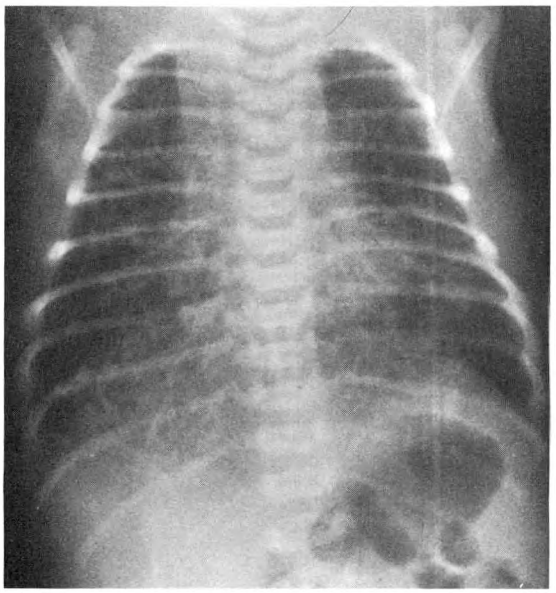


FIG. 2. Anteroposterior roentgenogram of a postmature newborn with fetal aspiration syndrome (FAS) demonstrates hyperaeration and bilateral pulmonary parenchymal densities.

circumvallate and reduced in weight. Two infants were postmature, 5 and 15 days. Three were delivered vaginally and 1 was delivered by cesarean section because of cephalopelvic disproportion. Postnatal survival ranged from 4 to 48 hours.

Meconium staining of amniotic fluid was noted at delivery in all 4 cases. In 2 cases yellow-green fluid could be expressed from the cut ends of the bronchi at the hilus of the lung. PM was present in 2 cases. The lungs were fleshy, reduced in crepitance, and the sectioned surfaces discolored yellow-green. Pulmonary interstitial air was noted grossly in 1 case. Microscopy, however, demonstrated sleeve-like perivascular dissection of air, often extending into the interlobular septa, in all cases (Fig. 6). Masses of folded squames occupied 50–75 per cent of the air spaces. Eosinophilic staining hyaline membranes lined terminal bronchioles and alveoli in the 3 cases in which survival ranged from 29 to 48 hours (Fig. 7), but not in the case in which death occurred at 4 hours.

DISCUSSION

Peterson and Pendleton[14] first emphasized the roentgenographic characteristics of FAS and contrasted this pattern with HMD. More recently Gooding and Gregory[10] have reported 75 cases in which they found significantly less PM and PT than was encountered in the present study. Air-trapping was reported as an uncommon finding in their cases. In our study, hyperaeration was the most frequent roentgenographic finding in FAS. This roentgenographic observation is important in understanding the pathogenesis of the complications of FAS. The importance of air-trapping and IIE in the formation of PM and PT in HMD has recently been reviewed;[2,3,8] and their role in FAS has also been suggested,[5,13] but certainly not emphasized in the radiologic literature.

Schaffer and Avery[16] mentioned fetal circulatory accidents, postmaturity, and placental insufficiency as causes of fetal distress that can lead to meconium passage and massive aspiration *in utero* or during

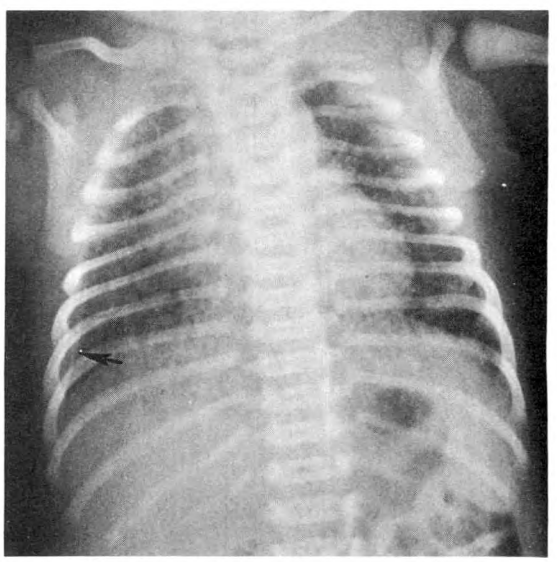


FIG. 3. Anteroposterior roentgenogram demonstrates hyperaeration, bilateral pulmonary parenchymal densities and a small pleural effusion (arrow) in a full-term newborn delivered through meconium stained amniotic fluid presenting with immediate postnatal mild respiratory distress.

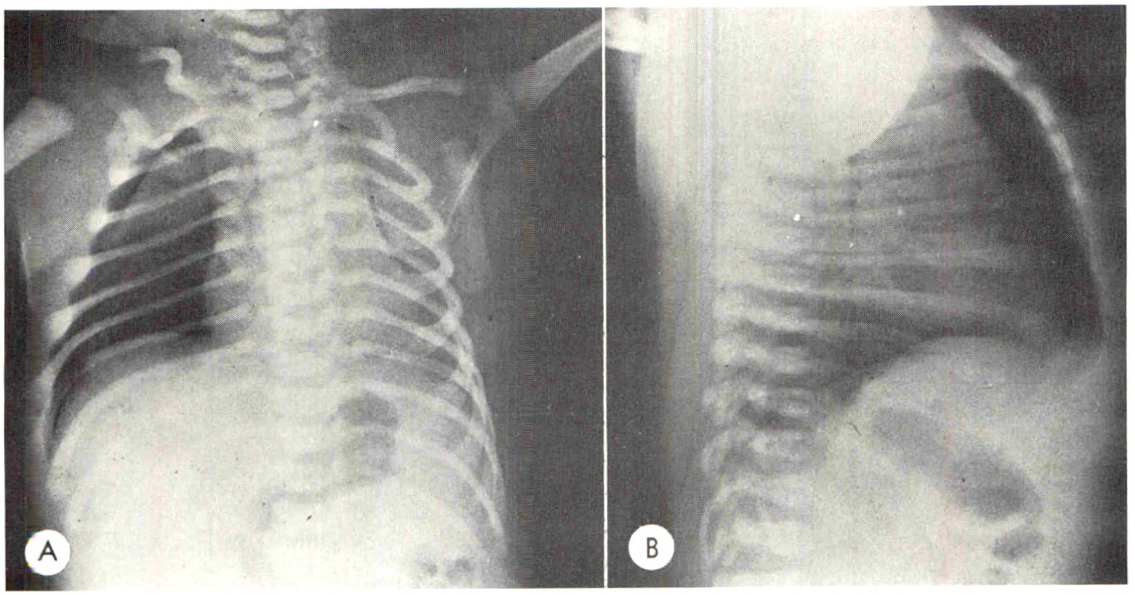


FIG. 4. (*A*) Postmature infant at 1 hour of age. The anteroposterior projection shows intrapulmonary interstitial emphysema in the mid and lower zones of the right lung and a nontension pneumothorax on the right. (*B*) Pneumomediastinum is seen in the lateral projection.

the first few breaths. In FAS, aspirated debris in the bronchi cause areas of atelectasis and consolidation. As the infant attempts to expand his lungs after birth, areas of alveolar overexpansion develop. These 2 processes developing simultaneously are recorded on the roentgenograms as patchy pulmonary densities and hyperaeration. Air in hyperinflated alveoli abutting on the pulmonary vessels may rupture

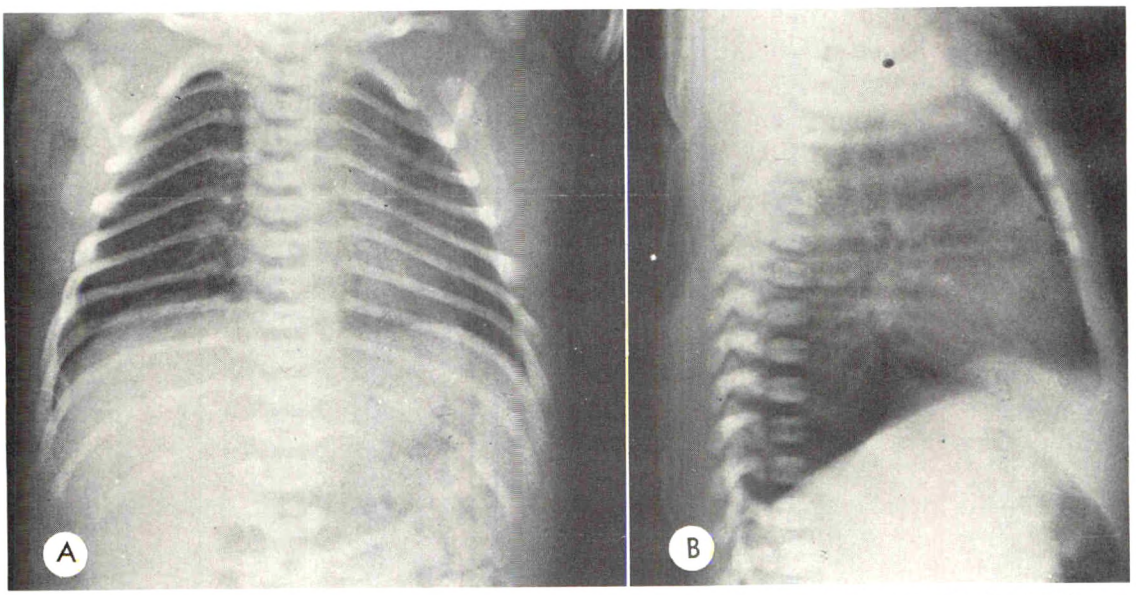


FIG. 5. (*A* and *B*) Repeat roentgenograms of the same infant as in Figure 4, *A* and *B*, obtained 20 hours later demonstrate almost complete disappearance of both the pneumothorax and pneumomediastinum.

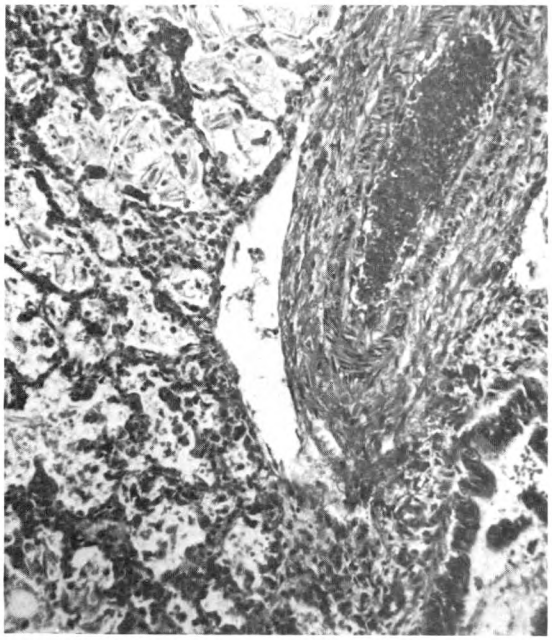


FIG. 6. Perivascular dissection by intrapulmonary interstitial emphysema. The alveoli at the left are packed with epidermal squames. (Hematoxylin and Eosin, 150×.)

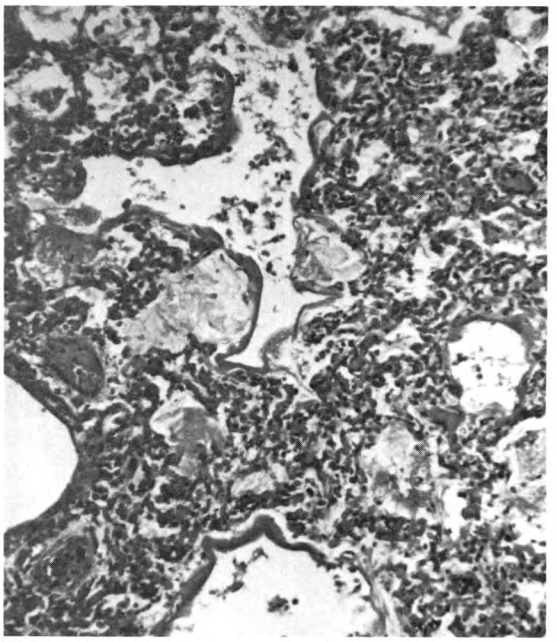


FIG. 7. Hyaline membranes line several terminal bronchioles and alveoli, many of which contain masses of epidermal squames. (Hematoxylin and Eosin, 150×.)

into the perivascular sheaths when the transpulmonary pressure created by the diaphragm causes a sufficient pressure gradient.[5] Subsequently this air may dissect through the supporting structures of the lung toward the hilus and ultimately rupture into the mediastinum and the pleural space, producing PM and PT.

The Macklins[13] noted that subpleural blebs occur with IIE, but are rarely ruptured at autopsy even when PT is present. The path of least resistance for IIE seems to be toward the mediastinum, not toward the pleural cavity. Therefore, PT probably arises from PM. The more frequent occurrence of PM than PT in this study supports this postulate. The rather benign behavior of PM and PT and lack of tension PT in FAS can be explained by the developmental maturity and normal compliance of the lungs in full-term newborns. Surgical decompression of PM and PT, even when quite large, is rarely necessary in FAS. With conservative, noninvasive management, the PM and PT rapidly resolve. The low mortality in FAS, even in those newborns with complications of extraventilatory air, sharply contrasts with the serious nature of these complications in HMD in which the hypoaerated, noncompliant lungs cannot accommodate so readily to IIE and PT.[3]

The 4 infants who died in this study were products of abnormal pregnancy or delivery: 1 was under-weight and had a circumvallate placenta, a condition which is associated with increased risk of fetal complications,[1] 2 were postmature, and 1 was delivered by cesarean section. The discovery of pulmonary hyaline membranes in 3 of the 4 fatal cases is unusual, since they are generally observed in the premature newborns with HMD.[11] Emery[6] found no hyaline membranes in his 15 cases of IIE due to FAS. Esterly *et al.*,[7] however, collected 46 cases of hyaline membrane formation in full size newborns, 10 of whom had gestational ages greater than 39 weeks. In 7 of these full size infants, hyaline membranes and FAS existed together. As noted

in the present cases, hyaline membranes in full-term newborns occur in both terminal bronchioles and alveolar spaces, whereas in the premature newborn, hyaline membranes lie in only the terminal bronchioles producing the pattern that Gruenwald[12] has termed the "atelectasis of prematurity." Although vernix is not thought to contribute directly to hyaline membrane formation, increased capillary permeability due to hypoxia which may exist in the newborn with FAS may contribute to hyaline membrane formation. Pulmonary vascular compression by interstitial emphysema may contribute to hypoxia by diminishing pulmonary blood flow.[6] The 3 infants in this study who had pulmonary hyaline membranes received 50–100 per cent inspiratory oxygen, and it has been demonstrated experimentally that increased oxygen can produce pulmonary hyaline membranes.[11] The thromboplastic activity of amniotic fluid may also enhance formation of pulmonary hyaline membranes from endogenous fibrin.[11]

CONCLUSION

The roentgenographic features of FAS are nonspecific. Patchy pulmonary parenchymal densities and hyperaeration may also occur in postnatal aspiration pneumonia. In addition, some cases of pulmonary hemorrhage, symptomatic hypoglycemia, obstructed pulmonary venous return, and congenital pulmonary lymphangiectasia may be difficult to distinguish roentgenographically from FAS.

Although early postnatal PM and PT may be the dominant roentgenographic features in FAS, the radiologist must be aware that these complications also can occur not only in some newborns with obstructed urologic malformations,[1] but also in some newborns insulted by overzealous postnatal resuscitation.

PM and PT in HMD generally develop from 12 to 48 hours of age as contrasted to their immediate postnatal occurrence in FAS. Nevertheless, by carefully considering the prenatal and obstetrical histories when confronted by the roentgenographic findings of hyperaeration and coarse, diffuse, linear radiodensities, frequently in combination with IIE, PM, and nontension PT, the radiologist should be able to recognize FAS as the cause of early neonatal respiratory distress.

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REFERENCES

1. Benirschke, K., and Driscoll, S. G. The Pathology of the Human Placenta. Springer-Verlag, Berlin, 1976, pp. 28–34.
2. Campbell, R. E. Intrapulmonary interstitial emphysema: complication of hyaline membrane disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 449–456.
3. Campbell, R. E., and Hoffman, R. R., Jr. Predictability of pneumothorax in hyaline membrane disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *120*, 274–278.
4. Chasler, C. N. Pneumothorax and pneumomediastinum in newborn. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *91*, 550–559.
5. Chernick, V., and Avery, M. E. Spontaneous alveolar rupture at birth. *Pediatrics*, 1963, *32*, 816–824.
6. Emery, J. L. Interstitial emphysema, pneumothorax and "air-block" in newborn. *Lancet*, 1956, *1*, 405–409.
7. Esterly, J. R., Langegger, F., and Gruenwald, P. Hyaline membranes in full size infants. *Virchows Arch. path. Anat.*, 1966, *341*, 259–270.
8. Fletcher, B. D., Outerbridge, E. W., and Dunbar, J. S. Pulmonary interstitial emphysema in newborn. *J. Canad. A. Radiologists*, 1970, *21*, 273–279.
9. Franken, E. A., Jr. Pneumomediastinum in newborn with associated dextroposition of heart. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 252–260.
10. Gooding, C. A., and Gregory, G. A. Roentgenographic analysis of meconium aspiration of newborn. *Radiology*, 1971, *100*, 131–135.
11. Gregg, R. H., and Bernstein, J. Pulmonary hyaline membranes and respiratory distress syndrome. *A.M.A. Am. J. Dis. Child.*, 1961, *102*, 871–890.
12. Gruenwald, P. Pulmonary pathology in respira-

tory distress syndrome. *Pediat. Clin. North America*, 1966, *13*, 703–710.

13. MACKLIN, M. T., and MACKLIN, C. C. Malignant interstitial emphysema of lungs and mediastinum as important occult complication in many respiratory diseases and other conditions: interpretation of clinical literature in light of laboratory experiment. *Medicine*, 1944, *23*, 281–351.
14. PETERSON, H. G., JR., and PENDLETON, M. E. Contrasting roentgenographic pulmonary patterns of hyaline membrane and fetal aspiration syndromes. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1955, *74*, 800–817.
15. RENERT, W. A., BERDON, W. E., BAKER, D. H., and ROSE, J. S. Obstructive urologic malformations of fetus and infant; relation to neonatal pneumomediastinum and pneumothorax: air-block. *Radiology*, 1972, *105*, 97–105.
16. SCHAFFER, A., and AVERY, M. E. Diseases of the Newborn. Third edition. W. B. Saunders Company, Philadelphia, 1971, pp. 70–76.

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CONGENITAL ABSENCE OF THE PULMONARY VALVE ASSOCIATED WITH CONGENITAL APLASIA OF THE THYMUS (DiGEORGE'S SYNDROME)*

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THE thymus is derived from the third pharyngeal pouch and the parathyroids are derived from both the third and fourth pharyngeal pouches.[4] Defective development of these 2 pouches results in hypoplasia or aplasia of the thymus and parathyroid glands accompanied clinically by neonatal hypocalcemia, defective thymic-dependent cellular immunity and facial anomalies.[4–6] This syndrome was first described in 1965 by DiGeorge,[2] who did not make reference to any associated cardiovascular abnormalities.

During the same year, Cameron[1] reported briefly upon 10 patients with absence or hypoplasia of the thymus but no evidence of hypoparathyroidism. All had congenital cardiovascular disease, including instances of ventricular septal defect, persistent truncus arteriosus, tetralogy of Fallot, aortic arch atresia or coarctation, and right aortic arch. Two patients had almost identical anomalies, consisting of absent pulmonic valve, aneurysmal dilatation of the central pulmonary arteries, ventricular septal defect, absent ductus arteriosus and emphysematous enlargement of the right upper lobe due to bronchial compression by the enlarged pulmonary vessels.

Kirkpatrick and DiGeorge[5] in 1968 made reference to the fact that we could expect other developmental anomalies of closely related analage such as the fourth branchial arch resulting in anomalies of the aortic arch. Recently, Freedom *et al.*[3] reviewed 23 cases of DiGeorge's syndrome and found that in 19 (83 per cent) aortic arch and/or intracardiac anomalies were present. The most common types of intracardiac disease were tetralogy (5 cases) and persistent truncus arteriosus (3 cases). One patient had transposition of the great arteries, pulmonary valve atresia, ventricular septal defect and a right patent ductus arteriosus. Another had pulmonary valve atresia, ventricular septal defect and patent ductus arteriosus. Still another had ventricular septal defect associated with interruption of the aortic arch and bicuspid aortic valve. One patient had isolated pulmonic stenosis. The patients generally come to medical attention during the neonatal period as a result of hypocalcemic tetany or seizures or symptoms suggesting congenital heart disease.

At the State University of New York, Downstate Medical Center, we have recently studied 2 patients with aplasia of the thymus and parathyroids and tetralogy of Fallot with complete absence of the pulmonic valve. They are thus somewhat similar to the 2 cases of absent pulmonic valve described by Cameron, although our patients had deficient development of both thymus and parathyroids, whereas Cameron's patients apparently had normal parathyroids. There is also similarity to the 5 cases of Freedom *et al.*, in that tetralogy of Fallot is present.

The purpose of this paper is to call the attention of radiologists to the association of tetralogy of Fallot and absence of the pulmonic valve with DiGeorge's syndrome, as well as to discuss the reasons why these cases failed to exhibit certain roentgenographic signs commonly associated with absence of the thymus and pulmonic valve.

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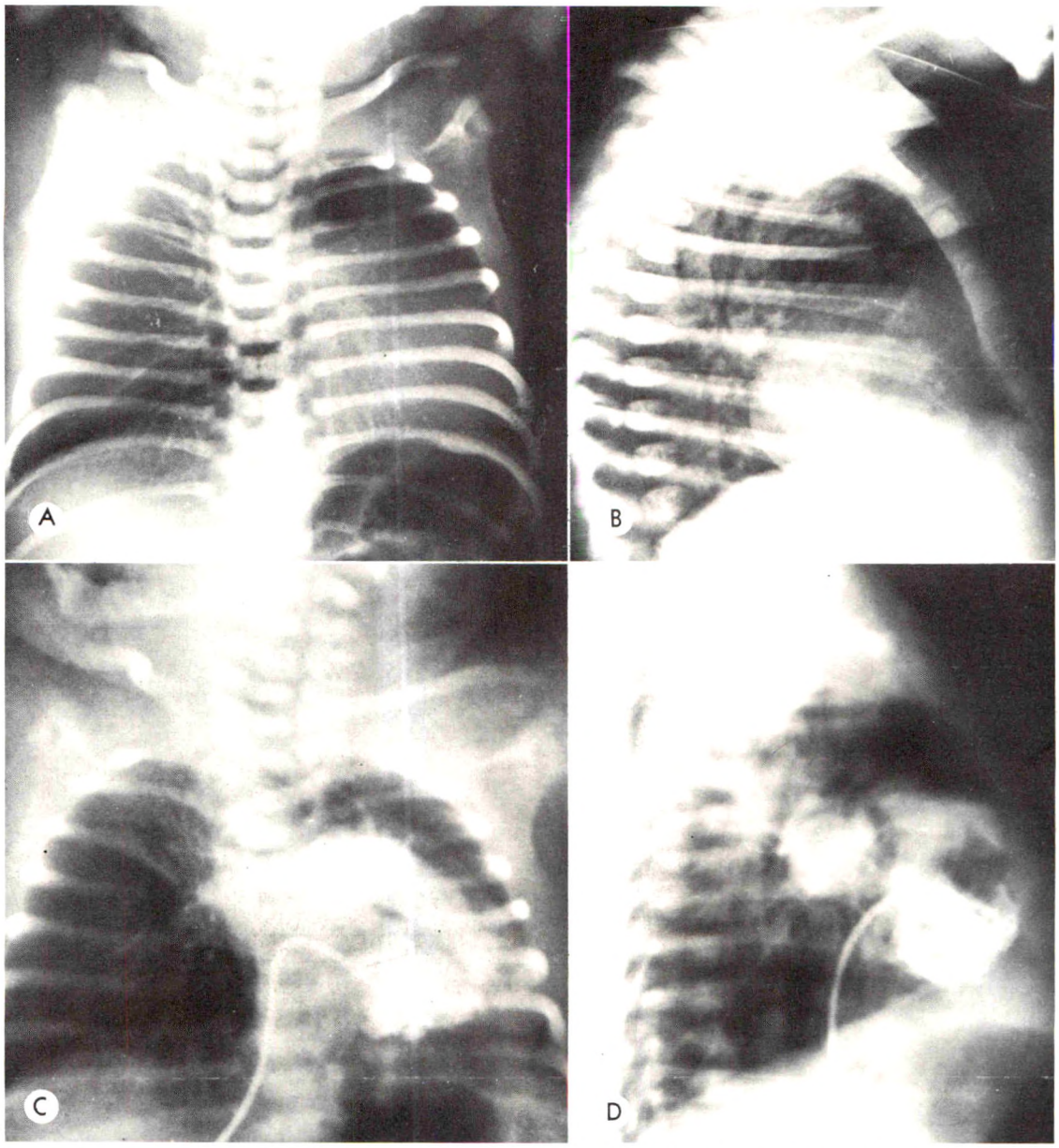


FIG. 1. Case 1. (*A*) Frontal and (*B*) lateral roentgenograms of the chest demonstrate shift of the heart and mediastinum to the left with atelectasis of the right upper lobe and emphysematous changes of the right middle and lower lobes. There is an increased retrosternal lucency on the lateral roentgenogram. (*C*) Frontal and (*D*) lateral right ventricular angiocardiograms show the typical features of tetralogy of Fallot. However, the pulmonary arteries are markedly dilated and this is suggestive of the diagnosis of absence of the pulmonic valve leaflets.

REPORT OF CASES

CASE 1. B/GA. (9253). A 2,310 gram female infant was born to an 18 year old mother following cesarean section due to cephalopelvic disproportion. The initial Apgar score was 7. Within the first hour of life, severe respiratory distress developed with tachypnea, and intercostal and substernal retractions. Chest roentgenograms (Fig. 1, *A* and *B*) showed the mediastinum shifted to the left with atelectasis of the right upper lobe; the right middle and lower lobes were emphysematous. Mild cardiomegaly

with conspicuously diminished vascularity was present. No mediastinal masses were evident but there was an abnormally lucent retrosternal space.

At 2 hours of age, a Grade 3/4 to-and-fro murmur was heard over the entire precordium, maximally audible at the lower left sternal border. Mild perioral cyanosis appeared with crying and the liver had enlarged. The electrocardiogram revealed right axis deviation and right atrial and ventricular hypertrophy and the neonate was digitalized.

Angiocardiograms (Fig. 1, *C* and *D*) demonstrated a ventricular septal defect, infundibular pulmonary stenosis and aneurysmal dilatation of the pulmonary artery and its main branches. The right intrapulmonary arterial branches were very attenuated. The left were not well visualized behind the heart but did appear smaller than normal. There was a right-sided aortic arch.

Serum calcium level on the second day of life was 6.9 mg. per cent and on the third day of life 6.3 mg. per cent. Respiratory distress increased with episodes of bradycardia and the development of generalized clonic convulsions characterized by rolling of the eyes upward, stiffening of the trunk and extremities, and clenching of the fingers.

The convulsions were temporarily controlled but during the evening of the third day of life, bilateral twitching of the upper extremities occurred. Repeated convulsions ensued with attacks of bradycardia and the baby developed consumptive coagulopathy and expired on the fourth day of life.

Postmortem examination revealed cardiac anomalies consistent with a diagnosis of tetralogy of Fallot. The pulmonary valve leaflets were absent, represented only by a cord-like ridge. There was aneurysmal dilatation of the main pulmonary artery and the proximal portion of both the right and left pulmonary arteries. No parathyroid or thymic tissue was identified after block dissection was performed with the neck organs subdivided into 4–5 blocks and then serially sectioned at 5 micra with every 10th section mounted.

CASE II. B/BF. (9431). A 2,840 gram Black male was born to a 19 year old mother with gestational diabetes. The initial Apgar score was 6 and cyanosis was noted at birth. The newborn did well until the second day of age when cyanosis was again noted and repeat physical examination revealed a Grade 3/4 pansystolic murmur maximally audible over the lower left sternal border radiating to the axilla.

Electrocardiography showed right axis deviation with biventricular hypertrophy. A roentgenogram of the chest (Fig. 2*A*) revealed cardiomegaly with a shift of the mediastinum and heart to the left and atelectasis of the right upper lobe; emphysematous changes in the right middle and lower lobes were present. The pulmonary blood flow was diminished bilaterally and an increased lucency in the retrosternal space observed.

Angiocardiography (Fig. 2, *B* and *C*) demonstrated a ventricular septal defect, pulmonary stenosis, overriding left-sided aortic arch and dilated main and right pulmonary arteries. There was a patent ductus arteriosus.

The infant appeared to do well clinically and was discharged to cardiology for follow-up. Initial calcium serum levels were not recorded.

He was re-admitted at 2 months of age for "bronchiolitis" with symptoms of cough, wheezing and irritability. However, he was discharged after 3 days and repeat electrocardiogram and chest roentgenograms during this admission were reported as unchanged.

At 3 months of age, the patient was again re-admitted with cough, fever and severe wheezing and was noted to be extremely irritable. Cyanosis had increased and his electrocardiogram showed changes of severe right ventricular hypertrophy. Serum calcium determined at admission was 7.3 mg. per cent. Within a few days, the patient was transferred to the intensive care unit with evidence of tremors and positive clonus. His condition continued to deteriorate with severe respiratory problems and tremors in addition to jack-knife like contractions. During his stay in the intensive care unit, he was given calcium on multiple occasions. Records were not available as to the level of his serum calcium when he was not on therapy.

Despite many attempts to correct his respiratory problems, he worsened, eventually developing consumptive coagulopathy and expired.

Postmortem examination revealed tetralogy of Fallot with overriding aorta, ventricular septal defect, pulmonic stenosis with absence of the pulmonary cusps and right ventricular hypertrophy as well as pulmonary infundibular stenosis. A single coronary artery was described.

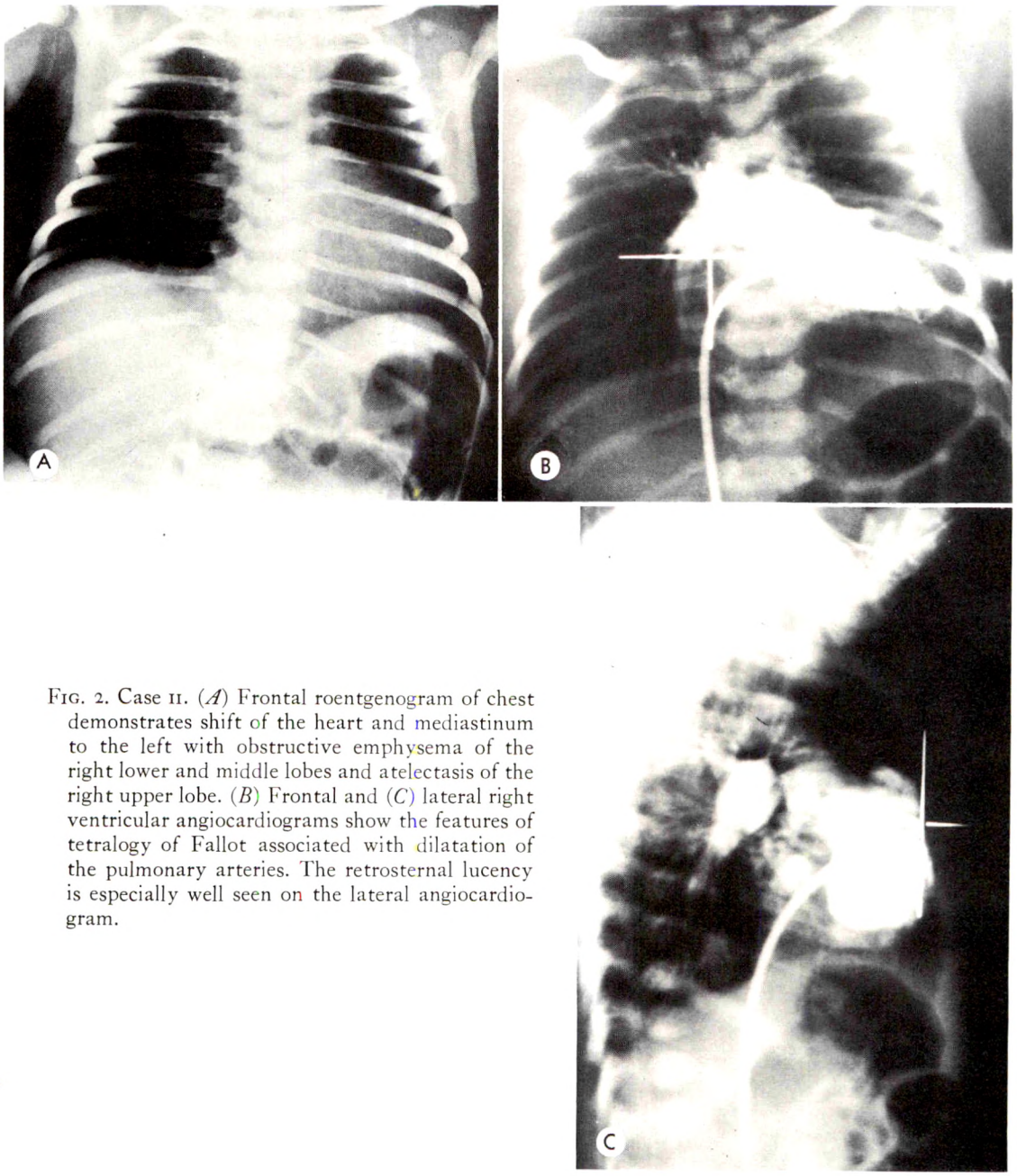


FIG. 2. Case 11. (*A*) Frontal roentgenogram of chest demonstrates shift of the heart and mediastinum to the left with obstructive emphysema of the right lower and middle lobes and atelectasis of the right upper lobe. (*B*) Frontal and (*C*) lateral right ventricular angiocardiograms show the features of tetralogy of Fallot associated with dilatation of the pulmonary arteries. The retrosternal lucency is especially well seen on the lateral angiocardiogram.

Block dissection was done and the neck organs were subdivided into 4–5 blocks; these were serially dissected at 5 micra and every 10th section mounted. No parathyroid or thymic tissue was identified. Cultures done post mortem revealed disseminated moniliasis.

DISCUSSION

The majority of patients with absence of the pulmonic valve have tetralogy of Fallot.[10] Almost all have ventricular septal defect.[9,10] Characteristic clinical signs associated with absent pulmonic valve include cyanosis, respiratory distress, a to-and-fro murmur along the left sternal border, diminished second pulmonic sound and electrocardiographic evidence of right ventricular hypertrophy. The main and right and

left pulmonary arteries become massively dilated in this condition and frequently compress adjacent bronchi to a significant degree. Previous authors have stressed the importance of this marked enlargement of the pulmonary arteries in making the roentgen diagnosis of absence of the pulmonic valve.[3] Less stress has been placed upon the lung changes due to bronchial compression—chiefly obstructive emphysema and atelectasis.[7,8]

The roentgen findings associated with isolated thymic aplasia have been discussed by Kirkpatrick and DiGeorge.[5] These patients show a narrow mediastinal shadow on the frontal chest roentgenogram and a retrosternal lucency in the lateral projection. We might therefore expect that in patients with DiGeorge's syndrome and absent pulmonic valve, the pulmonary artery enlargement would be even more striking roentgenographically because of the absence of the overlying thymic tissue. Somewhat surprisingly, our 2 patients showed neither prominence of the pulmonary arteries nor a narrow mediastinal silhouette on the frontal roentgenograms. The lateral roentgenograms did show retrosternal lucency. Both patients had atelectasis with emphysematous changes secondary to bronchial compression. Apparently the resulting shifts in cardiac position obscured both the pulmonary artery and the narrowed mediastinal silhouette.

Most descriptions of the angiocardiographic features of absent pulmonic valve have been based upon venous or right ventricular angiography, rather than selective pulmonary angiography. Right ventricular injections of contrast material in these cases will generally demonstrate the ventricular septal defect or tetralogy of Fallot, as well as marked enlargement of the main and right and left pulmonary arteries. If selective pulmonary arteriography is performed, regurgitation will be clearly evident. In most instances of isolated tetralogy of Fallot, the pulmonary arteries are small. If, however, the tetralogy is associated with absence of the pulmonic valve, the pulmonary arteries are much enlarged. This will allow the diagnosis of absence of the pulmonic valve to be suspected in such patients even if only venous or right ventricular angiography is performed.

Although DiGeorge's syndrome in its fullest extent encompasses complete failure of development of both the third and fourth pharyngeal pouches, it is apparent that a wide spectrum of involvement of these pouches exists.[4] Thus we may find maldevelopment only of the third pouch (agenesis or hypoplasia of the thymus) or incomplete development of both the third and fourth pouches (hypoplasia and ectopia of both thymus and parathyroids). Among the 31 patients with DiGeorge's syndrome and congenital cardiovascular disease described by Cameron,[1] Freedom *et al.*[4] and ourselves, there are now 4 who have had absence of the pulmonic valve. Since the latter is ordinarily a very uncommon form of congenital heart disease, this obviously represents a significant association. As in most instances of absence of the pulmonic valve, these patients have also had either ventricular septal defect or tetralogy of Fallot.

Our experience suggests that when the diagnosis of DiGeorge's syndrome with cyanotic congenital heart disease is entertained, the specific diagnosis of absence of the pulmonic valve can be made if certain clinical and roentgen features are present. The most important clinical features are a to-and-fro murmur along the left sternal border and respiratory distress. The most important single roentgen feature is localized obstructive emphysema or atelectasis, resulting from bronchial compression by the enlarged pulmonary arteries. This may lead to shifting of the heart and mediastinum which can obscure the expected signs of a narrow mediastinal silhouette and enlarged central pulmonary arteries. Angiocardiography can confirm the marked enlargement of these vessels and pulmonic regurgitation, in addition to demonstrating the presence of tetralogy of Fallot or ventricular septal defect.

SUMMARY

Two cases of DiGeorge's syndrome associated with tetralogy of Fallot and absence of the pulmonic valve are reported. Both infants had complete absence of the thymus and parathyroids. This brings to a total of 4 the number of reported cases of absence of the pulmonic valve in this rare syndrome.

Some of the expected roentgen signs, such as a narrow mediastinal silhouette and marked enlargement of the central pulmonary arteries, may be masked by cardiomediastinal shift due to bronchial compression.

The angiographic findings are discussed.

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We wish to thank Dr. John A. Kirkpatrick for his advice.

REFERENCES

1. CAMERON, A. H. Malformations of thymus and cardiovascular system. *Arch. Dis. Childhood*, 1965, *40* 334. (Abst.).
2. DIGEORGE, A. M. Discussions on new concept of cellular basis of immunity. *J. Pediat.*, 1965, *67*, 907–908.
3. DURNIN, R. E., WILLNER, R., VIRMANI, S., LAWRENCE, T-Y., and FYLER, D. C. Pulmonary regurgitation with ventricular septal defect and pulmonic stenosis—tetralogy of Fallot variant. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 42–51.
4. FREEDOM, R. M., ROSEN, J. S., and NADAS, A. S. Congenital cardiovascular disease and anomalies of third and fourth pharyngeal pouch. *Circulation*, 1972, *46*, 165–172.
5. KIRKPATRICK, J. A., JR., and DIGEORGE, A. M. Congenital absence of thymus. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *103*, 32–37.
6. KRETSCHMER, R., SAY, B., BROWN, D., and ROSEN, J. S. Congenital aplasia of thymus gland (DiGeorge's syndrome). *New England J. Med.*, 1968, *279*, 1295–1301.
7. KURLANDER, G. J. Bronchial obstruction from aneurysm of pulmonary artery associated with absence of pulmonary valve. *Lancet*, 1965, *85*, 129–132.
8. OSMAN, M. Z., MENG, C. C. L., and GIRDANY, B. R. Congenital absence of pulmonary valve: report of eight cases with review of literature. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 58–69.
9. PERNOT, C., HOEFFEL, J. C., HENRY, M., WORMS, A. M., STEHLIN, H., and LOUIS, J. P. Radiological patterns of congenital absence of pulmonary valve in infants. *Radiology*, 1972, *102*, 619–622.
10. RUTTENBERG, H. D., CAREY, L. S., ADAMS, P., JR., and EDWARDS, J. E. Absence of pulmonary valve in tetralogy of Fallot. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED. 1964, *91*, 500–510.

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PROTECTION OF CERTAIN STRUCTURES FROM HIGH DOSES OF IRRADIATION*

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THE delivery of curvative doses of radiation therapy sometimes leads to pronounced local tissue damage. The normal tissue effects are prohibitive when vital structures are affected and a lethal outcome is probable. Severe disabling functional impairments, although not lethal, similarly militate against high doses of irradiation. Compromises with curative doses often are made in patients where the radiation effects are neither life threatening nor incapacitating, but are associated with pronounced morbidity. An example of the latter is severe growth impairment in children. Indeed, all the foregoing considerations become accentuated in children because radiation not only impairs normal growth and development,[11] but can lead to late effects[12,17] that may be long-delayed.

These problems associated with the delivery of curative doses of radiation therapy have been recognized for decades. Many maneuvers have been used to avoid radiation damage. These techniques include the use of multiple portals of entry, the interposition of external and internal heavy metal blocks,[4] the employment of stents, and the infusion of epinephrine into the arterial tree of irradiated structures.[16] More recently, an old technique,[7] the repositioning of organs, has gained wide acceptance in the case of ovarian relocation in patients with Hodgkin's disease.[10,14] Other relatively simple methods are available to protect other organs. Some of these techniques have been described in the past, but have not gained the popularity they perhaps deserve.

This paper will describe methods used by us to protect the lung, the testes, and the bowel.

MATERIAL AND METHODS

LUNGS

It sometimes becomes necessary to treat portions of the chest wall because of diffuse involvement with a malignant neoplastic process. Elaborate multiple tangential fields can be used.[9] Uniformity of dose is difficult to achieve. The problem is compounded when the upper thorax is to be treated because the structures of the shoulder girdle come into the treatment fields. Other methods employ several adjoining electron beam portals.[3] Electron beams are not universally available, and the dosimetry and techniques used are not simple.

Many of these problems can be circumvented by collapsing the lung.[1] A major portion of the thorax can then be irradiated with simple, parallel-opposed field methods, blocking those regions that need not be treated.

CASE 1. M.D., 6½ year old female. A golf-ball sized mass was noted in the right infraclavicular region in October of 1971. Local excision was performed at another institution, and a diagnosis of rhabdomyosarcoma was made. She was then referred to this Center at which time there was a residual mass measuring 2×3 cm. There also was induration in the local region as well as a 15 cm. scar that extended across the right anterior chest wall ending near the right nipple. There was no evidence of remote disease by physical examination, or by extensive laboratory and roentgenographic investigations. It was recognized that most of the anterior chest

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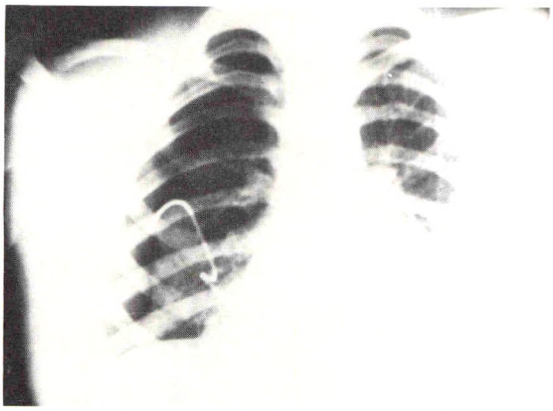


FIG. 1. Case 1. Bone changes are apparent in the right acromion. A radiopaque catheter that extends into the right pleural cavity is visible at the lower edge of the roentgenogram. It was easy to deflate the lung during the 5 days of the week that radiation therapy was given and to re-inflate the lung over the weekend when the child was permitted to return home.

wall as well as the deep structures surrounding the shoulder girdle harbored residual tumor.

Treatment using multiple chemotherapeutic agents and radiation therapy was instituted. The treatment plan called for irradiation of the anterior chest wall and of the entire right shoulder girdle including the proximal humerus. The underlying right lung was known to be clear of tumor. An artificial pneumothorax was induced, thus collapsing the right lung away from the fields of treatment. Through-and-through irradiation using cobalt 60 teletherapy technique was used to treat the shoulder girdle. The treatment plan included electron beam irradiation of the right anterior chest wall. A total of 7,000 rads over a 9 week elapsed time was delivered to the right shoulder, while the chest wall received 5,100 rads in 52 elapsed days.

The pneumothorax was induced using a small catheter which was left in place (Fig. 1). The lung was collapsed during the 5 days of the week that irradiation was given; it was re-expanded over the weekend when the child was allowed to return home. The pneumothorax was re-induced the following Monday, checked fluoroscopically, and radiation therapy resumed.

The patient is free of local or remote disease 21 months after initiation of therapy. There is no evidence of pulmonary functional impairment, and the chest roentgenograms appear normal (Fig. 2, *A* and *B*).

CASE II. B.L., 6 month old male. A left supraclavicular and an axillary mass, each measuring approximately 4×4 cm., were detected in August 1972. Incomplete excision at another institution led to a diagnosis of angiosarcoma. When seen at this Center, there was a left axillary mass measuring 10×10×4 cm. with a smaller supraclavicular tumor. Roentgenographic examination revealed bilateral pulmonary metastases. Physical examination coupled with extensive roentgenographic and laboratory studies revealed no other evidence of disease. Chemotherapy using multiple agents was instituted. Radiation therapy was given to the bulky left axillary mass and supraclavicular lymph nodes using a 20 mev. electron beam. Two hundred rads daily was given to a total tumor dose of 1,000 rads in 5 days. There was no response, so the dose fractionation was increased to 400 rads per increment to an additional 1,200 rads in 3 treatments. Pronounced, but not complete shrinkage of the tumor resulted. At the same time there was a moderate skin erythema. He was given a rest period followed by a left modified neck dissection and subtotal removal of the left supraclavicular and axillary masses. Postoperatively, he did well; 1,200 rads over 16 elapsed days using cobalt 60 teletherapy was delivered to both lungs.

A pneumothorax was then induced on the left side and an additional 1,950 rads in 16 days was given using Co^{60} techniques. This portal included through-and-through irradiation of the left chest wall, axilla, and lower neck. An additional booster dose of 750 rads at 2 cm. depth in 3 elapsed days was given to the neck using Co^{60} techniques. The lung was re-expanded at the completion of therapy. The patient remained free of disease and without visible roentgen changes in the left lung, or incapacity for a period of 11 months. He relapsed thereafter and died of disseminated tumor, always without roentgen or physical signs of radiation associated lung damage.

TESTICLE

It becomes necessary in some males with only one testicle to irradiate structures close to the gonad. An example is the patient who has had transcrotal biopsy of a testicular tumor prior to radical orchiec-

tomy. It is our practice to perform a hemiscrotectomy in such patients. Even so, there may be considerable doubt that the scrotal septum or other portions of the scrotal sac are free of tumor. Irradiation of the scrotum is to be recommended in such circumstances. It is extremely difficult, if not impossible, to shield the remaining unaffected testicle. It receives a relatively high dose no matter what method of blocking is employed.

We have used another method. The remaining testicle is repositioned outside the radiation field. The technique used by one of us (P.R.E.) is as follows: through a small inguinal incision the testis is delivered and repositioned in a subcutaneous pocket in the thigh. An anchoring suture fixes the testis to fascia to prevent torsion. The gonad is much more readily shielded, and because of distance from the field edge receives much less scattered radiation. The organ is returned to its normal location at the completion of radiation therapy.

CASE III. K.S., 4 year old male. A left scrotal swelling was noted in August 1970. A left orchiectomy was performed through the scrotum at another institution revealing a rhabdomyosarcoma. He then was referred to this Center where physical, laboratory, and roentgenographic examinations revealed no evidence of distant metastases except for positive findings on lymphangiography. The patient underwent a left hemiscrotectomy. This was coupled with dissection of the superficial and deep groin lymph nodes, together with those of the left iliac and para-aortic chains. The contents of the ipsilateral inguinal canal were also removed.

The right testicle was then repositioned in the right thigh, where it would receive only scattered radiation, estimated to be less than 10 per cent of the tumor dose. Combined chemo- and radiotherapy was initiated. Multiple agent chemotherapy was prescribed, part of which was given at this Center. Radiation therapy was planned for the surgical scar, inguinal canal, ipsilateral pelvis, and para-aortic regions. A total dose of 4,000 rads over a 5 week period was given to each of these areas according to plan and by agreement with a radiation therapist in the city where the patient lives. Dose specifications were met by the cooperating radiation therapist. The testis was then returned to the normal location in the scrotum. Subsequent examination of the testicle reveals normal size and consistency; it remains to be seen whether normal sexual

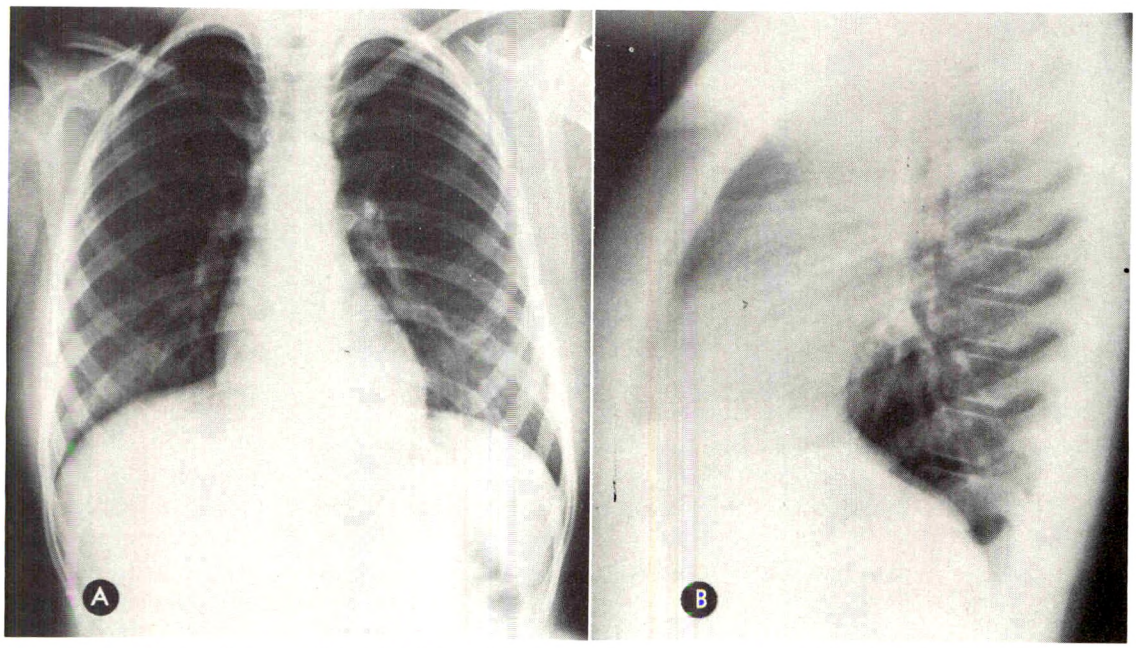


FIG. 2. Case 1. (*A* and *B*) Normal chest roentgenograms 21 months after therapy.

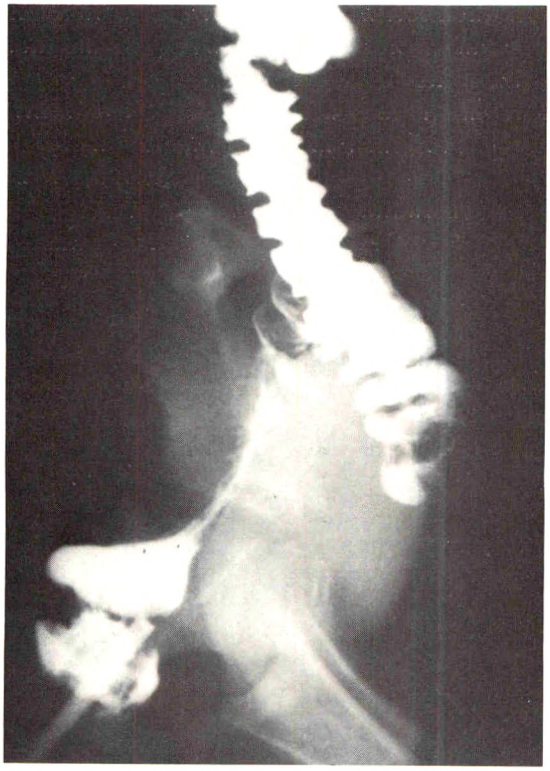


FIG. 3. Case IV. Anterior displacement of the rectum and bladder by a large soft tissue mass associated with a destructive process in the sacrum.

maturation will occur at the age of puberty, and whether the patient eventually proves to be potent. The patient is free of disease 32 months after completion of therapy.

BOWEL

Compromises with curative doses of radiation therapy must be made in some patients who require treatment of the abdomen or pelvis. This is because of prohibitive complication rates when high doses are given. The probability of inducing severe radiation enteritis is increased in patients who have had prior surgery, with the high probability of loops of bowel becoming adherent to the abdominal parieties, and thus subject to the full effects of treatment.

The gut can be displaced from the radiation field by introducing a temporary or permanent pack made of suitably inert material. We have employed "Ivalon" because prior experience has shown the substance to be well tolerated by patients for periods of 2 to 10 years after implantation of the plastic.[13]

CASE IV. F.D., 7 year old female. A sacral mass was detected in December 1971. Biopsy at another institution revealed a malignant tumor. The patient was then referred to this Center where a large mass was found in the presacral region displacing the rectum and bladder anteriorly (Fig. 3). No invasion of either structure was evident on barium enema examination. There were, however, destructive changes visible in the body of S-3 on the left. The histologic slides were reviewed and a diagnosis of malignant small cell sarcoma, favoring Ewing's sarcoma was made. No evidence of remote disease was visible on physical examination or by laboratory or roentgen investigations.

Multiple agent chemotherapy was instituted. Radiation therapy was given to the bulky tumor using opposing anterior and posterior fields; 3,500 rads in a period of 3 weeks was delivered. There was marked shrinkage of the tumor as a result of the treatments given. The patient was then explored transabdominally, and lack of invasion of the posterior rectal wall was confirmed. An "Ivalon" sponge was inserted behind the rectum, displacing that structure well away from the bony sacrum and lower lumbar spine (Fig. 4). It was therefore possible to arrange 2 lateral treatment portals, designed to include the lower lumbar vertebral bodies and the sacrum, but easily skirting the anteriorly-placed rectum. An additional 3,000 rads in 3 weeks was delivered through 2 lateral pelvic (opposing) fields. The treatment was well tolerated, there was no proctitis at the time nor subsequently. The patient is free of disease and of radiation therapy related bowel symptoms 18 months after the completion of therapy.

DISCUSSION

A decision to administer radiation therapy always entails a weighing of the relative risks involved in delivering a certain dose. The therapist must take into account possible failure to achieve local control when choosing a relatively low dose to protect normal structures. Alternatively, he must keep in mind the immediate and late

consequences to normal structures irradiated to the high doses necessary for achieving tumor ablation. These effects can be severely disabling, if not lethal, depending on the structures affected.

Much time, thought, money, and effort has been expended in finding ways around this therapeutic dilemma. The advent of megavoltage and telecobalt therapy units was a major advance along these lines because of their skin and bone-sparing capacities.[15] New particulate beams are being studied because better effective depth doses can be obtained through their advantageous physical and radiobiologic properties.[2,6]

There remain simple, rather mechanical means that can be used in certain circumstances to protect sensitive structures, while delivering the high doses to target volumes required for cure. We have described methods that can be used to spare the lung, the testicle, and the rectum. The technique used to displace the rectum can be modified readily to protect other segments of the gastrointestinal tract. The apparent success of the maneuvers amply justifies their use, particularly when the simplicity of the surgical measures employed is re-called.

Other possible maneuvers come to mind; for example, the relocation of the patient's own kidney or both kidneys into the pelvis, using familiar and successful surgical techniques developed for renal transplantation.[8] In these cases, there would be no problem of rejection, of course.

The liver drops away from the diaphragm when pneumoperitoneum is induced. Suitably shaped and directed portals with the patient in the upright position could be used to spare the liver, while irradiating the lung and its parieties. This maneuver could also be used in treating anterior abdominal wall tumors. The pneumoperitoneum will distend the abdominal wall, separating that structure from the underlying bowel. Lateral opposing fields across the table with the patient supine could then be used, thus sparing the structures beneath.

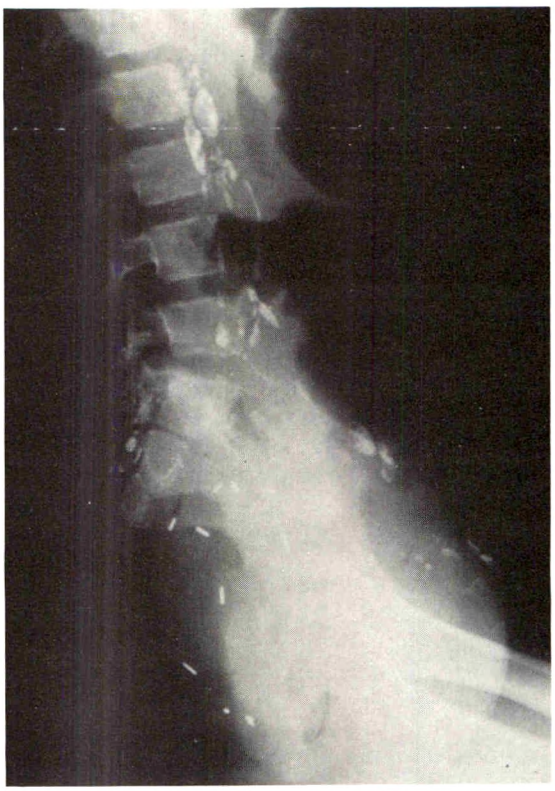


FIG. 4. Case IV. Anterior position of the rectum following "Ivalon" sponge packing of the presacral region. The position of the pack is outlined by metallic clips. The rectum lies anterior to the sponge.

Enhanced radiation reactions, both in neoplastic and normal tissues, are known to occur when irradiation is combined with certain chemotherapeutic agents, notably actinomycin D.[5] An increased incidence of normal tissue damage can be anticipated as more combined chemo- and radiotherapy regimens are adopted in the future. Radiation therapy doses that were well tolerated in the past might well become marginal or worse under these circumstances, and the late effects of immediate damage eventually prove to be lethal. Measures such as those described can be used when feasible to forestall such sequelae.

SUMMARY

The radiation therapist is often confronted with a therapeutic dilemma. He

must decide whether to compromise with curative doses of radiation therapy, or run the alternate risk of producing severely incapacitating, if not lethal, damage to normal tissues necessarily included in the beam.

Many methods are used by radiation therapists to minimize normal tissue damage.

Relatively simple surgical techniques described in this paper were used to protect the lung, the testicle, and the rectum.

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Case III is a patient of Drs. Peter Fagan and Prestiss Dettman, Cleveland, Ohio, whose cooperation and assistance are gratefully acknowledged.

REFERENCES

1. ALARCON, D., and ZARALA, E. Intrapleural pneumothorax as means to protect the lung after roentgen therapy. *Cancer*, 1969, *23*, 513–514.
2. BARENDSEN, G. W., BROERSE, J. J., and VAN PUTTEN, L. M. Fundamental and practical aspects of application of fast neutrons in clinical radiotherapy. Meetings held at the Radiobiological Institute TNO, Rijswijk Z. H., The Netherlands, June 22–24, 1970. Introduction to the Proceedings. *European J. Cancer*, 1971, *7*, 2–3.
3. CHU, F., SCHEER, A., and GASPAR-LANDERO, J. Electron beam therapy in management of carcinoma of breast. *Radiology*, 1960, *75*, 559–567.
4. D'ANGIO, G. J., and TEFFT, M. Radiation therapy in management of children with gynecologic cancer. *Ann. New York Acad. Sc.*, 1967, *3*, 675–693.
5. D'ANGIO, G. Use of combined actinomycin D and radiotherapy in children with Wilms' tumor. *Front. Rad. Ther. & Onc.*, 1969, *4*, 174–180.
6. GRIEM, M. Symposium on role of heavy particle radiation in treatment of cancer. *Radiolgy*, 1973, *108*, 119–158.
7. KREBS, C., BLIKENKRONE, N., and MOSEKILDE, V. Preservation of ovarian function in early cervical cancer after surgical lifting of ovaries and radiation therapy. *Acta radiol.* (Therapy), 1963, *1*, 176–182.
8. KUSS, R., TEIUNTURIER, J., and MILLIEZ, P. Quelques essais de greffs du rein chez l'homme. *Mém. Acad. chir.*, 1951, *77*, 755–764.
9. MEURK, M. L., and CHU, F. C. H. Dose distribution with four radiation techniques for carcinoma of breast. *Radiology*, 1959, *73*, 607–617.
10. NAHAS, W. A., NISCE, L. Z., D'ANGIO, G. J., and LEWIS, J. L., JR. Lateral ovarian transposition. *Obst. & Gynec.*, 1971, *38*, 785–788.
11. NEUHAUSER, E., WITTENBORG, M., BERMAN, C., and COHEN, J. Irradiation effects of roentgen therapy on growing spine. *Radiology*, 1952, *59*, 637–650.
12. O'MALLEY, B., D'ANGIO, G. J., and VAWTER, C. F. Late effects of roentgen therapy given in infancy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *89*, 1067–1074.
13. PENFOLD, J. C. B., and HAWLEY, P. R. Experiences of Ivalon-sponge implant for complete rectal prolapse at St. Marks Hospital, 1960–1970. *Brit. J. Surg.*, 1972, *59*, 846–848.
14. RAY, G., TRUEBLOOD, N., ENRIGHT, L., KAPLAN, H., and NELSEN, T. Oophoropexy: means of preserving ovarian function following pelvic megavoltage radiotherapy for Hodgkin's disease. *Radiology*, 1970, *96*, 175–180.
15. SMITH, I. H., FETTERLY, J. C. M., LOTT, J. S., MACDONALD, J. C. F., MYERS, L. M., PFALZNER, P. M., and THOMSON, D. H. Cobalt 60-teletherapy. In: A Handbook for the Radiation Therapist and Physicist. Hoeber Medical Division, Harper & Row, New York, 1964.
16. STECKEL, R. J., TOBIN, P., ROSS, G., STEIN, J. J., and STEVENS, G. H. Radiation protection of vital organs using selective arterial catheter: experimental and clinical aspects. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 841–847.
17. TEFFT, M., VAWTER, G. F., and MITUS, A. Second primary neoplasms in children. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *103*, 800–822.

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CONTROL OF SCATTERED RADIATION BY AIR GAP TECHNIQUES: APPLICATIONS TO CHEST RADIOGRAPHY*

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DIAGNOSTIC radiographic image contrast is enhanced by preventing Compton scattered photons that arise within the patient from reaching the film. A grid absorbs much of the scattered radiation, but it also attenuates the primary photon beam significantly and, therefore, requires an increase in patient exposure to attain a particular film density. Scattered radiation can also be reduced by introducing an air gap between the patient and the film. Many scattered photons then miss the cassette because their direction was significantly altered in a Compton interaction. The undeflected primary photons will not be affected by the air gap, except for the divergence of the beam. Thus with an air gap a larger target-film distance is required to match the magnification and focal spot unsharpness factors of conventional techniques. Under these circumstances, however, a reduction of relative film exposure due to scattered radiation is achieved. Furthermore, patient exposure will be lower if an air gap is used instead of a grid.

This technique has only been occasionally mentioned in the literature[1,3] and has also been referred to in passing in some radiologic physics texts.[5,12,13]

In this paper we present the results of a systematic study of some of the parameters that must be considered in establishing air gap techniques for chest radiography.

EXPERIMENTAL METHOD

Patient attenuation and scatter was simulated by a pressdwood phantom made up with $24 \times 24 \times \frac{1}{4}$ inch sheets so that the thickness could be readily changed in a horizontal x-ray beam. The water-equivalent thickness of the particular batch of pressdwood was first determined by using the automatic brightness control system of image intensified fluoroscope as an exposure meter. A measured thickness of water in a plastic container was placed in a vertical x-ray beam. The tube operating potential established by the servo system to obtain a preset brightness level was noted. Then the thickness of pressdwood necessary to produce this same servo system response was determined. A series of similar measurements established that 1 cm. of the pressdwood was equivalent to 1.04 cm. of water.

For measurements of radiation transmitted through and scattered by the phantom, the radiation was detected by a system consisting of a Par Speed intensifying screen attached to the inner surface of a thin, opaque, bakelite light shield that formed one side of a light-tight box. Light from the screen was focused by a lens onto the photocathode of a photomultiplier tube. The potential produced by the passage of the photomultiplier current through a resistor was read with an A.C. voltmeter. The meter response, then, should be proportional to the light emitted by the intensifying screen. Tests showed that the meter response was linearly related to the screen exposure rate, measured in roentgens per minute with an EIL exposure meter, when the exposure rate was changed over a wide range by varying the distance between the x-ray tube and the intensifying screen.

Measurements were made of luminance due to primary and to the sum of primary plus scattered radiation. To obtain the

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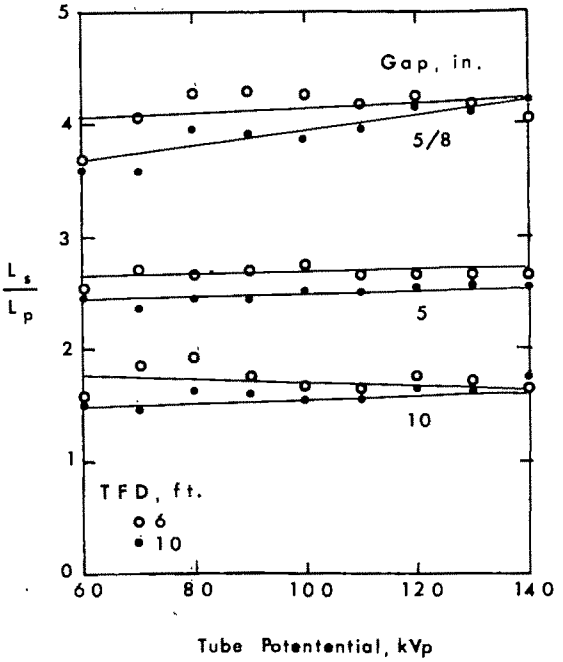


FIG. 1. Variation of the light emitted from an intensifying screen due to scattered radiation, L_s, to the light emitted due to primary radiation, L_p, as a function of tube potential for 3 different air gaps and 2 different target-film distances (TFD). Data taken with an absorber equivalent to 13.1 cm. of water. Field size 14×17 inches.

primary luminance data, a very narrow beam was used with the absorber as close as possible to the x-ray source. A second collimator was placed between the observer and the fluorescent screen. Measurements of the sum luminance were made with air gaps of ⅛, 1, 5, 10 and 15 inches. The 1 inch air gap represents the distance between the front intensifying screen and the back of the sensing device for automatic exposure timing in a typical upright cassette holder used in chest radiography. The scattered component of the luminance was then determined by subtracting the primary light measurements from the measurements of the sum. To evaluate the effectiveness of a particular air gap, the ratio of the light due to scatter to the light due to primary radiation (L_s/L_p) was calculated. Under a given set of exposure conditions, the smallest ratio of L_s/L_p will yield the greatest visual contrast.

Data were obtained varying the tube potential from 60 to 140 kVp. Measurements were made using a 14×17 inch field at 6 and 10 feet target-film distances with various phantom thicknesses.

Typical results of an experimental run are shown in Figure 1, where the variation of L_s/L_p is plotted as a function of x-ray tube potential for a 14×17 inch field and an absorber equivalent to a 13.1 cm. water phantom. The dots are data points obtained with a TFD of 10 feet. The circles are for a TFD of 6 feet. Similar sets of measurements were obtained with the absorber thicknesses of 8.1, 17.0 and 21.7 cm. Thus a total of 24 sets of data are available for analysis.

The variation of L_s/L_p as a function of absorber thickness and air gap width at 100 kVp, 10 feet TFD and a 14×17 inch field is shown in Figure 2. The same data plotted as a function of air gap width are shown in Figure 3.

To investigate the effect of air gaps on patient exposure, the accuracy of an Iontomat automatic timer connected to a vertical wall mounted cassette holder was first

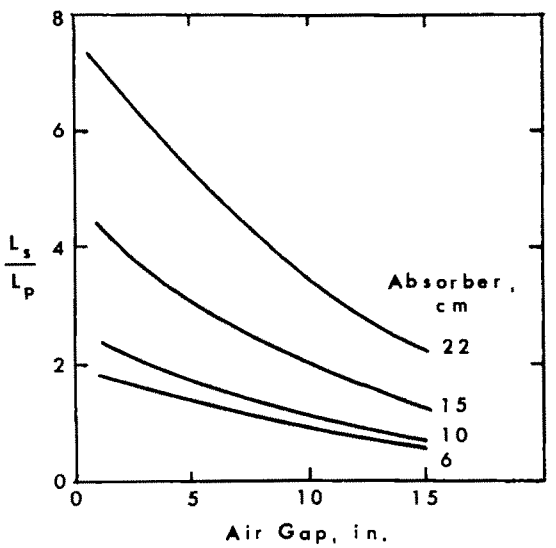


FIG. 2. Variation of L_s/L_p as a function of absorber thickness for various different air gaps. Data taken at 100 kVp with a 14×17 inch field. TFD was 10 feet (ft.).

TABLE I

PATIENT EXPOSURE

(14×17 inch field)

	kVp	Grid	Air Gap, (in.)	TFD, (ft.)	Exposure, (mR)
Posteroanterior phantom for 22 cm. patient, (ρ=0.5)	80	No	1	6	35
			5	8	31
				10	33
			10	8	41
				10	43
Lateral phantom for 30 cm. patient, (ρ=0.6)	125	12:1,O	1	6	295
	100 l/in	100 l/in.	5	8	55
				10	53
			10	8	79
				10	75

tested. This was done by measuring the exposure with an EIL meter near the cassette holder, while the exposure rate was varied over a wide range by inserting aluminum sheets in the beam at the level of the collimator to simulate patient attenuation.[6] Replicate exposures demonstrated a reproducibility of ±2 per cent. Varying the aluminum filtration from 35 to 46 mm. to simulate lateral chest thickness variation from 25.5 to 35.5 cm. at 125 kVp with a grid demonstrated that the film exposure was constant with a precision of ±3 per cent.

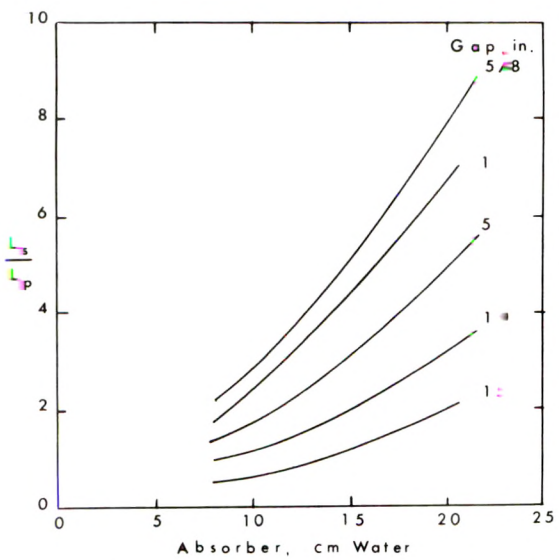


FIG. 3. Data of Figure 2 plotted to show the variation of L_s/L_p as a function of gap width for various absorber thicknesses.

Measurements were then made with the pressdwood phantom simulating a 22 cm. patient being examined in the posteroanterior projection at 80 kVp and simulating a 30 cm. diameter lateral projection at 125 kVp with a 12:1 100 line per inch grid. Automatic timing was used. The exposure in the beam was measured with the EIL exposure meter. From these readings the exposure at the level of the entrance surface of the simulated patient was calculated. The data are shown in Table 1.

Although the interest of this investigation was directed toward chest radiography, so that 14×17 inch fields were used throughout, a set of measurements were made of L_s/L_p as a function of field size with a 1.5 inch air gap. Data were taken at 100 kVp with an absorber equivalent to 21.5 cm. of water. The results of this experiment are shown in Figure 4.

DISCUSSION

VARIATION OF L_s/L_p WITH TUBE POTENTIAL

The plots of the ratio of L_s/L_p as a function of tube potential for the various air gap widths and absorber thicknesses, such as those shown in Figure 1, lead to the hypothesis that there was no systematic vari-

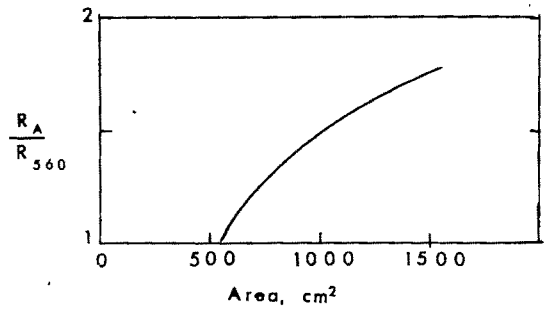


FIG. 4. Variation of the ratio $R=L_s/L_p$ as a function of field size normalized to the value of the ratio at 516 cm.² (8×10 inches).

ation of this ratio with tube potential. To test the hypothesis, it was assumed that the data could be fitted to a straight line. The slope and the standard deviation of the slope were calculated for each set of data. The results of the regression analysis are shown in Table II. The average value of the ratio L_s/L_p is $\bar{E}$. The standard deviation of $\bar{E}$ is σ_R. The slope of the least squares fit to the points is S and σ_S is the standard deviation of the slope. Of the 24 sets of data analyzed, 11 had positive slopes and 13 had negative slopes. In 16 experiments the slope plus or minus twice the standard deviation of the slope included 0. Of the remaining 8 experiments, 4 had positive slopes and 4 had negative slopes. It was concluded from these data that there is no systematic dependance of the ratio L_s/L_p on tube potential.

This result can be explained on the basis of the theory of Compton scattering. Figure 5 shows the results of the calculation of the variation of the Compton differential cross section as a function of the angle through which the photon is scattered from the direction of the incident photon. It is seen that when the incident photon energy varies from 10 keV to 140 keV the most probable angle of scatter changes only from about 53° to 45°. Furthermore, the probability of scatter in the forward direction does not change very much. The cross section changes only from 2.68×10^{-27} cm.² to 2.28×10^{-27} cm.² at the most probable scattering angle, a variation of only 15 per cent. Thus, over the diagnostic energy range, the flux of Compton scatter photons in the forward direction does not change very much. Accordingly, the exposure due to scatter photons should be proportional to the primary flux. It follows that the ratio L_s/L_p should be independent of the primary photon energy.

TABLE II

ANALYSIS OF EXPERIMENTAL DATA

($R=L_s/L_p$)

Absorber (cm.)	TFD (ft.)	Gap width (in.) 5/8 $\bar{R}\pm\sigma_R$	5/8 $S\pm\sigma_S$	5 $\bar{R}\pm\sigma_R$	5 $S\pm\sigma_S$	10 $\bar{R}\pm\sigma_R$	10 $S\pm\sigma_S$
8.1	6	2.52±0.13	0.029±0.013	1.67±0.08	−0.017±0.009	1.08±0.08	−0.013±0.011
	10	2.17±0.16	0.029±0.020	1.45±0.09	0.009±0.010	0.98±0.10	0.010±0.014
13.1	6	4.14±0.20	0.026±0.025	2.66±0.05	0.001±0.007	1.72±0.12	−0.016±0.015
	10	3.92±0.24	0.08 ±0.012	2.49±0.07	0.022±0.66	1.59±0.09	0.022±0.009
17.0	6	6.70±0.32	0.050±0.04	4.33±0.18	−0.035±0.020	2.69±0.14	−0.027±0.015
	10	6.39±0.13	0.007±0.014	4.11±0.21	−0.06 ±0.020	2.56±0.18	−0.04 ±0.01
21.7	6	9.08±0.27	−0.050±0.038	5.81±0.23	−0.054±0.030	3.80±0.27	−0.77 ±0.032
	10	8.69±0.14	−0.053±0.07	5.61±0.35	−0.10 ±0.060	3.40±0.36	−0.13 ±0.05

RELIABILITY OF DATA

The data of Figure 1 were obtained by reading the value of L_s+L_p at 10 kVp intervals from a smooth plot of the variation of L_s+L_p as a function of tube potential and subtracting the value of L_p at the same kVp intervals read from another smooth plot of the variation of L_p as a function of the tube potential. This difference was then divided by the values of L_p. Thus experimentally

$$\frac{L_s}{L_p} = \frac{(L_s + L_p) - L_p}{L_p} .$$

An analysis of the combination of errors following Wilson[16] shows that the standard deviation, σ, is related to the assumed fractional error, f, in each measurement by the following relationship (Table III):

$$\sigma = \sqrt{2} f \sqrt{k^2 - k + 1}$$

where $k=(L_s+L_p)/L_p$.

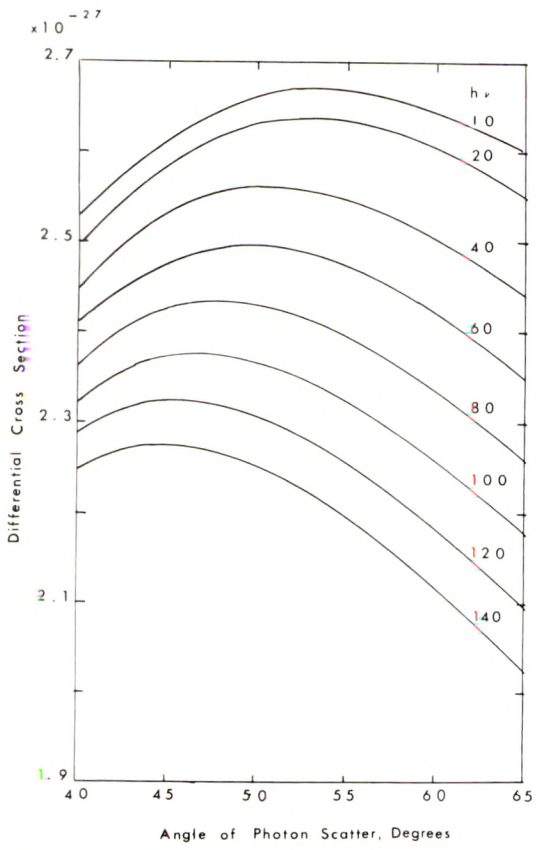


FIG. 5. Variation of the Compton differential cross section for photon scattering $(d_c\sigma)/d\phi$ as a function of the angle of scatter, ϕ, of the photon from the direction of the incident photon. The family of curves was calculated for various energies, $h\nu$, of the incident photon.

TABLE III

ANALYSIS OF EXPERIMENTAL ERRORS

(absorber thickness 13.1 cm.)

Air Gap (in.)	TFD (ft.)	$\bar{R}$	σ	
			Experiment	Expected
5/8	6	4.14	0.20	0.19
	10	3.92	0.24	0.18
5	6	2.66	0.05	0.14
	10	2.49	0.07	0.13
10	6	1.72	0.12	0.09
	10	1.59	0.09	0.09

The average values of the ratio $\bar{R}=L_s/L_p$ for the data shown in Figure 1 and the experimentally observed standard deviations are shown in Table 1 assuming that $\bar{R}$ is independent of tube potential. Also shown are the calculated standard deviations of R if it is assumed that there is a fractional error of 3 per cent in each of the measurements from which R was obtained. It is seen that there is good agreement between the expected and observed standard deviations. It seems reasonable, accordingly, to conclude that the variation in the data points from the least square lines seen in Figure 1 is random.

VARIATION OF L_s/L_p WITH TARGET-FILM DISTANCE

The ratio of the data points at 6 and 10 feet from Table 1 is 1.08 ±0.04. At the time these measurements were made the strong dependence of L_s/L_p with field size, Figure 4, was not appreciated. Accordingly it is believed that this slight difference is a systematic error in the adjustment of the light localizer diaphragm and there is no variation of the ratio L_s/L_p with target-film distance, within these limits.

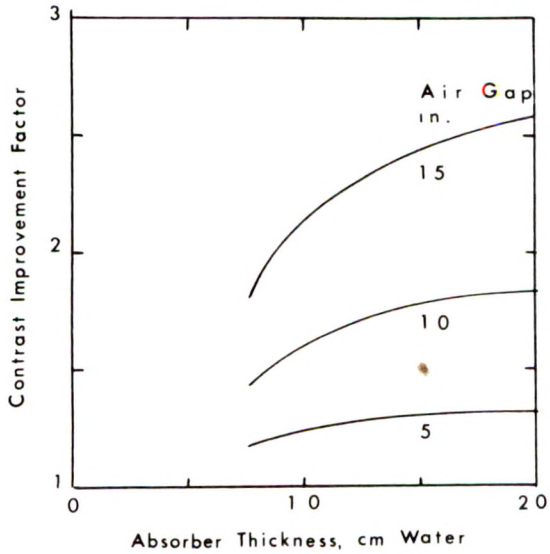


FIG. 6. Variation of the contrast improvement factor K, as a function of absorber thickness for various air gap widths.

VARIATION OF L_s/L_p WITH AIR GAP WIDTH AND ABSORBER THICKNESS

The variation of L_s/L_p with absorber thickness and air gap width is shown in Figure 2 with the thickness as the principal parameter and Figure 3 with gap width as the principal parameter. Recalling that the posteroanterior projection of the chest can be very nearly simulated by a solid absorber if the approximation is made that the chest density is equal to 0.5,[6] it is seen that for a 1 inch air gap, which is typical of a wall mounted cassette holder and for an average 22 cm. chest diameter simulated by 11 cm. of water, 3 out of 4 photons that produce light in the intensifying screen are Compton scattered photons and, accordingly, carry no information. This observation is in good agreement with the data of Wilsey,[15] although it is higher than the value quoted by Seeman.[12]

CONTRAST IMPROVEMENT FACTOR

A contrast improvement factor for grids, K, was defined by Bonenkamp and Hondius Boldingh[2] to establish an index of effectiveness of various grids:

$$K = \frac{1 + L_s/L_p}{1 + L_s'/L_p'},$$

where L_p is the screen luminance due to primary radiation and L_s is the luminance due to scattered radiation. The primed quantities are measured with a grid, the unprimed quantities without a grid. In a similar fashion, a contrast improvement factor for air gaps can be defined:

$$K = \frac{1 + (L_s/L_p)_{\text{normal}}}{1 + (L_s/L_p)_{\text{air gap}}}.$$

The contrast improvement factors for air gaps calculated from the data in Figure 2 are shown in Figure 6.

These data can also be used to compare the air gap method with conventional grids. This is done in Table IV where the grid data are taken from Hondius Boldingh.[8] Typical data are shown for light, medium, and heavy grids. It is seen that the 10 inch air gap is comparable to a medium weight grid and that for thin absorbers, a 5 inch air gap is comparable to a light grid.

THE EFFECT OF SCATTER RADIATION ON VISUAL CONTRAST

The concept of visual contrast has been proposed to investigate the effect of scattered radiation on the contrast perceived by observer.[7] Visual contrast was defined in terms of the luminance of adjacent areas on a view box. The contrast of these two quantities should be proportional to the

TABLE IV

CONTRAST IMPROVEMENT FACTOR

(100 kVp, 30×30 cm. field)

Absorber (cm. water)	Air Gap		Grid		
	5 in.	10 in.	No. 5*	No. 7*	No. 10*
10	1.39	1.81	1.50	1.75	1.85
20	1.37	2.21	2.10	2.35	2.85

Grid	Ratio	lines/cm.	mg./cm.²
5	7:1	29	390
7	9:1	23	460
11	15:1	27	830

* Data from W. Hondius Boldingh.[8]

contrast in the illumination of the observer's retina. The visual contrast was shown to be:

$$C_v = \frac{1 - \left[\frac{\alpha + R}{1 + R}\right]^{\gamma}}{1 + \left[\frac{\alpha + R}{1 + R}\right]^{\gamma}}, \qquad (1)$$

where $\alpha = E_{op}/E_{bp}$, the ratio of the exposure under an object due to primary radiation and the exposure to the background due to primary radiation, and $R = E_s/E_{bp}$, where E_s is the film exposure due to scattered radiation. The slope of the characteristic curve of the film is γ.

The contrast that would be observed in the radiation flux at the level of the x-ray film, if no scatter were present would be

$$C_0 = \frac{E_{bp} - E_{op}}{E_{bp} + E_{op}} = \frac{1 - \alpha}{1 + \alpha}. \qquad (2)$$

The relative visual contrast, that is the visual contrast divided by the visual contrast, C_{vo}, that would be observed if no scatter were present, is plotted in Figure 7 for a range of object contrast as a function of $R = E_s/E_{bp}$. A typical value of $\gamma = 2.7$ was used. It is seen that the degradation of visual contrast due to scattered radiation does not depend very much on the object contrast.

The techniques can also be compared by calculating the relative visual contrast that would be expected. This has been done for the case of chest radiography in Table v. It is seen that in the posteroanterior projection relative visual contrast is increased appreciably when an air gap is used. In the lateral projection under these assumed conditions the air gap techniques increase the visual contrast markedly over that which would be obtained with no grid (0.17), but they cannot achieve as good a reduction of the scattered radiation as is obtained with a grid technique.

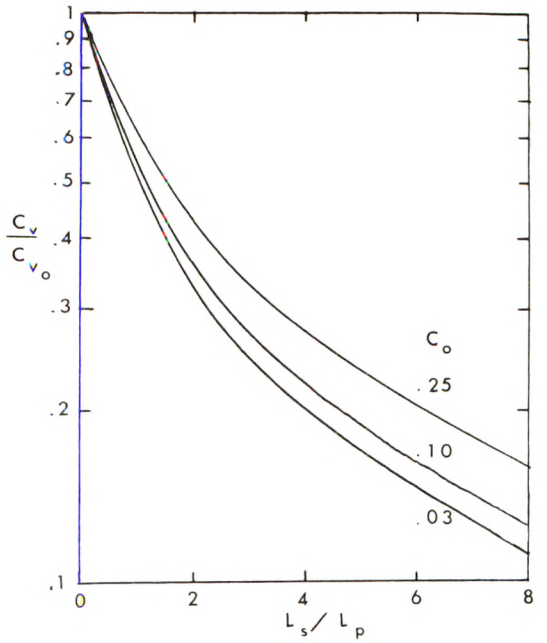


Fig. 7. Variation of the relative visual contrast, the visual contrast at the level of the observer's retina divided by the visual contrast that would be present if there were no scattered radiation, as a function of the ratio of the film exposure due to scattered radiation, E_s, to the film exposure due to primary radiation, E_{bp}. Calculations were made for 3 different degrees of subject contrast, C_0.

Magnification and Air Gap Techniques

Air gap techniques change the relative magnification of the examination. This may be compensated for by using a larger target-to-film distance. To examine this effect in chest radiography, calculations were made of the magnification of an object located at midplane in 22 cm. diameter chest for posteroanterior projection and at the midplane of 30 cm. diameter chest for the lateral projection. The relative magnification was calculated as a function of air gap width and TFD and compared to the magnification for conventional exposures using a 6 foot TFD. Results of this calculation are shown in Figure 8, where it is seen that the magnification of a 5 inch air gap at a 10 foot TFD is very nearly equal to the magnification of a conventional examination at 6 foot TFD. A roentgenogram made with a 10 inch air gap at 10 foot TFD would show a slightly greater magnification than for conventional techniques.

Another approach to this problem is to consider the effect on modulation transfer

TABLE V

COMPARISON OF VISUAL CONTRAST WITH CONVENTIONAL AND AIR GAP TECHNIQUES IN CHEST RADIOGRAPHY

Projection	Measured Thickness, (cm.)	Density, (gm./cm.^{3})	Equivalent Water Thickness (cm.)	L_s/L_p			Relative Visual Contrast for $C_o=0.1$		
				Air Gap			Air Gap		
				Conventional	5 in.	10 in.	Conventional	5 in.	10 in.
Posteroanterior	22	0.5	11	2.8	2.0	1.2	0.28	0.36	0.50
Lateral	30	0.6	18	5.8, 2.1*	4.2	2.6	0.17, 0.34*	0.21	0.30

* Calculated from the data of Hondius Boldingh for a medium weight grid.

function of the system. Typical results of this approach are shown in Figure 9 for the posteroanterior projection and Figure 10 for the lateral projection. Experimental data for Par Speed screens by Rossman were used.[11] The uniform focal spot illumination approximation of Morgan was used to calculate the focal spot MTF.[10] It was assumed that exposure times could be made short enough that motion unsharpness could be neglected.

It is seen that the MTF with a 5 inch air gap at 10 foot TFD is very similar to that of conventional techniques in both projections. The high frequency response with a 10 inch air gap is slightly poorer.

PATIENT EXPOSURE

The data of Table I show that the posterior surface exposure of the patient was about the same for 5 or 10 inch air gaps as compared with conventional techniques. For an exposure simulating a lateral projection at 125 kVp the patient exposure was reduced by a factor of 5.5 for a 5 inch and by a factor of 3.8 for a 10 inch air gap compared with a conventional technique using a 12:1 grid.

DEPENDENCE OF L_s/L_p ON FIELD SIZE

The strong dependence of L_s/L_p on field size shown in Figure 4 can be explained qualitatively from Figure 11. An absorber is irradiated first with field A. The contribution of radiation scattered from some small volume a_1 to a point in the absorber, P_1, is indicated by the arrow. With a larger field, such as B, additional volumes, such as b_1, are also irradiated. For photon beams in the diagnostic energy region, however, the dose at P_1 becomes almost independent of field size as the field size increases. This is because the attenuation of the scattered photons in the absorber is so severe that points such as b_1 make very little contribution to the dose at P_1 because the path length from b_1 to P_1 is so long. This phenomenon is illustrated by the data of Jackson[9] which are quoted in Table VI.

The situation is quite different in the radiographic situation where the point of interest, P_2, representing the radiographic film, is outside the absorber. Here, as in the illustration, the path length through the absorber is only slightly longer from b_2 to P_2 than from a_2 to P_2. The attenuation in

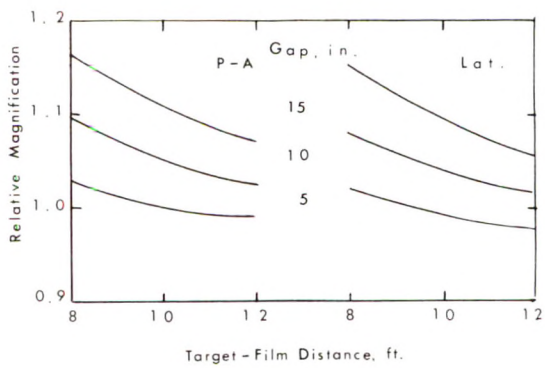


FIG. 8. Relative magnification of an object at midplane in a 22 cm. posteroanterior chest and midplane in the 30 cm. lateral chest as a function of target-film distance for various air gap widths. Results have been normalized by dividing by the magnification that would be present with conventional techniques.

air is negligibly small. Thus volumes such as b_2 make a significant contribution to the dose at P_2. Thus even for fields as large as 1,535 cm.^{2} (14×17 inches) the contribution of scattered radiation is still increasing rapidly, as can be seen in Figure 4.

SUMMARY AND CONCLUSIONS

The ratio of the light emitted by an intensifying screen in the cassette due to scattered radiation, L_s, to the amount of light emitted due to primary radiation, L_p, has been determined as a function of the parameters that must be considered in establishing air gap techniques in chest radiography. Data were obtained varying the tube potential from 60–140 kVp. Measurements were made using a 14×17 inch field at 6 and 10 foot TFD with absorbers simulating water phantoms varying in thickness from 7.4 to 21.7. The air gap was varied between $\frac{5}{8}$ and 15 inches.

It was found that there was no systematic variation of L_s/L_p as a function of tube potential. This experimental result was explained by calculations based on the theory of the Compton differential cross-section. It was found that when the incident photon energy varies from 10 keV to 140 keV the most probable angle of scattering changes only from about 53° to 45° and the cross-section changes only by about 15 per cent.

To compare the air gap technique with conventional grids, contrast improvement factors were calculated. It was found that a 10 inch air gap is comparable to a medium weight grid and that for thin absorbers, a 5 inch air gap is comparable to a light grid. These conclusions were confirmed in the instance of chest radiography by calculations of the resulting visual contrast.

In air gap techniques the TFD must be increased to compensate for the magnification introduced by the air gap. Calculation showed that a 10 foot TFD with a 5 inch air gap gave magnification essentially the same as that with conventional techniques. The magnification for a 10 inch air gap was about 5 per cent greater in both postero-

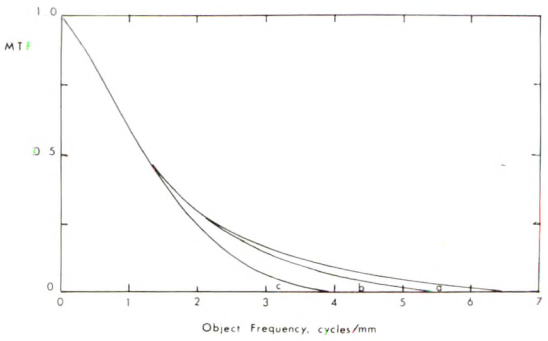


Fig. 9. Calculated Modulation Transfer Functions (MTF) for an object at midplane in a 22 cm. posteroanterior chest. A 2 mm. focal spot was assumed with Par Speed film and screens. Motion unsharpness was neglected. (a) TFD 6 ft.; 1 inch gap between back of automatic exposure timing ionization chamber and cassette. (b) TFD 10 ft.; holder plus 5 inch air gap. (c) TFD 10 ft.; holder plus 10 inch air gap.

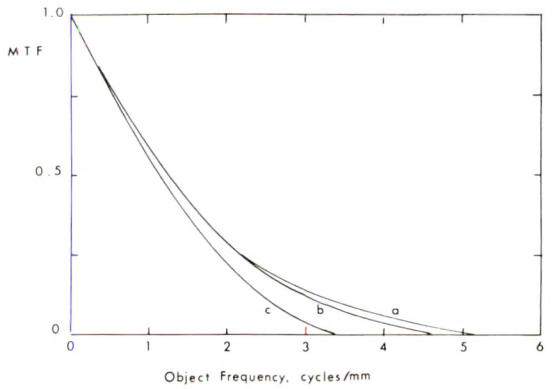


Fig. 10. MTF calculated for the case of a test object at midplane in a 30 cm. lateral chest. Otherwise conditions as in Figure 9.

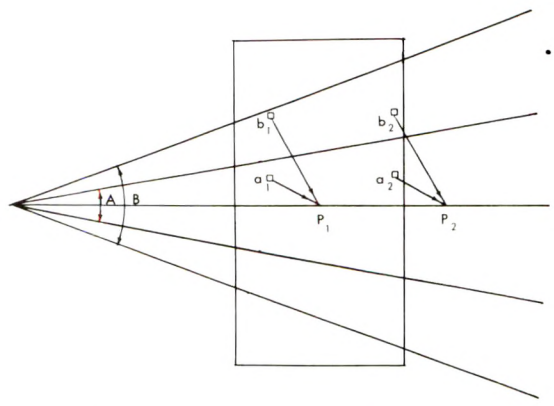


Fig. 11. Diagram illustrating the dependence of the ratio L_s/L_p as a function of area irradiated for points inside and outside of the absorber. See text.

TABLE VI

EFFECT ON CENTRAL AXIS DEPTH DOSE WHEN THE SURFACE AREA IS REDUCED BY 200 CM.²*
(HVL 3 mm. Al, 10 cm. depth)

Change in Surface Area (cm.²)	Percentage Reduction in Depth Dose
1,200–1,000	1
1,000–800	2
800–600	4
600–400	6
400–200	11
200–50	24

* Data from Reference 9.

anterior and the lateral projections. Calculations of the modulation transfer functions showed that the MTF with a 5 inch air gap at 10 foot TFD was essentially the same as conventional technique. When the air gap was increased to 10 inches, the high frequency response of the system was slightly poorer.

Patient exposure is about the same for air gap techniques as conventional techniques in the posteroanterior projection. Air gaps of 5 or 10 inches at 10 foot TFD reduce patient exposure by factors of 5.5 and 3.8, respectively, compared to a conventional technique using a 12:1 grid.

Finally, although the interest of this investigation was in chest radiography, a set of measurements were made of L_s/L_p as a function of field size. It was found that this ratio is strongly dependent on the area irradiated.

In summary, for a posteroanterior projection in chest radiography, air gap techniques at 10 foot TFD produce somewhat better visual contrast with no change in resolution or patient exposure. For the lateral projection the visual contrast is poorer than that obtained with a conventional technique using a grid, the resolution is about the same, but patient exposure is reduced by a factor of 4 to 6.

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REFERENCES

1. ARDRAN, G. M., and CROOKS, H. E. Reduction of scatter fog in chest radiography. *Brit. J. Radiol.*, 1964, *37*, 477–479.
2. BONENKAMP, J. G., and HONDIUS BOLDINGH, W. Quality and choice of Potter Bucky grids I: new method for unambiguous determination of quality of grid. *Acta radiol.*, 1959, *51*, 479–489.
3. CHITCHAKKUL, N., YOUNG, A. M. Y., and GREENFIELD, M. A. Air gap technique and its effect on radiographic detail and contrast. *Phys. Med. Biol.*, 1972, *17*, 709.
4. EVANS, R. D. The Atomic Nucleus. McGraw-Hill Book Company, Inc., New York, 1955, p. 690.
5. GOODWIN, P. M., QUIMBY, E. H., and MORGAN, R. H. Physical Foundations of Radiology. Fourth edition. Harper and Row, New York, 1970, p. 131.
6. GOULD, R. G., and HALE, J. Use of aluminum to simulate attenuation in diagnostic roentgen beams. *Acta radiol.* (Diag.), 1973, *14*, 193–195.
7. HALE, J., and MISHKIN, M. M. Serial direct magnification cerebral angiography: theoretical aspects. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 616–621.
8. HONDIUS BOLDINGH, W. Quality and choice of Potter Bucky grids V: contrast improvement factor. *Acta radiol.*, 1961, *55*, 225–235.
9. JACKSON, W. Dose assessment in diagnostic radiology. *Brit. J. Radiol.*, 1967, *40*, 301–308.
10. MORGAN, R. H. Frequency response function: valuable means of expressing the information recording capability of diagnostic x-ray systems. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *88*, 175–186.
11. ROSSMAN, K. Measurement of modulation transfer function of radiographic systems containing fluorescent screens. *Phys. Med. Biol.*, 1964, *9*, 551–557.
12. SEEMAN, H. E. Physical and Photographic Principles of Medical Radiography. John Wiley & Sons, Inc., New York, 1963, p. 58.
13. TER-POGOSSIAN, M. M. Physical Aspects of Diagnostic Radiology. Harper and Row, New York, 1967, p. 282.
14. WEYL, C., and WARREN, S. R., JR. Apparatus and Technique for Roentgenography of the Chest. Charles C Thomas, Publisher, Springfield, Ill., 1935.
15. WILSEY, R. B. Physical foundations of chest roentgenography: Part III, contrast. AM. J. ROENTGENOL. & RAD. THERAPY, 1933, *30*, 523–528.
16. WILSON, E. B. An Introduction to Scientific Research. McGraw-Hill Book Company, Inc., New York, 1952, p. 272.

VOL. 122, NO. 1

EVALUATION OF PULMONARY PERFUSION PATTERNS IN CHILDREN WITH TETRALOGY OF FALLOT

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SUCCESSFUL management of children with cyanotic congenital heart disease requires knowledge of their pulmonary blood flow patterns. Pulmonary blood flow may change as the natural history of such lesions evolve, but more frequently as a result of surgical procedures. The evaluation of pulmonary blood flow in 18 children with the tetralogy of Fallot employing radionuclide lung imaging and angiocardiography indicates that interpretation of lung scans are dependent on many factors in addition to the anatomy of the original anomaly.

MATERIAL AND METHOD

Eighteen children with the tetralogy of Fallot were investigated. The children were divided into 3 groups according to surgical repair. Three children were placed in 2 groups for a total of 21 studied.

Group I. Four children were evaluated before any surgical treatment.

Group II. Seven children had some type of shunt operation prior to investigation.

Group III. Ten children had undergone complete repair.

Table 1 indicates the sex, age, and various physiologic parameters obtained in cardiac catheterization.

Anterior, posterior and lateral views of the lung were obtained with either a single or dual probe rectilinear scanner employing I^{131} MAA. Maximum count rate and visual display were employed to estimate relative perfusion of each lung. Angiocardiography was obtained in each instance by means of image amplification and cine recording and/or a cut film changer. Contrast medium injections into the right ventricle, by means of a power injector, were carried out in both oblique and/or frontal and lateral projections.

RESULTS

Group I. Three of the 4 children had reduced pulmonary perfusion on the left shown both on lung scans and angiocardiograms (Fig. 1, *A* and *B*). The left pulmonary artery was smaller than the right in all 3. In each, the left lung received less contrast material (Fig. 1*B*). Both pulmonary arteries were extremely small in Patient 3 who had uniform accumulation of tracer in the lungs. Infundibular pulmonary stenosis was present in all 4 patients.

In each instance, the tracer study was an accurate reflection of the relative perfusion of each lung.

Group II. Functioning systemic to pulmonary artery shunts were demonstrated angiographically in all 7 children. Radionuclide lung images failed to accurately delineate pulmonary perfusion in these children. In 4 children with functional right subclavian, right pulmonary artery anastomoses, 2 had increased tracer on the left, 1 had marked accumulation on the right and 1 had uniform distribution (Fig. 2). In 3 children with functioning aorta to pulmonary artery anastomoses there was marked ipsilateral accumulation of tracer (Fig. 3*A*).

In this group pulmonary perfusion patterns were most accurately evaluated by angiography. One child with a functioning

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TABLE I

SUMMARY OF FINDINGS IN 18 PATIENTS

Patient	Sex	Age (yr.)	O_2 Saturation Systemic Artery	Pressure—mm. Hg						Comments
				Systemic Artery	Right Ventricle Inflow	Right Ventricle Outflow	Pulmonary Artery			
							Main	Right	Left	
Group I										
1	M	4	71%	100/52	100/0–7	—	—	—	—	
2	M	5	90%	102/58	100/0	—	—	12/4	—	
3	M	3	71%	100/52	115/0–7	—	—	—	—	
4	M	6	75%	85/55	85/7–5	37/−2–5	15/2	12/0	—	
Group II										
5	M	7	85%	100/50	95/0	—	—	—	—	Subclavian right pulmonary artery anastomosis
6	M	9	82%	80/50	105/0–5	80/100	—	—	—	Subclavian right pulmonary artery anastomosis
7	M	14	—	105/65	—	—	—	—	—	Subclavian right pulmonary artery anastomosis
8	M	8	82%	96/38	96/38	—	—	—	—	Subclavian right pulmonary artery anastomosis
9	M	7	85%	94/42	80/0	25/75/0	—	—	—	Aorta—left pulmonary artery anastomosis
10	M	11	82%	100/60	100/0	25/0	25/8	—	25/8	Aorta—left pulmonary artery anastomosis
11	M	1	76%	110/60	110/0	—	22/8	—	20/8	Aorta—right pulmonary artery anastomosis
Group III										
12	M	9	85%	112/68	100/10–20	—	42/20	42/20	—	Open VSD-CHF
2	M	5	95%	100/40	35/0–5	—	25/5	20/5	—	Pulmonary insufficiency
13	F	10	95%	105/56	35/0	—	27/8	26/5	—	VSD open-minimal shunt
14	F	11	92%	105/50	50/0	30/0	10/6	—	10/6	Pulmonary insufficiency
15	M	7	95%	108/67	33/0	—	−2/8	22/8	—	Subaortic stenosis
16	F	5	95%	105/55	40/0	—	35/4	25/6	18/3	Mild left pulmonary artery coarctations
17	M	12	95%	100/54	30/0	—	24/2	24/1	—	Pulmonary insufficiency—LPA obstructed
18	M	13	93%	100/62	105/0	—	22/10	12/5	—	Main and bilateral pulmonary artery coarctations
8	M	8	70%	102/63	90/30	—	83/25	43/25	—	VSD open-pulmonary insufficiency-heart failure
7	M	16	95%	105/70	30/0–5	—	25/8	—	20/8	Pulmonary insufficiency

VSD = ventricular septal defect; CHF = congenital heart failure; LPA = left pulmonary artery.

aorta to left pulmonary anastomosis had flow to the right lung *via* the right heart while flow to the left lung was excessively *via* the left heart (Fig. 3, *B* and *C*).

Group III. Five of 10 children had decreased perfusion to the left lung confirmed by radionuclide imaging and angiocardiograms. In these 5 children, the left pulmonary artery was smaller than the right, and a larger amount of contrast material passed into the right lung. The flow from the right ventricle was mainly directed into the right pulmonary artery and the aorta no longer filled from the right ventricle through the septal defect (Fig. 4, *A* and *B*).

Three of the children had absent blood flow to the left lung confirmed by radionuclide imaging and angiograms. In each child the left pulmonary artery failed to fill with right ventricular angiography (Fig. 5, *A* and *B*). Radionuclide imaging accurately demonstrated pulmonary perfusion patterns in this group of patients.

DISCUSSION

Evaluation of pulmonary perfusion by means of radionuclide lung imaging has been advocated in children with cyanotic congenital heart disease. Previous reports indicate that children with tetralogy of

Fallot and functioning systemic to pulmonary artery anastomoses have increased tracer accumulation in the lung contralateral to the side of the systemic to pulmonary artery shunt.[4,5] However, tracer accumulation was greater in the ipsilateral lung in 4 of 7 children included in this study. One child had experienced thrombosis of the contralateral pulmonary artery as the result of previous surgery. The remaining 3 children had very large aorta to pulmonary artery anastomoses.

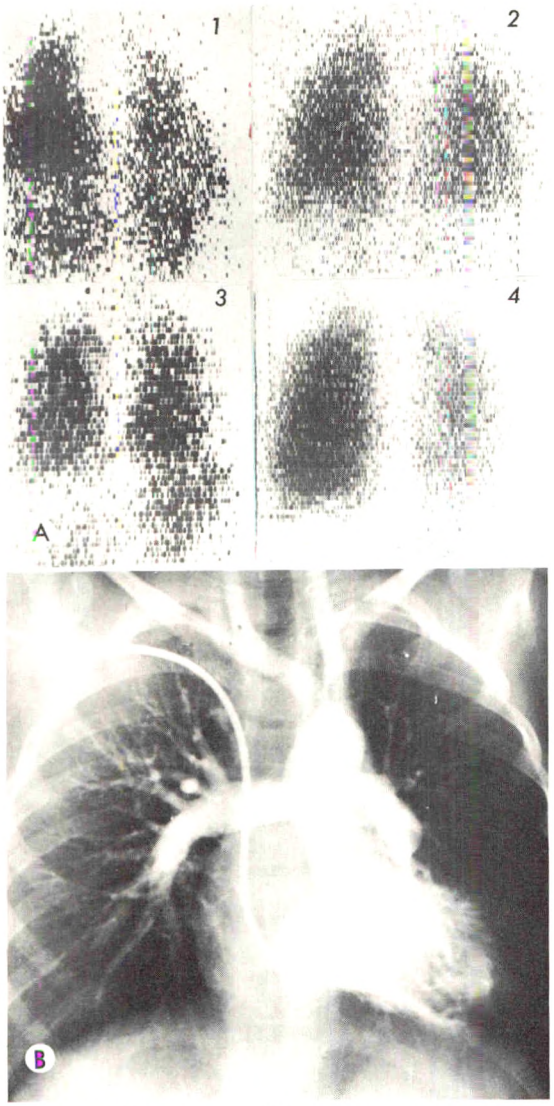


FIG. 1. (*A*) Lung scans of Patients 1–4 from Group I. Patients 1, 2, and 4 have reduced tracer on the left. Patient 3 demonstrates uniform tracer accumulation. (*B*) Anteroposterior right ventricular angiocardiogram of Patient 4. A jet is directed into the right pulmonary artery. The left pulmonary artery is smaller and arises more medially than normal, somewhat at an acute angle. There is filling of the aorta *via* a ventricular septal defect.

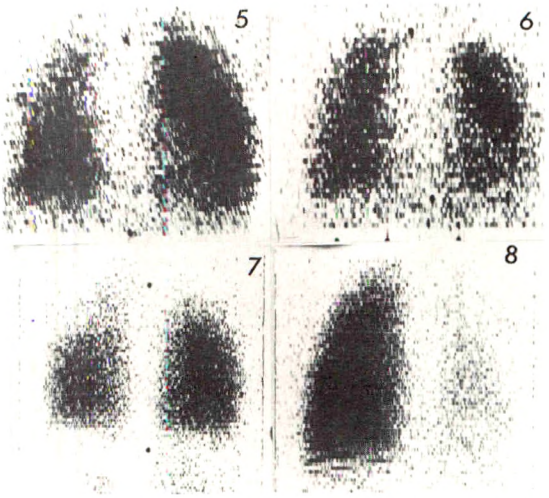


FIG. 2. Lung scans of Patients 5–8 from Group II who had functioning right subclavian right pulmonary artery anastomoses. Patients 5 and 7 have increased tracer in the left lung, Patient 6 demonstrates uniform distribution of tracer, and Patient 8 has nearly absent tracer on the left.

Of the 3 remaining children, 2 had lung images in which the major distribution of radionuclide favored the lung contralateral to a functioning shunt. Presumably in these children, the dominant flow from the right ventricle is to the contralateral pulmonary artery. The dominant flow to the ipsilateral lung is *via* the subclavian shunt. If the flow through the pulmonary valve and ventricular septal defect is about equal, there will be greater dilution of tracer in the blood of the aorta than in the blood of the pulmonary artery due to mixing with tracer-free blood from the left ventricle. Thus, the ipsilateral lung tends to accumulate less tracer. On the other hand, if the flow through the ventricular septal defect is much greater than through the pulmonary valve, and there is a large shunt present, greater accumulation can occur on the ipsilateral side by means of the shunt. For the subjects included in this study, it appears that radionuclide distribution in the lungs

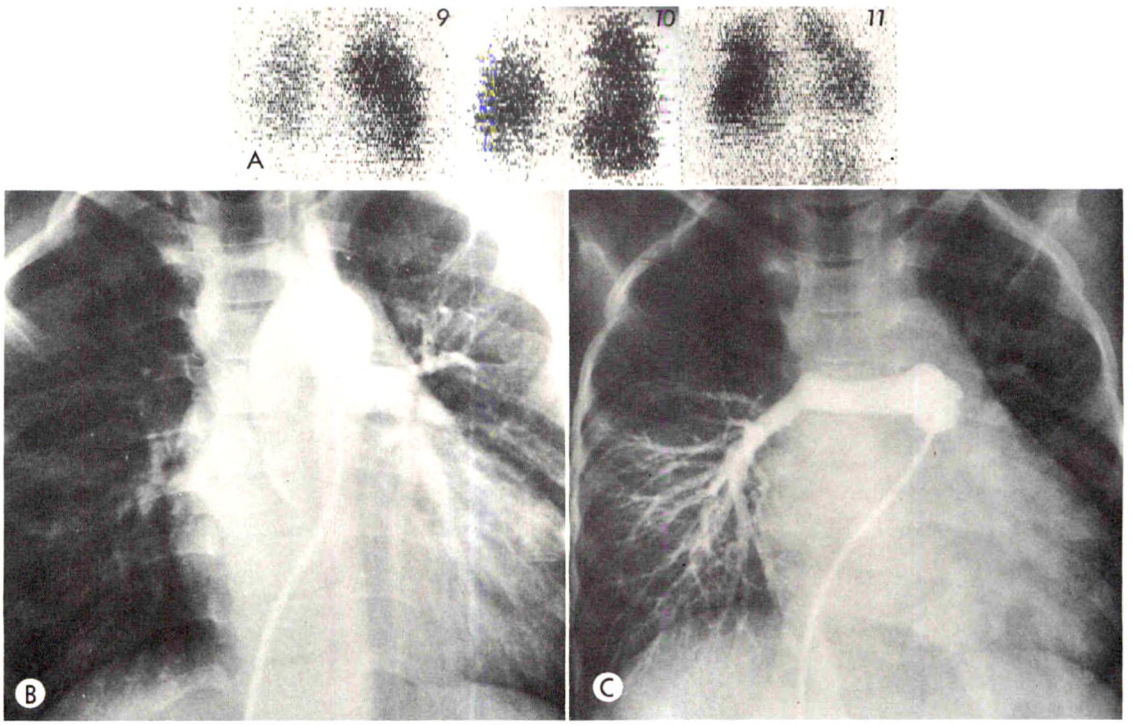


FIG. 3. (*A*) Lung scans of Patients 9–11 of Group II with functioning aorta to pulmonary artery anastomoses. There is a greater accumulation of tracer in the lung on the right of Patient 11 and on the left of Patients 9 and 10. Each had increased accumulation on the side of the anastomosis. (*B*) Anteroposterior view of an ascending aorta injection of contrast medium of Patient 10. The functioning descending aorta to left pulmonary artery anastomosis is shown. Note preferential filling of the left pulmonary artery. (*C*) Anteroposterior view of a pulmonary artery injection of contrast medium of Patient 10. There is no filling of the left lung or the right upper lobe. Previous surgical anastomosis between the right subclavian and the right pulmonary arteries had failed.

after a shunt procedure depends on the following factors: (1) Total volume of blood passing through the pulmonary valve *versus* the ventricular septal defect; (2) patency and size of the shunt; (3) patency of the pulmonary arteries; and (4) additional areas of stenosis or atresia of the pulmonary arteries.

In view of these variables, it is clear that radionuclide imaging alone cannot be used to predict the functional state of lung perfusion in a child with the tetralogy of Fallot following a systemic to pulmonary artery anastomosis.

Lung images more accurately reflect pulmonary perfusion in the untreated and totally repaired patient with tetralogy of Fallot. In these patients lung scans frequently show decreased flow to the left lung. The angiograms showed the left pulmonary artery to be somewhat smaller than the right. Flow is directed predominantly to the right lung because of the anatomy of the outflow tract of the right ventricle and pulmonary arteries. This apparently persists despite total repair. The absent perfusion on the left in 3 "totally repaired" patients was a surprising finding, but a potentially lethal complication since any disease which affects the opposite side can rapidly lead to respiratory failure. All 3 had apparent thrombosis of the left pulmonary artery as a result of surgery.

Lung scans remain useful as a screening method in evaluation of pulmonary perfusion in children with the tetralogy of Fallot,

but are much less informative than angiocardiograms which also reveal information regarding cardiac anatomy.

SUMMARY

Twenty-one children with the tetralogy of Fallot were evaluated for pulmonary blood flow patterns employing radionuclide lung imaging and angiocardiograms. Four were studied before surgery, 7 after a shunt operation and 10 after total repair.

Before surgery there is increased tracer concentration in the right lung because of the anatomy of the outflow tract of the right ventricle and pulmonary arteries. After a successful shunt operation, the distribution of tracer depends on multiple factors not entirely discernible from radionuclide lung images. After total repair radionuclide distribution is comparable to that before surgery, favoring the right lung, and frequently, exclusively so.

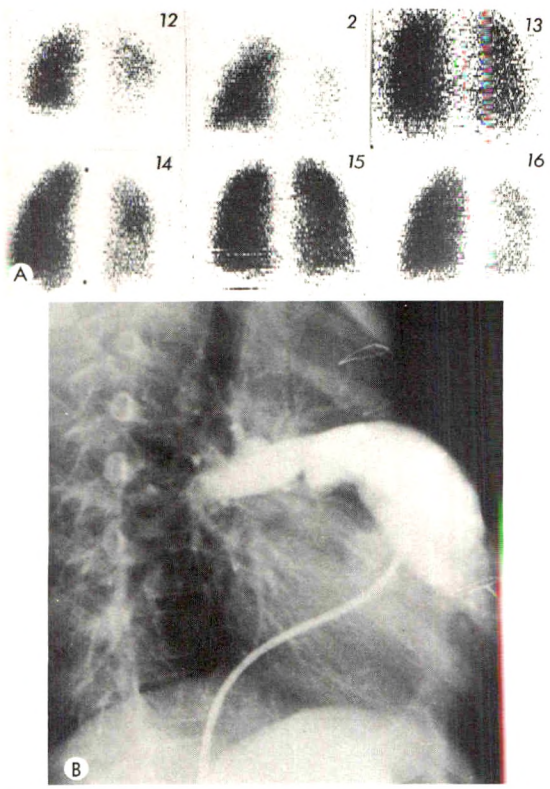


Fig. 4. (*A*) Lung scans of Patients 2, 12–16 from Group III. Only Patient 15 had uniform distribution of tracer; the others had decreased tracer accumulation on the left. (*B*) Right anterior oblique right ventricular angiocardiogram of Patient 16. The anterior bulge of the right ventricular outflow tract is characteristic of the patient who has had resection of muscle in this area. Note the direction of flow from the main pulmonary artery toward the right, while the smaller left pulmonary artery is seen in axial view.

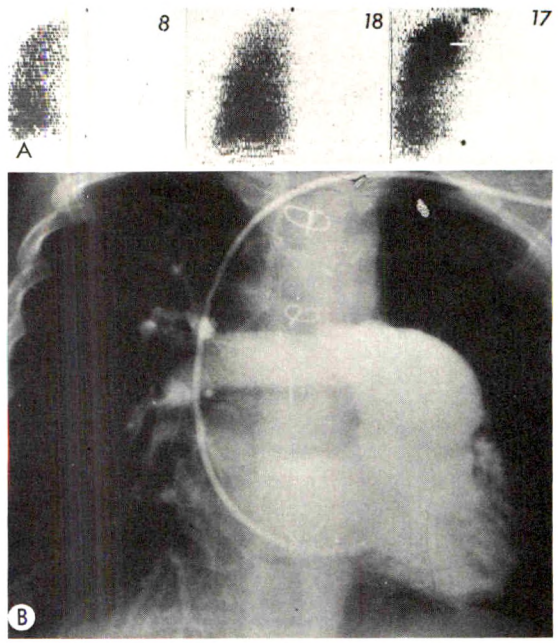


Fig. 5. (*A*) Lung scans of Patients 8, 17, 18 from Group III who had absent perfusion of the left lung. (*B*) Anteroposterior right ventricular angiocardiogram obtained following complete repair of Patient 17. There is a bulge in the outflow tract of the right ventricle. Only the right pulmonary artery is filled with contrast material.

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REFERENCES

1. Chen, J. T. T., Capp, M. P., Johnsrude, I. S., Goodrich, J. K., and Lester, R. G. Roentgen appearance of pulmonary vascularity in diagnosis of heart disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *112*, 559–570.

2. CHEN, J. T. T., ROBINSON, A. E., GOODRICH, J. K., and LESTER, R. G. Uneven distribution of pulmonary blood flow between left and right lungs in isolated valvular pulmonary stenosis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 343–350.
3. FRIEDMAN, W. F., and BRAUNWALD, E. Alterations in regional blood flow in mitral valve disease studied by radioisotope scanning. *Circulation*, 1966, *34*, 363–376.
4. FRIEDMAN, W. F., BRAUNWALD, E., and MORROW, A. G. Alterations in regional pulmonary blood flow in patients with congenital heart disease studied by radioisotope scanning. *Circulation*, 1968, *37*, 747–758.
5. HAROUTUNIAN, L. M., NEILL, C. A., and WAGNER, H. N. Radioisotope scanning of lung in cyanotic congenital heart disease. *Am. J. Cardiol.*, 1969, *23*, 387–395.
6. KIRKLIN, J. W. Tetralogy of Fallot: Caldwell Lecture, 1967. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 253–266.
7. LESTER, R. G., ROBINSON, A. E., and OSTEEN, R. T. Tetralogy of Fallot: detailed angiocardiographic study. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 92–99.
8. LEV, M., and ECKNER, F. A. O. Pathologic anatomy of tetralogy of Fallot and its variations. *Dis. Chest*, 1964, *45*, 251–261.
9. NAGAO, G. I., DAOUD, G. I., MCADAMS, A. J., SCHWARTZ, D. G., and KAPLAN, S. Cardiovascular anomalies associated with tetralogy of Fallot. *Am. J. Cardiol.*, 1967, *20*, 206–215.
10. RAO, B. N., ANDERSON, R. C., and EDWARDS, J. F. Anatomic variations in tetralogy of Fallot. *Am. Heart J.*, 1971, *81*, 361–371.
11. SAGEL, S. S., and GREENSPAN, R. H. Non uniform pulmonary arterial perfusion. *Radiology*, 1971, *99*, 541–548.

VOL. 122, NO. 1

THE RADIOGRAPHIC DIAGNOSIS OF PULMONARY HYPERTENSION FOLLOWING BLALOCK-TAUSSIG SHUNTS IN PATIENTS WITH TETRALOGY OF FALLOT*

By KENDALL MARR, M.D., FRANK A. GIARGIANA, JR., M.D., *and* ROBERT I. WHITE, JR., M.D.
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COMPLICATIONS of the subclavian-pulmonary artery anastomosis—Blalock-Taussig (B-T) shunt—include obstruction, stenosis, congestive heart failure and pulmonary hypertension.[1,3–9] If severe enough, pulmonary hypertension precludes total correction and thus there is a need for early recognition of the development of this complication.

Because of our clinical suspicion that there were reliable roentgenographic signs suggesting pulmonary hypertension, a study of patients having B-T shunts and follow-up cardiac catheterizations documenting either pulmonary hypertension or normal pulmonary artery pressure was undertaken.

MATERIAL AND METHOD

Among the 685 patients with tetralogy of Fallot, who were operated on between 1945 and 1951 at The Johns Hopkins Hospital, 601 had a B-T shunt.[7] Only 20 of these patients were known to have developed pulmonary hypertension. All of these patients had an end-to-side rather than an end-to-end anastomosis between the subclavian and pulmonary arteries. Pulmonary hypertension was documented by cardiac catheterization in 12 of these patients. In the remaining 8 patients, pulmonary hypertension was documented either by pressure measurements at the time of a second operation or by postmortem examination where the pathologic changes of severe pulmonary hypertension were demonstrated. Serial chest roentgenograms were available for review in 7 of the catheterized patients, and these provided the basis for this study. In addition, chest roentgenograms of 6 randomly selected patients with documented normal pulmonary artery pressures following B-T shunts were used as controls.

The cardiothoracic ratio (CTR), and the maximum diameter of the pulmonary artery segment supplying the right middle and lower lobes hereafter referred to as the descending portion of the right pulmonary artery (RPA), were measured from the pre- and postoperative chest roentgenograms of each patient.[2] The ratio of the maximum diameter of the descending portion of the right pulmonary artery to the cardiac width (RPA/C) was calculated. This ratio accurately reflects disproportionate changes in pulmonary artery and cardiac diameters.

Data were also abstracted from the history of each patient. Special emphasis was directed to changes in cyanosis, changes in the intensity of the continuous murmur and the presence or absence of hemoptysis.

RESULTS

The pulmonary arteries increased in size centrally and peripherally, in all patients following the B-T shunt. As pulmonary hypertension developed, the size of the central pulmonary arteries increased disproportionately when compared to the peripheral vessels (Fig. 1–3). The peripheral pulmonary vessels remained unchanged from the 1 year postoperative appearance in 4 patients. In 2 patients, there was a slight increase in the size of the peripheral pulmonic vessels as pulmonary

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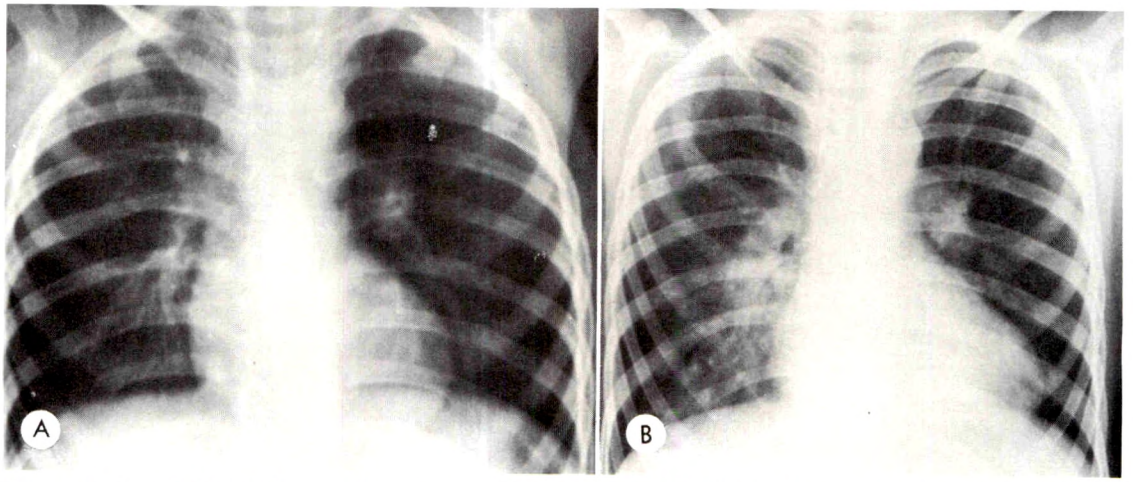


FIG. 1. Patient RH. (*A*) Preoperative chest roentgenogram and (*B*) 1 year postoperative roentgenogram. The central and peripheral pulmonary vasculature are increased 1 year postoperatively.

hypertension developed, while in the last patient they decreased in size from the early post shunt appearance. All of the patients with normotensive pulmonary arteries developed a mild increase in their central and peripheral pulmonary vasculature which remained constant on serial roentgenograms following the B-T shunt.

The CTR in the pulmonary hypertensive group increased in the year following the shunt, as it did in the control group (Fig. 4). After that, the CTR remained unchanged or increased in size in the hypertensive group and decreased in the normotensive group.

Increase in size of the central pulmonary arteries was the most reliable sign of pulmonary hypertension. This was reflected in the RPA and RPA/C measurements (Fig. 5 and 6). All patients with pulmonary hypertension developed a maximum diameter of the descending RPA in excess of 20 mm.; this was true regardless of the side of the B-T shunt. In 2 patients (WB and GK) the right pulmonary artery doubled in size 1 year following operation. Increases in the central pulmonary artery size were more gradual in the remaining patients. In the group with normal pulmonary artery pressures, most of whom were followed at least 10 years, the diameter of the descending RPA never exceeded 20 mm. (Fig. 5).

At the time of our review, 5 of the 7 hypertensive patients were living, 1 died during attempted total correction and 1 was lost to follow-up. Two of the patients were severely incapacitated by dyspnea and fatigue, while the remaining 3 patients, despite some symptoms, were able to attend school or work. Hemoptysis was a major symptom in 3 patients and was recurrent in 2 of these. The development of pulmonary hypertension was recognized clinically in these patients by both increasing cyanosis and hematocrit, and a diminution or absence of the continuous murmur.

Data from cardiac catheterization were available for review from 6 of the 7 patients with pulmonary hypertension (Table 1). Elevated pulmonary artery pressures were documented as early as 4 years after B-T shunting (Patient GK). Serial catheterization in patient RH over a 3 year period demonstrated a marked increase in pulmonary artery pressure and a fall in arterial oxygen saturation. These data are consistent with a marked increase in pulmonary vascular resistance and a fall in pulmonary blood flow.

DISCUSSION

Pulmonary hypertension following B-T shunts is one of the most serious complica-

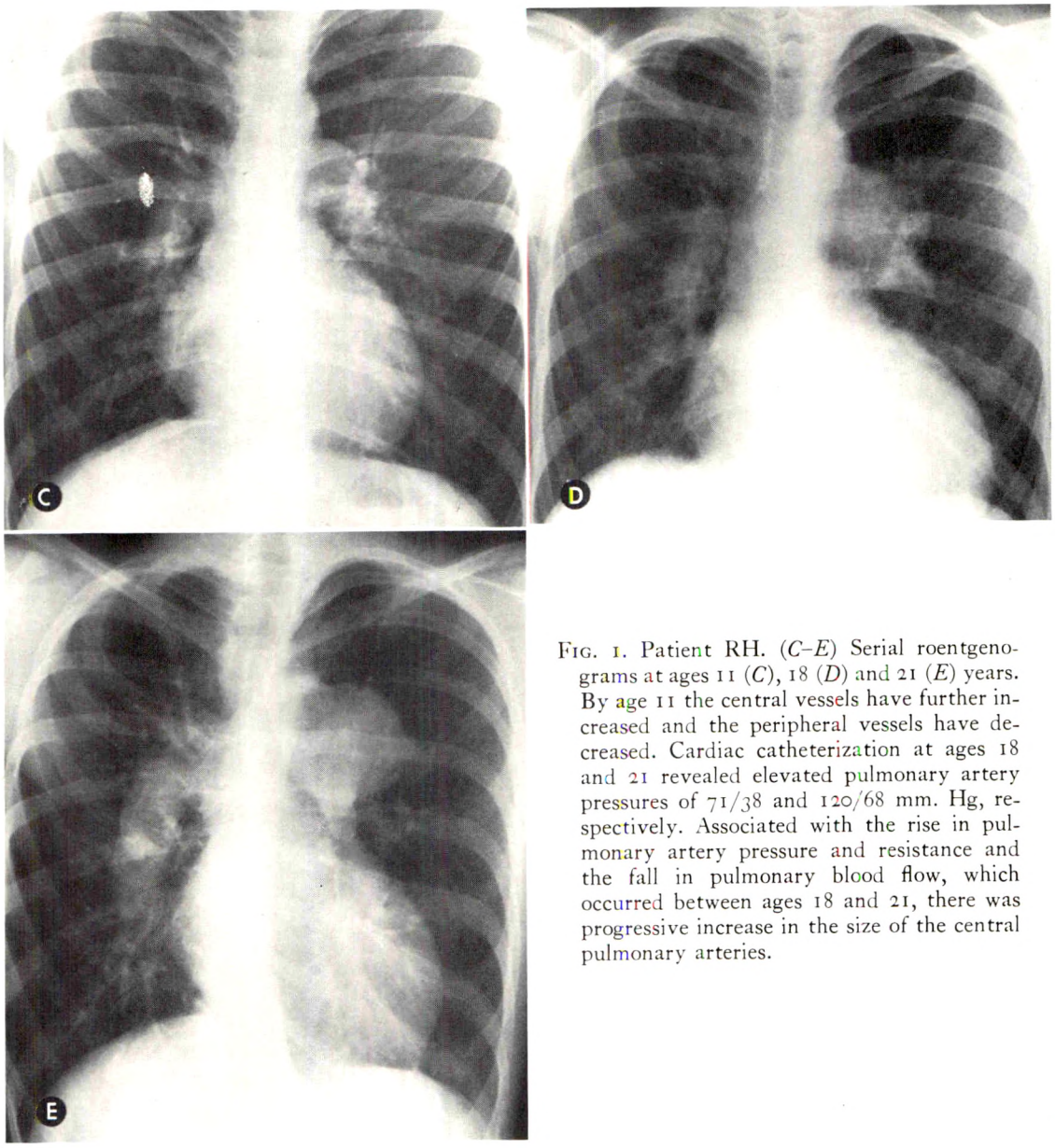


Fig. 1. Patient RH. (*C–E*) Serial roentgenograms at ages 11 (*C*), 18 (*D*) and 21 (*E*) years. By age 11 the central vessels have further increased and the peripheral vessels have decreased. Cardiac catheterization at ages 18 and 21 revealed elevated pulmonary artery pressures of 71/38 and 120/68 mm. Hg, respectively. Associated with the rise in pulmonary artery pressure and resistance and the fall in pulmonary blood flow, which occurred between ages 18 and 21, there was progressive increase in the size of the central pulmonary arteries.

tions that can occur after this type of surgery.[1,3–9] The pulmonary vasculature when examined pathologically has the typical changes of the "Eisenmenger reaction" including intimal fibrosis, medial hypertrophy and atherosclerosis of the pulmonary arteries.[9] Once the pulmonary vascular resistance (PVR) becomes elevated to a level approaching systemic vascular resistance (SVR), the patient is no longer a candidate for corrective surgery.

Clinically, patients with pulmonary hypertension, obstruction, or severe stenosis of their B-T shunts may present similarly, in that their continuous murmur diminishes or disappears, while the degree of cyanosis increases and the hematocrit rises. One frequent symptom which may occur in these patients is repeated hemoptysis.[5] When the chest roentgenographic findings are those of enlarging pulmonary arteries, then all of these symptoms and

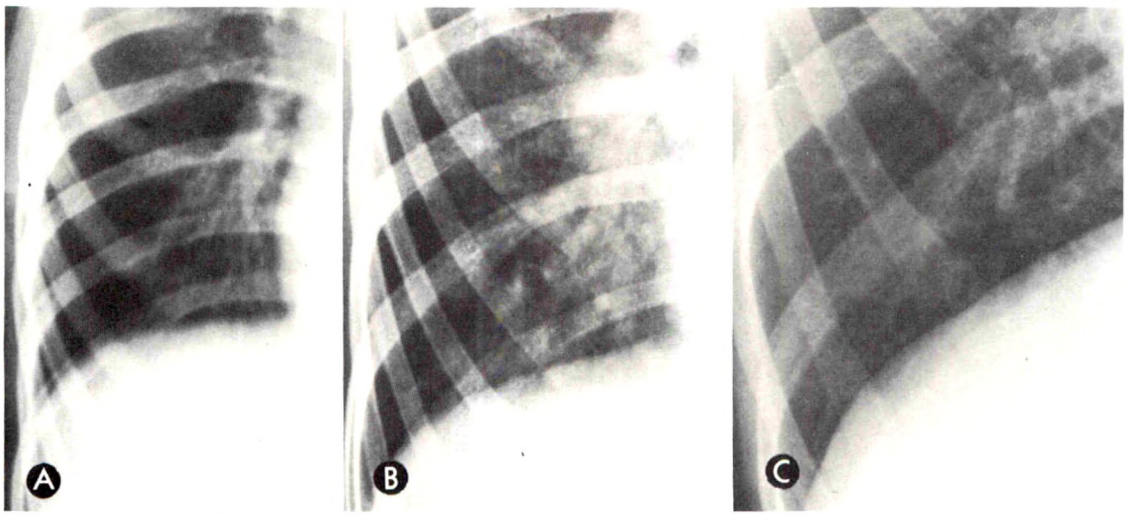


FIG. 2. Patient RH. (*A–C*) Close up views of the peripheral pulmonary vessels. Increased pulmonary vasculature is demonstrated on the postoperative roentgenograms (*B*). By age 11 (*C*), 8 years after the right B-T shunt the peripheral vessels have decreased in caliber. This is unusual and in most cases of pulmonary hypertension the peripheral vessels remained unchanged from the postoperative appearance, while the central vessels increased in size.

signs can be attributed to pulmonary hypertension.

The diagnosis of pulmonary hypertension can be suggested by analysis of serial chest roentgenograms as demonstrated in this paper. Although the time interval between the B-T shunt and catheterization was relatively long (10 years in all but 1 patient), the roentgenographic findings suggesting pulmonary hypertension occurred much earlier, often within 1 to 2 years of surgery. Increasing size of the central pulmonary vasculature was present in all patients with pulmonary hypertension. This finding allows us to separate those patients from the patients who develop obstruction or stenosis of their B-T shunt. In this latter group, the postoperative appearances of the central pulmonary vessels remain unchanged on serial roentgenographic examination.

Our experience with the "Eisenmenger reaction" occurring in patients with left-to-right shunts is similar to our experience with pulmonary hypertension in patients following B-T shunts, in that the peripheral vessels in most cases really don't taper down or become "pruned" as is so often stated. Instead, the central vessels increase in size dramatically creating the optical illusion of a "relative pruning" of the peripheral pulmonary vasculature. It is also clear that hemodynamically the elevated pulmonary artery pressures are not simply a result of increased PVR, but instead are the result of a combination of increased pulmonary blood flow (through the B-T) and increased PVR. As pulmonary hypertension progresses, the pulmonary blood flow (PBF) decreases and the pulmonary vascular resistance increases. Increasing PVR is reflected in these patients by reductions in arterial oxygen saturation, rising hematocrit, increased cyanosis, decreased continuous murmur and hemoptysis.

Early recognition of the development of pulmonary hypertension is possible, as demonstrated in patient RH (Fig. 1 and 2). In this patient, there were serial changes on the chest roentgenograms and also serial catheterizations suggesting that at one point in his course the patient was potentially correctable, while later his PVR was at a level that would have made total correction prohibitive. The radiologist is in

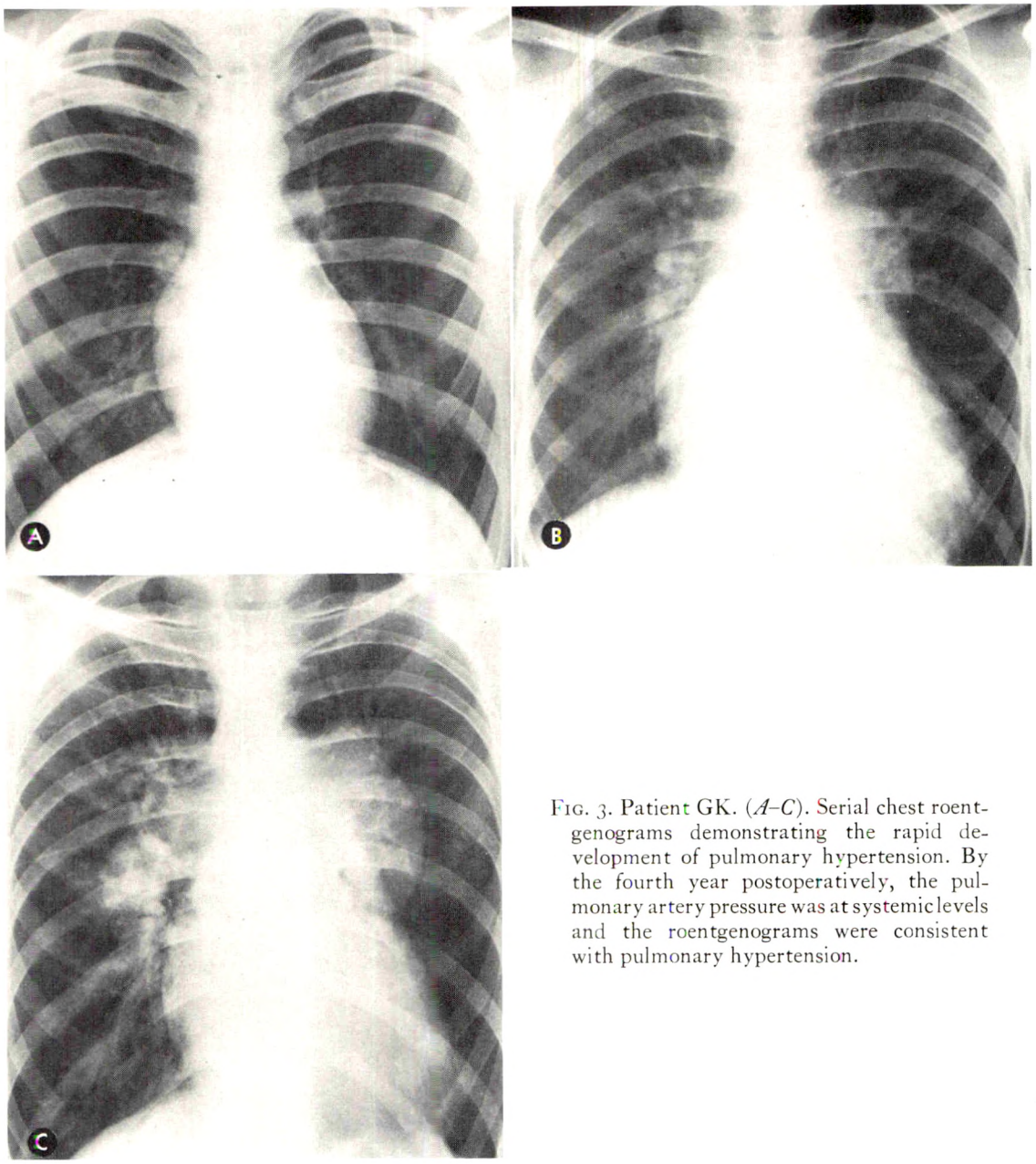


FIG. 3. Patient GK. (*A–C*). Serial chest roentgenograms demonstrating the rapid development of pulmonary hypertension. By the fourth year postoperatively, the pulmonary artery pressure was at systemic levels and the roentgenograms were consistent with pulmonary hypertension.

a unique position to be the first to suggest the diagnosis of developing pulmonary hypertension by analyzing postoperative chest roentgenograms. While rare this complication, if discovered early, can be aborted by total correction of tetralogy of Fallot and ligation of the B-T shunt. Failure to suggest pulmonary hypertension and failure to demand hemodynamic evaluation in the patient with enlarging pulmonary arteries following a B-T shunt is an error of omission which does a serious disservice to the patient.

SUMMARY

Serial chest roentgenograms of 7 patients with tetralogy of Fallot, who developed pulmonary hypertension following

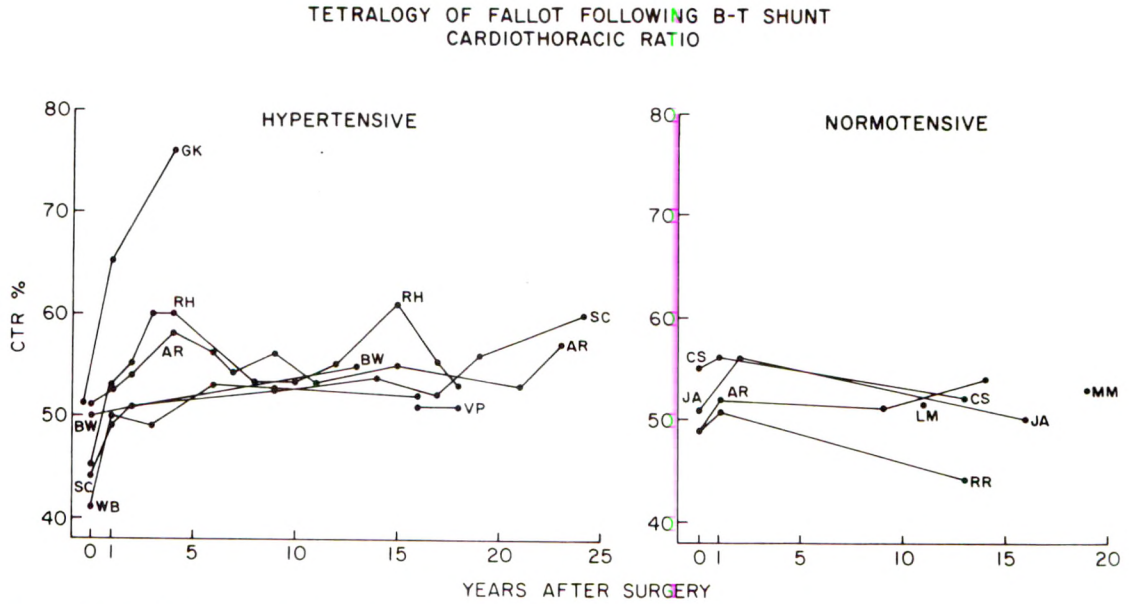


FIG. 4. Graph plotting cardiothoracic ratio (CTR) to time postoperatively in hypertensive and normotensive patients following B-T surgery. In patients with pulmonary hypertension, the CTR increased and remained so, while the CTR decreased with time in the normotensive patients.

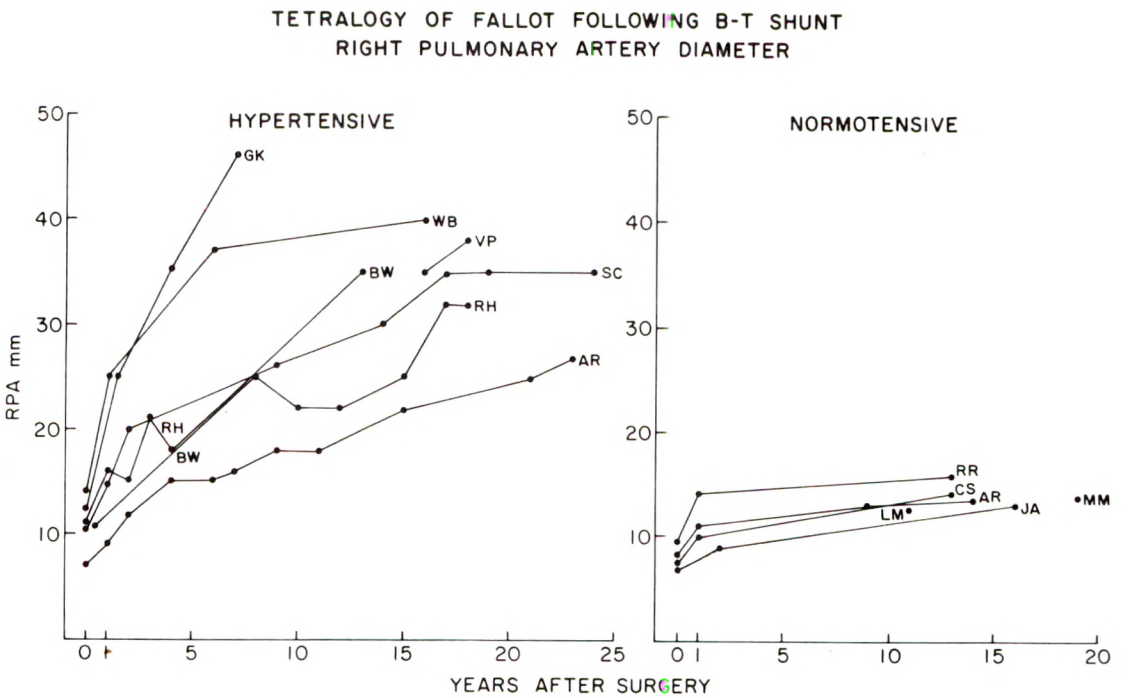
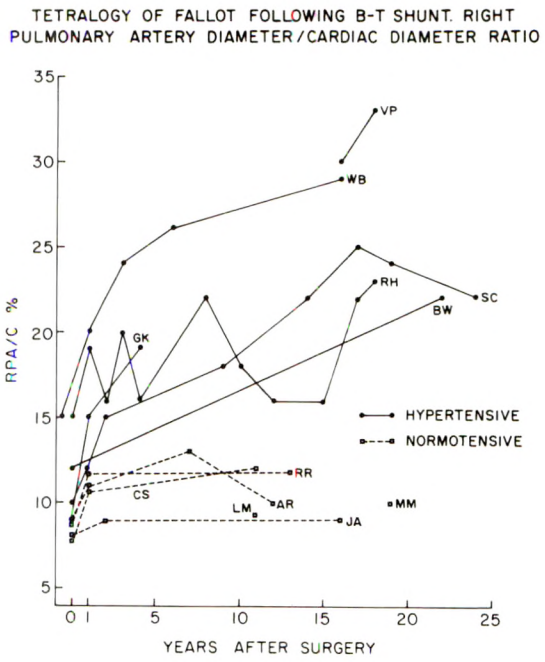


FIG. 5. Graph relating right pulmonary artery diameter (RPA) measurements to years after B-T surgery. In patients with pulmonary hypertension the diameter of the RPA progressively increased in size at varying rates, while the RPA increased slightly in normotensive patients postoperatively and then remained constant.

FIG. 6. Graph relating right pulmonary artery diameter to transverse cardiac diameter (RPA/C) on serial chest roentgenograms after B-T surgery. In the patients with pulmonary hypertension the RPA/C ratio increased, indicating that the most dramatic change occurring in pulmonary hypertension was enlargement of the pulmonary arteries.



subclavian-pulmonary artery anastomosis —Blalock-Taussig (B-T) shunt—were compared to a similar group of patients with normal pulmonary artery pressures following B-T shunts.

The pulmonary hypertensive group developed a disproportionate increase in the central pulmonary vascularity when compared to the peripheral vessels. In contrast to the normotensive group, the cardio-

TABLE I

CLINICAL AND HEMODYNAMIC SUMMARY: PULMONARY HYPERTENSION POST B-T SHUNT

Name Hospital No.	Age at B-T	Hemoptysis	Continuous Murmur At Cath	Cyanosis	Age at Cath	PA Press Syst/Diast ( )	O_2 Saturation % PA/AO
WB 409351	12	—	absent	marked	28	137/81 (97)*	71/79
SC 404356	15	—	faint	slight	28	85/47 (60)	80/85
RH 540343	3	—	present	mild	18	71/38 (52)	76/90
			absent	marked	21	120/68 (89)*	64/68
GK 534992	18	once	absent	moderate	22	113/83 (93)*	79/81
VP 462985	6	recurrent	present	moderate	22	82/50 (67)	80/82
AR 418947	4	recurrent	absent	mild	19	100/63 (75)*	86/86
BW 430983	5	NA	NA	NA	14	NA	NA

B-T = Blalock-Taussig shunt; Cath = Cardiac Catheterization; PA Press = Pulmonary Artery Pressure; Syst/Diast ( ) = Systolic/Diastolic (mean); O_2 = Oxygen; AO = Aorta; NA = Not Available.

* Aortic pressure equaled pulmonary artery pressure.

thoracic ratio, the right pulmonary artery (RPA) diameter, and the ratio between RPA diameter and transverse cardiac diameter remained elevated or progressively increased following surgery.

It is concluded that progressive enlargement of the central pulmonary arteries following B-T surgery is a reliable sign of the development of pulmonary hypertension. If not recognized early, irreversible changes in pulmonary vascular resistance occur, and the patient is no longer a suitable candidate for total correction.

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REFERENCES

1. BERNUTH, G., RITTER, D. G., FREY, R. L., WEIDMAN, W. W., DAVIS, G. D., and MCGOON, D. C. Evaluation of patients with tetralogy of Fallot and Potts anastomosis. *Am. J. Cardiol.*, 1971, *27*, 259–263.
2. CHANG, J. C. H. Normal roentgenographic measurement of right descending pulmonary artery in 1,085 cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *87*, 929–935.
3. DAOUD, G., KAPLAN, S., and HELMSWORTH, J. A. Tetralogy of Fallot and pulmonary hypertension: complication after systemic to pulmonary anastomosis. *A.M.A. Am. J. Dis. Child.*, 1966, *111*, 166–177.
4. HANCOCK, E. W., HULTGREN, H. N., and MARCH, H. W. Pulmonary hypertension after Blalock-Taussig anastomosis. *Am. Heart J.*, 1964, *67*, 817–823.
5. HAROUTUNIAN, L. M., and NEILL, C. A. Pulmonary complications of congenital heart disease: hemoptysis. *Am. Heart J.*, 1972, *84*, 540–559.
6. MCGAFF, C. J., ROSS, R. S., and BRAUNWALD, E. Development of elevated vascular resistance in man following increased pulmonary blood flow from systemic-pulmonary anastomosis. *Am. J. Med.*, 1962, *33*, 201–212.
7. ROSS, R. S., TAUSSIG, H. V., and EVANS, M. E. Late hemodynamic complications of anastomotic surgery for treatment of tetralogy of Fallot. *Circulation*, 1958, *18*, 553–561.
8. TAUSSIG, H. V., CROCETTI, A., ESHAGHPOUR, E., KEINONEN, R., KHEE, N. Y., VACHMAN, D., MOMBERGER, N., and KIRK, H. Long-term observations on Blalock-Taussig operation III: common complications. *Johns Hopkins Med. J.*, 1971, *129*, 274–289.
9. WAGENVOORT, C. A., DUSHANE, J. W., and EDWARDS, J. E. Hypertensive pulmonary arterial lesions as late result of anastomosis of systemic and pulmonary circulation. *Mayo Clin. Staff Proc.*, 1960, *35*, 186–191.

VOL. 122, NO. 1

MYOCARDIAL CALCIFICATION OF THE INFANT HEART FOLLOWING INFARCTION*

DETECTED ON THE CHEST ROENTGENOGRAM: A CASE REPORT

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THERE are numerous reports of microscopic calcification of the infant myocardium. Such calcific deposits have been observed with endocardial fibroelastosis,[8,10] myocarditis,[12,15] intramural fibroma,[16] ischemia or infarction,[7,13,14] and metastatic deposits in hypercalcemic states such as chronic renal disease, vitamin D intoxication, infantile hypercalcemia and others.[9,13]

Valvular calcification, again microscopic, has been reported to occur in the infant with congenital cardiac lesions, particularly aortic and mitral stenosis. The occurrence of microscopic calcification of the coronary arteries in this age group is well known and has been associated among other entities, with erythroblastosis fetalis, infantile hypercalcemia, polyhydramnios and hypervitaminosis D.[4]

Roentgenologic detection of calcific deposits in the infant heart centers on a few accounts of pericardial calcification following purulent pericarditis,[3] calcification of the septal muscle mass in idiopathic hypertrophic subaortic stenosis,[6] and the rare observation in this age group of calcium within a cardiac tumor such as myxoma[5] and teratoma.[2]

It is the purpose of this report to describe myocardial calcification detected on plain chest roentgenograms in a 5 week old infant suffering myocardial ischemia from anomalous origin of the left coronary artery. Roentgenographically visible calcification of this type during infancy has heretofore not been reported. Of further interest in this case is the clinical presentation which was atypical, consisting entirely of respiratory symptoms with few signs pointing directly to cardiac disease.

REPORT OF A CASE

D.D., a 5 week old male infant, was the product of a 34 week gestation complicated by intermittent vaginal bleeding since the second month. Birth weight was 5 pounds, 1 ounce. The infant displayed irritability during and after feeding and also presented with intermittent tachypnea. These symptoms progressed and at 5 weeks of age he suffered an episode of severe cyanosis and apnea. The patient was resuscitated and improved, but continued to have respiratory signs of tachypnea and intercostal retraction.

Physical examination revealed mild to moderate respiratory distress, respiratory rate 60 per minute, pulse 145, and normal temperature. No heart murmur was present. A reducible right inguinal hernia was found.

Hemogram, urinalysis, electrolytes, and sweat chlorides were normal.

Chest roentgenography and tomography demonstrated slight left ventricular enlargement with faint curvilinear calcifications overlying the anterior portion of the left ventricle. These calcific deposits were presumed to be in the pericardium or possibly myocardium (Fig. 1, *A* and *B*; and 2). Inverted T waves over the left precordium consistent with left ventricular ischemia was demonstrated on ECG.

Angiocardiography including left ventriculography revealed a dilated and poorly contractile left ventricle. The aortic root injection showed a normal right coronary artery with retrograde filling of the left coronary artery which emptied into the pulmonary artery (Fig. 3, *A* and *B*).

Following cardiac catheterization the patient developed severe congestive heart failure and expired during emergency surgery to ligate the left coronary artery.

Autopsy findings were: 1. anomalous left coronary artery originating from the pulmonary artery; 2. fine, reticular subendocardial calcifi-

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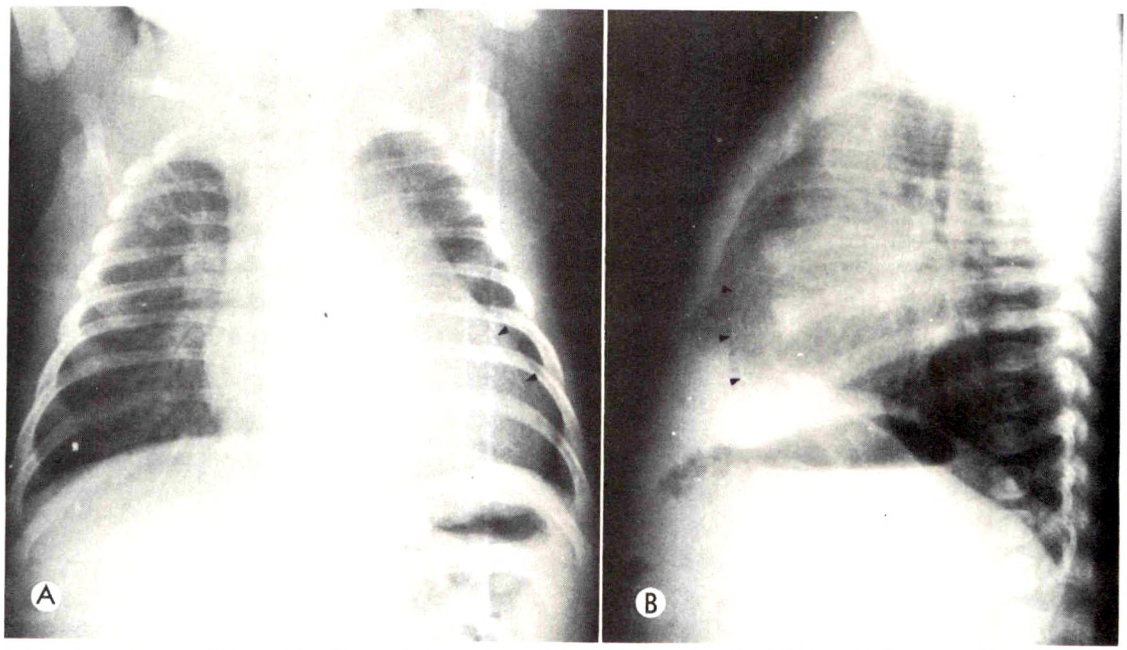


FIG. 1. (*A* and *B*) Faint, curvilinear calcification (arrows) is evident over the anterior portion of the left ventricle which is slightly enlarged.

cation of the entire anterior and lateral aspect of the left ventricle consistent with 1 to 3 month old myocardial infarction, with recent acute extension into the anterior interventricular septum (Fig. 4, *A* and *B*).

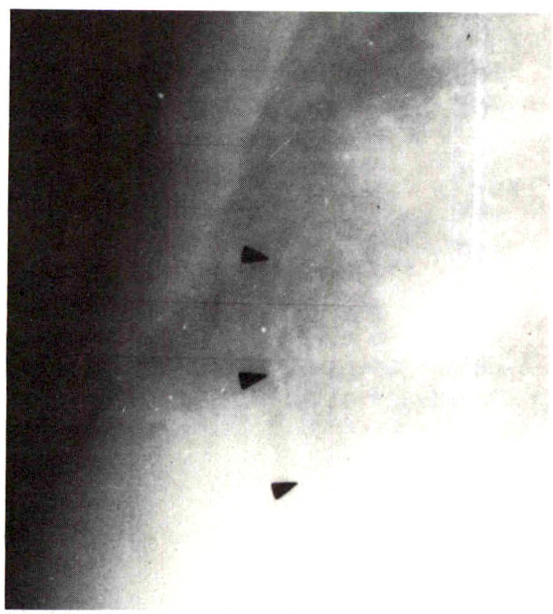


FIG. 2. Close-up of lateral chest roentgenogram reveals curvilinear and reticular calcifications to better advantage.

DISCUSSION

Anomalous origin of the left coronary artery is one cause of ischemic heart disease during infancy. In an extensive review of infantile ischemic heart disease, Bor[4] reported additional abnormalities of the coronary arteries including inflammatory disease such as syphilis, polyarteritis nodosa, and interstitial myocarditi calcific degenerative processes similar to that seen in adults with thrombosis and aneurysm formation, and coronary artery embolism associated with bacterial endocarditis. Calcification of the myocardium is not mentioned in this report.

The incidence of anomalous origin of the left coronary artery is 1 out of 300,000 births.[12] Most commonly the left coronary artery arises from the main pulmonary artery rather than the aortic root. Other forms include origin of the right coronary and sometimes both coronary arteries from the pulmonary artery. When the left coronary artery originates from the pulmonary artery, a coronary arteriovenous fistula results since a high pressure system (aorta and right coronary artery) communicates

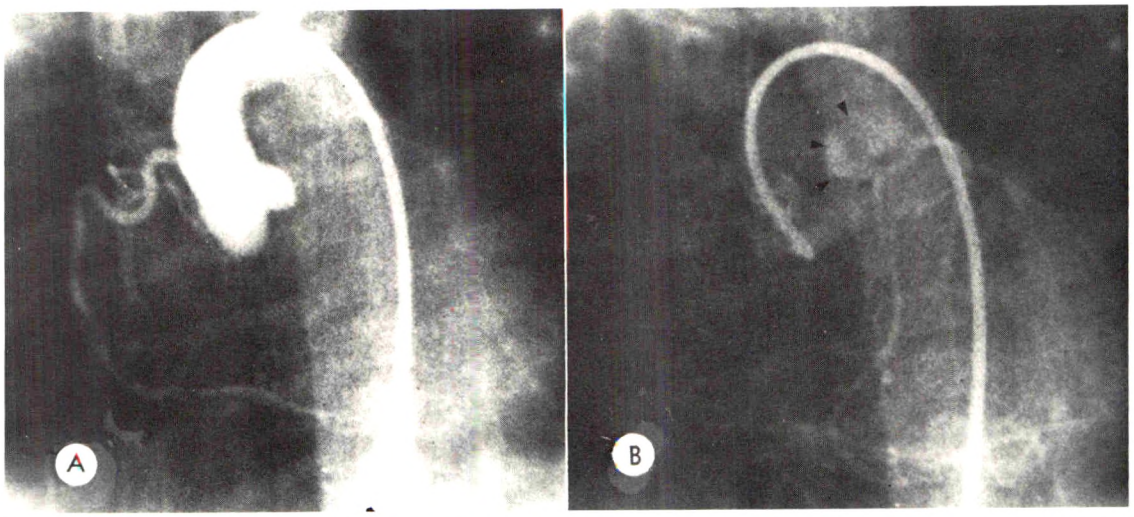


FIG. 3. (*A* and *B*) Aortograms demonstrate right coronary artery with normal origin, retrograde filling of left coronary and faint opacification of pulmonary artery (arrows).

with a relatively low pressure circulation (left coronary artery and pulmonary artery). The net effect is to shunt blood preferentially from the higher resistance left ventricular capillary network to runoff into the pulmonary artery by way of the left coronary artery. Varying degrees of myocardial ischemia result eventually leading to infarction, dilatation, and subsequent heart failure.

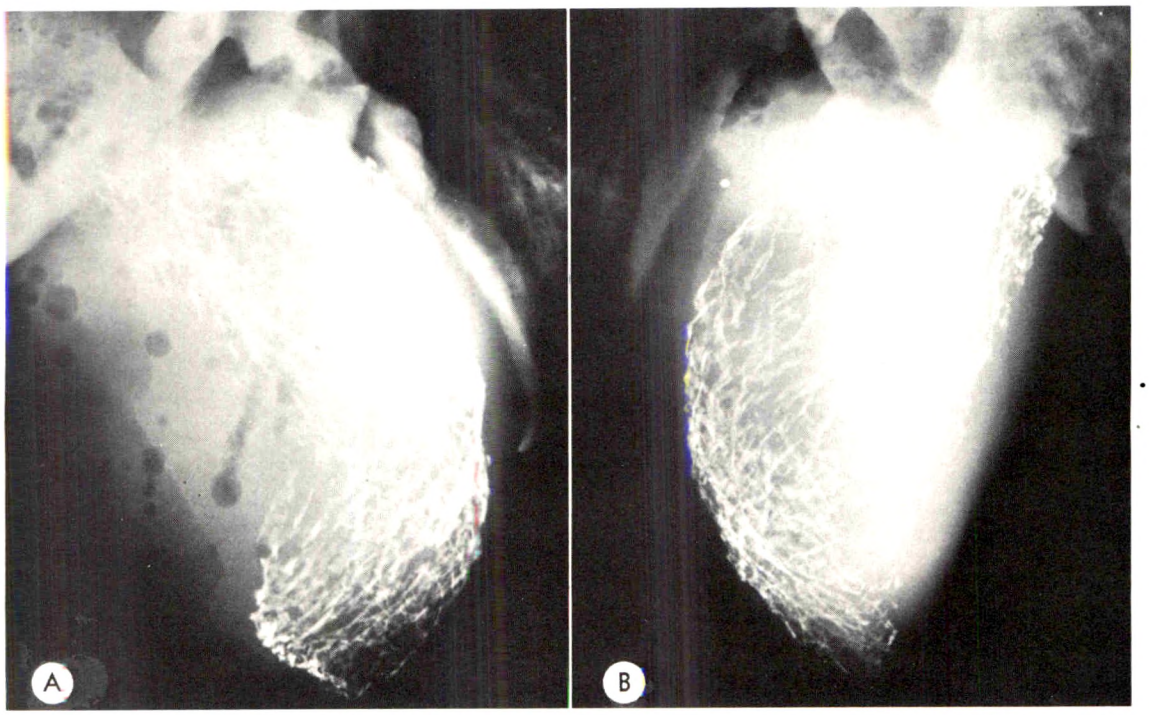


FIG. 4. (*A*) Anteroposterior and (*B*) lateral roentgenograms of postmortem heart, utilizing soft tissue technique, demonstrate extensive, fine, reticular calcification of the entire anterior and lateral wall of the left ventricle.

The diagnosis should be considered when heart failure develops in the first year of life: chest roentgenograms reveal cardiomegaly particularly if left-sided, and the ECG demonstrates evidence of myocardial ischemia in the anterior portion of the left ventricle (inverted T waves in leads 1 and 2). Aortography with injection of contrast material into the aortic root in the left anterior oblique projection confirms the diagnosis by showing rapid visualization of a large right coronary artery, retrograde opacification of the left coronary artery and subsequent opacification of the pulmonary artery.

Prognosis in the majority of cases is poor with less than 10 per cent survival.[1] Surgical treatment consists of ligation of the left coronary artery in order to obliterate the shunt and thus promote formation of collateral circulation and perfusion of the left ventricular capillary network.[17]

SUMMARY

A case of myocardial calcification visible on the plain chest roentgenogram in a 5 week old infant suffering myocardial infarction secondary to anomalous origin of the left coronary artery is reported.

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REFERENCES

1. ARYANPUR, I. Progressive infarction in anomalous origin of left coronary artery from pulmonary: indication for early surgery. *Chest*, 1972, *62*, 292–296.
2. BECK, C. S. Intrapericardial teratoma and tumor of heart. *Ann. Surg.*, 1942, *116*, 161–174.
3. BENZIN, G., and KAPLAN, S. Purulent pericarditis. *A.M.A. Am. J. Dis. Child.*, 1963, *106*, 290–294.
4. BOR, I. Myocardial infarction and ischaemic heart disease in infants and children: analysis of 29 cases and review of literature. *Arch. Dis. Childhood*, 1969, *44*, 268–281.
5. BUENGER, R., OGLESBY, P., and EGBERT, H. Calcified polyp of heart. *Radiology*, 1956, *67*, 531–536.
6. CAFFEY, J. Pediatric X-ray Diagnosis. Yearbook Medical Publishers, Inc., Chicago, 1972, pp. 524–537.
7. COLEMAN, E. N., and MACDONALD, A. M. Intrauterine myocardial infarction. *Arch. Dis. Childhood*, 1962, *37*, 444–447.
8. COSGRAVE, G. E., and KAUMP, D. H. Endocardial sclerosis in infants and children. *Am. J. Clin. Path.*, 1946, *16*, 322–340.
9. GORE, I., and ARONS, W. Calcification of myocardium: pathology study of 13 cases. *Arch. Path.*, 1949, *48*, 1–12.
10. HAMME, B., and RANSTROEM, S. Calcification of heart muscle in infancy. *Acta path. microbiol. scandinav.*, 1957, *41*, 111–118.
11. KEITH, J. D. Anomalous origin fo left coronary artery from pulmonary artery. *Brit. Heart J.*, 1959, *21*, 149–156.
12. LORENZ, V. G. Herzmuskelverkalkungen bei isolierten (Fiedlerscher) Myokarditis im Kindesalter. *Arch. Kinderh.*, 1971, *183*, 217–222.
13. MACHNIK, G., and CHRISTOF, R. Ein Beitrag zur Arteriopathia Calcificans Infantum. *Arch. Kinderh.*, 1969, *180*, 70–80.
14. RICHART, R., and BENIRSCHTE, K. Myocardial infarction in perinatal period: report of two cases. *J. Pediat.*, 1959, *55*, 706–712.
15. STOEBER, E. Weitere Untersuchungen über epidemische Myocarditis (Schwielenherz) des Säuglings. *Ztschr. Kinderh.*, 1952, *71*, 319–356, 592–596.
16. WAALER, E., SVENDSEN, S., and HALVORSEN, J. Intramural calcified fibroma of heart. *Acta paediat. scandinav.*, 1971, *61*, 217–222.
17. WASSELHOEFT, H., FAWCETT, F., and JOHNSON, A. Anomalous origin of left coronary artery from pulmonary trunk. *Circulation*, 1968, *38*, 403–425.

VOL. 122, NO. 1

CONGENITAL LEFT VENTRICULAR DIVERTICULUM*

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CONGENITAL left ventricular diverticulum is an unusual cardiac malformation, the first case having been recorded in 1838.[18,19] Since that time, approximately 36 cases have been reported.[1,3,5,7–9,11–16,18,19] Although surgical correction has been the treatment of choice, associated intracardiac as well as extracardiac abnormalities have contributed mainly to the over-all morbidity and mortality in many of these cases.

Potts *et al.*[14] first described an unusual combination of upper anterior abdominal wall, ventral diaphragm and pericardial defects associated with a congenital left ventricular diverticulum, but it was not until 1958 when Cantrell and co-workers[3] presented 5 patients with a complex of anomalies involving the abdominal wall, sternum, diaphragm, pericardium and heart. The recurrence of this syndrome has been further described by a number of other authors.[5,7–9,11–13,16–19] Awareness of this group of malformations is of more than academic interest because of potential curability.

Two such cases have been recently seen at the Texas Children's Hospital and provide the basis of this report.

REPORT OF CASES

CASE 1. A.D.R. The patient is a 3 year old Latin American male, who was referred to the Department of Pediatric Cardiology for evaluation of a pulsatile epigastric mass. Gestational and birth histories were normal; however, an omphalocele was present at birth and successfully corrected at 1 day of life. At the age of 6 weeks, persistent vomiting necessitated another surgical procedure for hypertrophic pyloric stenosis. On both occasions a pulsatile epigastric mass was visualized but not disturbed.

The patient later underwent cardiac catheterization for evaluation of this lesion and a left ventricular diverticulum was discovered. Due to family difficulties, the patient was unable to obtain any surgical evaluation until the present admission, over 2 years later.

The remainder of the patient's history including growth and development was unremarkable.

Physical examination revealed a well developed and alert, active, acyanotic child in no distress. Vital signs were normal, and pulses were equal in all extremities. The sternum was foreshortened and beneath it could be seen an elongated pulsating epigastric structure beating synchronously with the heart (Fig. 1). The point of maximum impulse was in the fifth left intercostal space and auscultation revealed a Grade 1/6 systolic ejection murmur along the left sternal border. The liver was barely palpable beneath the right costal margin. An electrocardiogram revealed terminal conduction delay pattern. Chest roentgenogram demonstrated a midline, globular-shaped heart which was borderline in size, but otherwise no other abnormalities could be seen on the examination (Fig. 2*A*). Lateral view of the chest demonstrated the shortened sternum previously suspected with beveling at the posteroinferior portion of the last segment (Fig. 2*B*).

Cardiac catheterization and angiocardiography were performed with recording of normal pressures and oxygen saturations. Angiocardiographically, the right ventricle appeared rotated from its normal position and comprised much of the right heart border in the frontal view. Left ventricular angiograms demonstrated the contractile, tube-shaped diverticulum (Fig. 3, *A–D*). The apex was in a more midline position than normal, while the left ventricle was in an anterior location. No other intra- or extracardiac abnormalities could be detected.

The diverticulum was surgically amputated *via* an abdominal approach without the use of cardiopulmonary bypass. The diverticulum ex-

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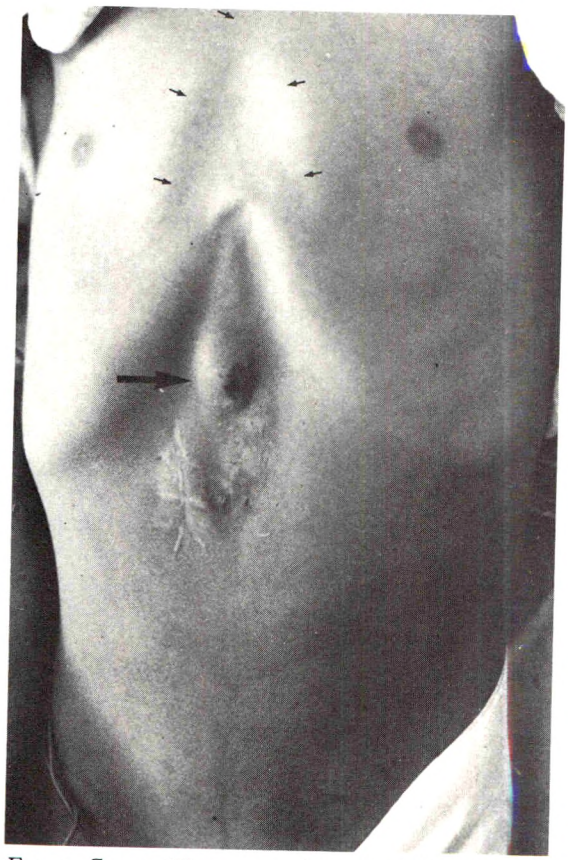
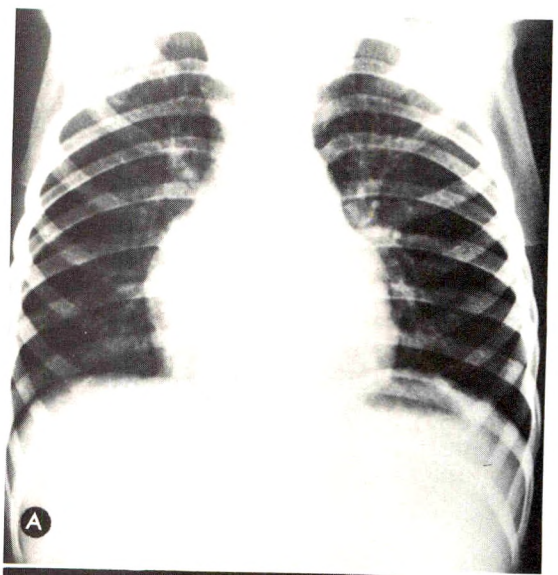


FIG. 1. Case 1. Three year old male child with congenital left ventricular diverticulum and associated anterior thoracic and upper abdominal defects. Note clinically foreshortened sternum, spreading of the lower rib cage (small arrows) and elongated, tube-like epigastric defect subsequently proven to be a left ventricular diverticulum (large arrow).

tended through the anterior portion of the diaphragm into the mid epigastric region.

The specimen was a saccular-shaped, 2.4×3 cm. muscular structure with varying thicknesses of 0.3 to 0.6 cm. Following successful surgery, the patient's immediate preoperative course was unremarkable; however, mild post pericardiotomy syndrome developed approximately 5 days later, but was satisfactorily treated. Several days later the patient became asymptomatic and was discharged.

CASE II. C.K. The patient is a 3 month old Caucasian male, who was admitted for evaluation of congenital heart disease. Gestational and birth histories were unremarkable. The child was noted at birth to have an omphalocele and



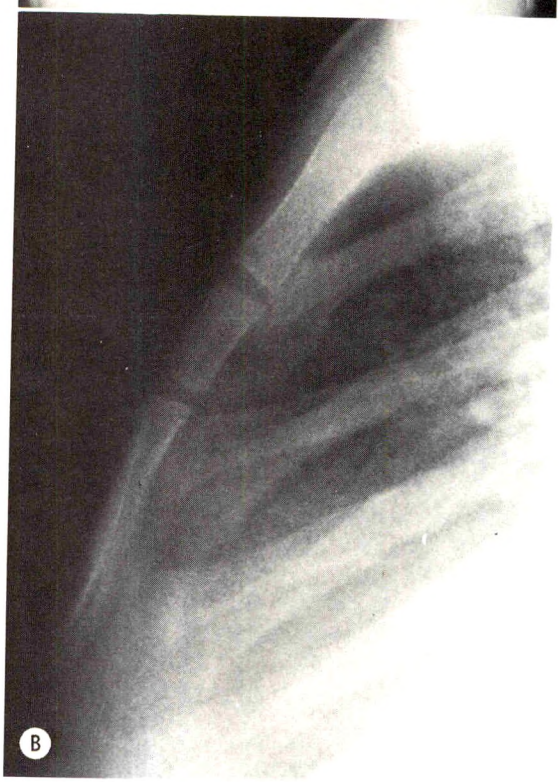


FIG. 2. Case 1. (*A*) Frontal chest roentgenogram shows a midline, globular shaped heart. The cardiac apex is in an indeterminate position. Notice prominence of the right heart border. (*B*) Lateral view shows foreshortened sternum with beveling of the posteroinferior aspect of the last ossified and relatively elongated distal sternal segment.

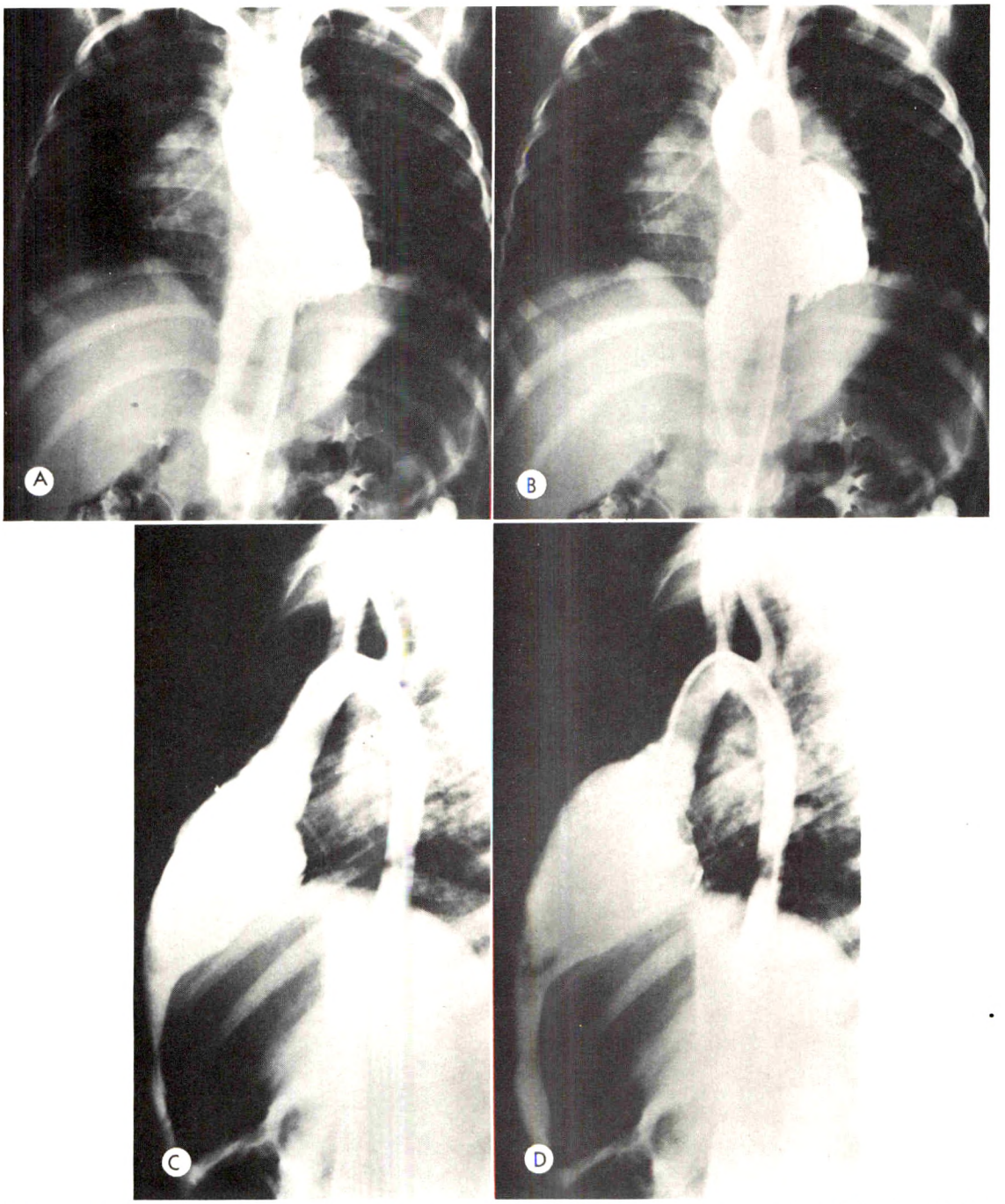


Fig. 3. Case 1. Left ventricular angiocardiograms. (*A*) Early systole showing contraction of the diverticular neck just prior to contraction of the sac itself. (*B*) Early diastolic phase with distention of the diverticular neck prior to filling of the sac. (*C*) Lateral view during systole demonstrates contraction of the diverticulum. Note the position of the left ventricle to be more anterior than usual. (*D*) Diastolic phase demonstrating distention of the diverticulum.

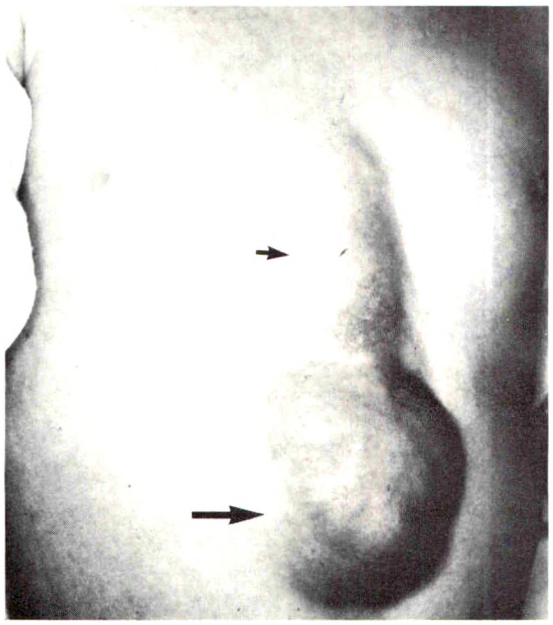


FIG. 4. Case II. Three month old baby with congenital left ventricular diverticulum and associated thoracic and abdominal defects. Note the epigastric mass (left ventricular diverticulum—small arrow) and omphalocele (large arrow).

a pulsating epigastric lesion. Shortly after birth the baby became cyanotic and was placed in an isolette. Cardiac catheterization several weeks later suggested tricuspid atresia. For a few weeks preceding admission the baby had been relatively healthy without evidence of cyanotic spells, fair weight gain and no feeding difficulties.

Physical examination disclosed normal vital signs. The skin was slightly mottled in appearance. The sternum was foreshortened and the lower anterior rib cage flared. In the epigastrium was a pulsating epigastric mass, while below this was a readily reducible, moderate-sized omphalocele (Fig. 4). Skin satisfactorily covered the omphalocele, but was discolored. A Grade 4 out of 6 holosystolic murmur could be heard over the left anterior chest wall and regular sinus rhythm was noted.

Laboratory data included: hemoglobin, 18.4 gm. per cent; hematocrit, 54.9 per cent; urinalysis within normal limits.

Chest roentgenograms showed a mild degree of cardiomegaly with central placement of the heart, possibly due to dextrocardia (Fig. 5*A*). Lateral projection showed ossification only of the manubrium with a beveling effect of the posteroinferior portion of this structure (Fig. 5*B*). In addition, the abdominal wall defect with protrusion of bowel within could be seen (Fig. 6).

Electrocardiogram showed low atrial pacemaker and biventricular hypertrophy. There was S-T segment elevation and premature ventricular contractions with compression of the diverticulum.

Cardiac catheterization was performed and the following anatomic abnormalities were identified: (1) tricuspid atresia; (2) atrial septal defect; (3) moderate-sized ventricular septal

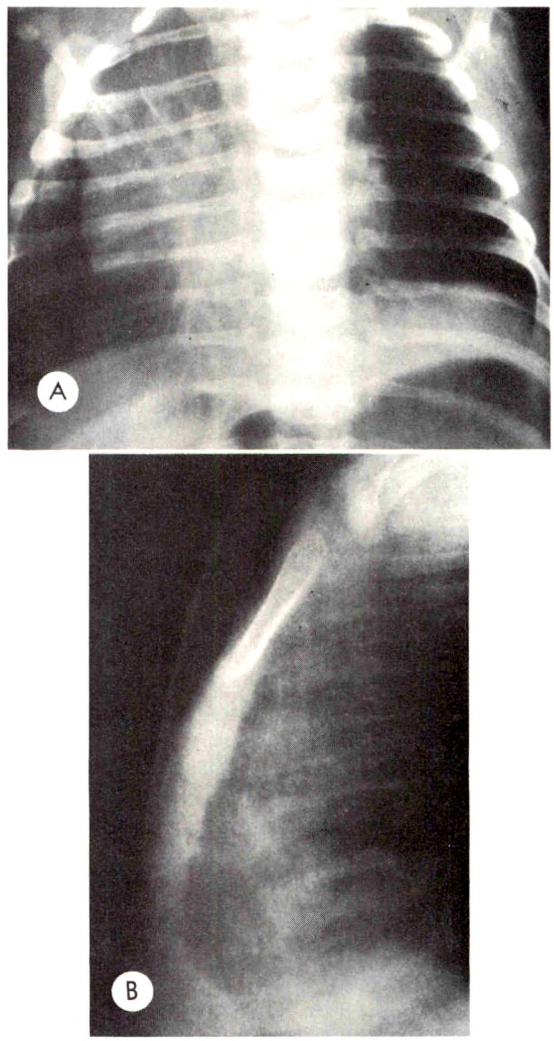


FIG. 5. Case II. (*A*) Frontal chest roentgenogram demonstrates slight cardiac enlargement with dextrocardia. Note diminished vasculature supplying the left lung. (*B*) Lateral view shows ossification only of the manubrium of the sternum with tapering of its posteroinferior aspect.

defect; (4) absent left pulmonary artery; (5) midline position of the heart (mesocardia); and (6) a prominent diverticulum of the left ventricle arising at its apex (Fig. 7, *A–D*).

Because of the child's young age and apparently stable symptomatology, it was elected to defer any surgical procedure and re-evaluate him at 1 year of age.

DISCUSSION

Congenital ventricular diverticula are of 2 types—fibrous and muscular. The origin of the fibrous form is uncertain, but they differ from the muscular variety in several ways. The location of the fibrous form is in the region of the mitral anulus, whereas the muscular variety occurs predominantly at the cardiac apex. In addition the fibrous defects are not accompanied by congenital midline thoracic or abdominal abnormalities and occur more frequently in the Negro.[6–10]

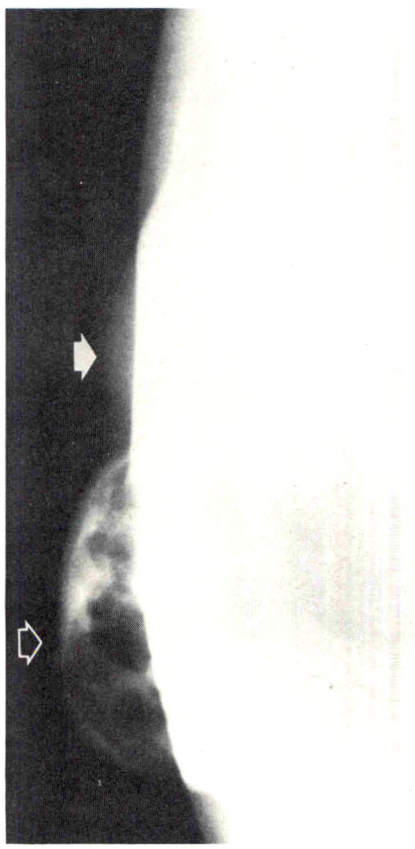


Fig. 6. Case 11. Lateral view of the lower thoracic and upper abdominal region demonstrates the omphalocele (open arrow). Note small soft tissue density just superior to the omphalocele representing the diverticulum (closed arrow).

CLINICAL FEATURES

The unusual group of anomalies which occurred in our 2 cases were brought into clear focus by Cantrell *et al.* in 1958.[3] These included: (1) a midline supraumbilical abdominal wall defect; (2) a defect of the lower sternum; (3) a deficiency of the anterior diaphragm; (4) a defect in the diaphragmatic pericardium; and (5) congenital intracardiac defects. Crittenden *et al.*, in 1959,[5] expanded upon the syndrome by including the feature of malrotation of the heart with dextroposition. In a follow-up article by these same authors,[11] a left ventricular diverticulum was found at the time of surgery in their 3 cases. This prompted them to state that the finding of a diverticulum "strongly suggests that it is a definite component of this syndrome rather than a coincidental anomaly as previously suspected."

Even though this syndrome presents variable features with respect to each defect, its recognition presents little difficulty once it is considered.

Sternal abnormalities may vary from a bifid or unilateral xiphoid process to those which present as an apparent deficiency of one or more sternal segments. The anterior rib cage in this area is often flared. Possibly the most critical abnormality of this entity is a defect of the anterior diaphragm which is contributed embryologically by the septum transversum. Because of the close contact of the diaphragmatic pericardium to the septum transversum, a defect in this portion of the pericardium occurs in nearly all cases. This allows the cardiac diverticulum to protrude from the chest into the upper abdomen. On occasion, intraabdominal contents such as bowel or omentum may protrude into the pericardial cavity *via* the defect.[17] Rarely is the diverticulum completely enclosed within the pericardium. The abdominal defects consist of mild to moderate diastasis recti and small umbili-

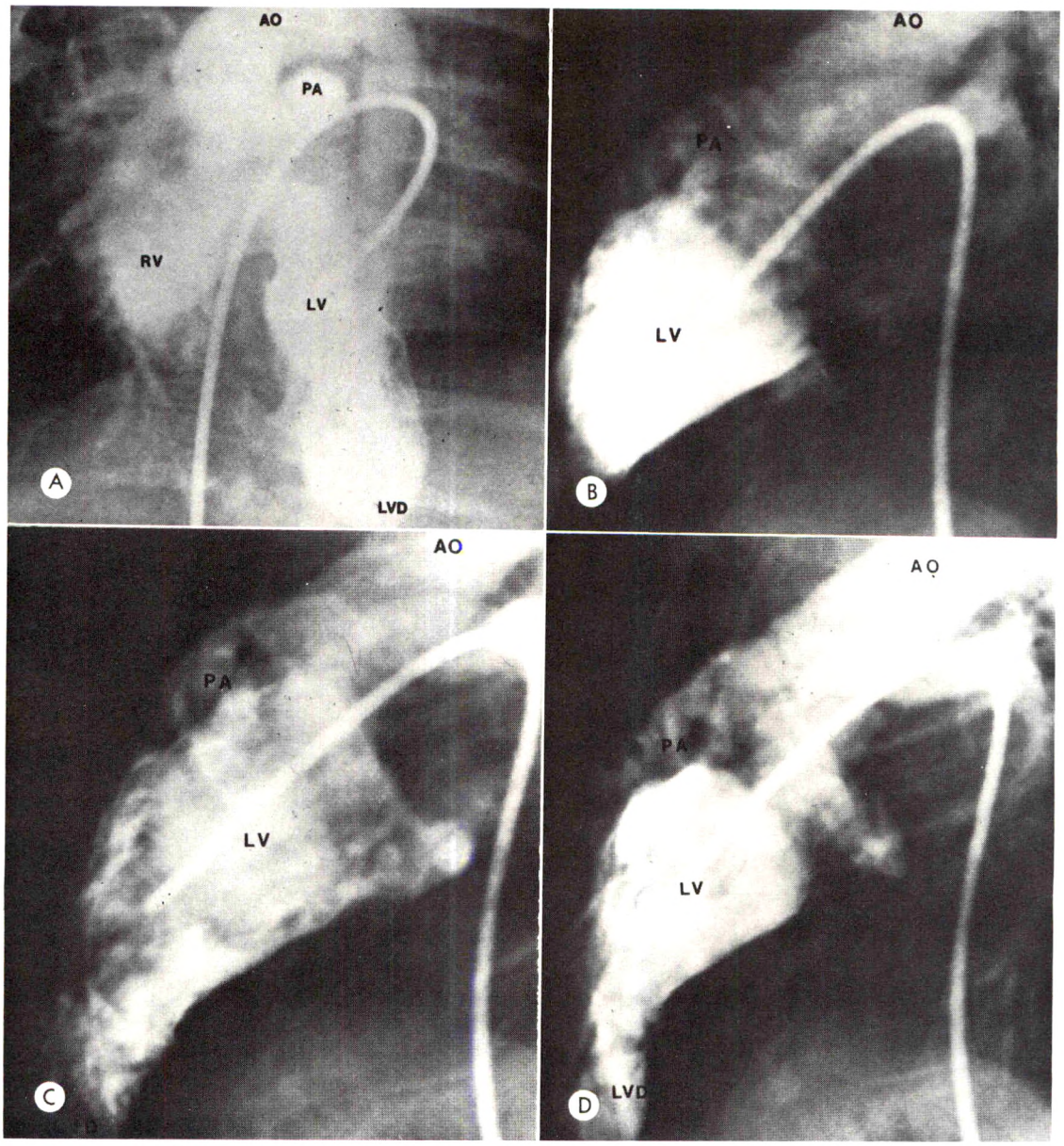


FIG. 7. Case 11. (*A*) Frontal angiocardiogram with injection into left ventricle. The catheter course progresses from the inferior vena cava to right atrium to the left atrium *via* an atrial septal defect and then to the left ventricle. Following injection of contrast medium there is filling of the right ventricle *via* a large ventricular septal defect. Note the simultaneous opacification of the aorta and pulmonary artery. (*B*) Lateral view during early to mid left ventricular diastole. The diverticulum has not filled. (*C*) Complete left ventricular diastole showing full distention of the diverticulum. (*D*) Early to mid left ventricular systole. The neck appeared to contract before the diverticulum. Note normal relationship of the pulmonary artery to the aorta (AO-aorta; PA-pulmonary artery; RV-right ventricle; LV-left ventricle; LVD-left ventricular diverticulum).

cal hernias to rather large omphaloceles. The upper rectus abdominis muscles are spread and attach to the lower thoracic cage in a more lateral location than is normal. Because of the sparse supporting tissues between the abnormal sternum and the umbilicus, one may readily appreciate the presence of visible cardiac pulsations. Intracardiac anomalies are variable and have been stressed by several authors.[3,5,7,11,12] By far the most common intracardiac lesion is an interventricular septal defect. It was recorded in all of the patients with this syndrome as initially described by Cantrell *et al.* (21 cases).[3] A more recent publication, however, cites the incidence to be 78 per cent.[12] Aside from the left ventricular diverticula in the patients herein recorded, Case 1 had no other associated cardiac defects, while Case 11 had numerous intracardiac anomalies along with atresia of the left main pulmonary artery. The presence of a muscular left ventricular diverticulum without other associated cardiac malformations (other than some degree of dextrocardia) is a distinct rarity. The most common complex cardiac malformation is tetralogy of Fallot.[3] Dextrocardia with varying degrees of malposition of the apex is commonly present. The latter can be demonstrated satisfactorily only by angiocardiography.

Of all the congenital cardiac diverticula reported, the most common location is at the apex of the left ventricle. The reason is uncertain, but in part may be due to the thinness of this part of the myocardium, as shown by Skapinker.[15] Abnormal embryologic development of the anterior diaphragm, through which a diverticulum might protrude, may also contribute to the apical predominance. Right ventricular as well as biventricular diverticula have also been documented,[4,6,7] but are not associated with thoracoabdominal defects.*

* Since this manuscript was submitted, a case of muscular right ventricular diverticulum with associated thoracoabdominal defects has been published. (Miller, J. D., and Matthews, E. C. *A.M.A. Am. J. Dis. Child.*, 1973, *126*, 814–816.

EMBRYOLOGY

A greater appreciation of the variable manifestations of this syndrome can be reached through some knowledge of the abnormal embryonic development. Several excellent articles have been written previously on the defective embryology of this entity,[3,5,14] and only segments of it will be reviewed at this time. The basic etiology of the embryologic lesions in these 2 cases as in the others with this syndrome remains unknown, but it is apparent that anomalous formation of a segment of the ventral mesoderm occurs. Perhaps the most important defect is abnormal development of the septum transversum which comprises the anterior one-third of the diaphragm. Once again, varying degrees of defectiveness of this structure, as with others embodying the syndrome, may occur. Imperfections of this structure are not related to defects of the foramen of Morgagni.[3] The diaphragmatic pericardium, which is derived from somatic mesoderm, lies in intimate relationship with the septum transversum. It seems unlikely that one of these structures would develop abnormally without a concomitant defect in the other. Consequently, the majority of patients will have defects of the ventral diaphragm and basilar pericardium. Uncertainty also remains concerning the embryologic etiology of the diverticulum. Whether there is abnormal fusion of the epimyocardium of the primitive cardiac loop at the future left ventricular apex to the anterior portion of the defective septum transversum, and subsequent descent of these structures during rotation of the heart, is not known.[8,14] It is apparent, however, that the vast majority of these patients have some degree of dextrocardia associated with the diverticulum. The explanation for the occurrence of the various other types of intracardiac malformations has not been clarified, but the commonly found interventricular septal defect may also be explained in part by incomplete cardiac rotation.

The sternum is derived from paired mesenchymal structures which lie on either

side of the midline and fuse in a cephalocaudad direction. All of the basic segments are present, but complete ventral growth is lacking. Consequently, some patients may present features suggesting absence of a portion of the sternum. The abdominal muscles are formed from the dorsal mesoderm of the somites.[3] The defects which arise within these tissues may represent incomplete ventral migration of these structures with failure to meet and fuse in the midline.[3,5] One can only speculate if the cardiac diverticulum prevents total migration of the aforementioned structures.

ROENTGENOLOGIC FEATURES

The diagnosis may be suspected by inspection of the patient, but there are several roentgenographic features which might enhance the clinical suspicion. The frontal chest roentgenogram shows the heart to be globular in shape. Its location may be midline, but some degree of dextrocardia is often apparent (Fig. 2*A*; and 5*A*). The size of the heart and appearance of the pulmonary vascularity depend upon associated cardiac defects. Lateral view of the chest will generally demonstrate a foreshortened sternum, while closer inspection of the lower portion of the sternum shows it to be beveled at its posteroinferior aspect. Perhaps constant cardiac pulsations in addition to poor soft tissue support at the sternal defect allow for this tapered appearance (Fig. 2*B*; and 5*B*). Below the sternum one appreciates a soft tissue bulge when the diverticulum is in diastole. Caudally, gas within an umbilical hernia or omphalocele may be visualized (Fig. 6). Ectopia cordis can be readily excluded roentgenographically by observation of the cardiac structure within the confines of the chest. The left ventricular diverticulum as well as any associated intra- or extracardiac defects can only be diagnosed with certainty preoperatively by angiocardiography. Its shape is commonly tube-like (Fig. 3, *A–D*; and 7, *A–D*). The axis of the heart following filling of the left ventricle is in a more cephalocaudad direction than is normal (due to incomplete cardiac levorotation), and most of the right heart border on the frontal angiogram comprises the right ventricle. Percutaneous injection of the diverticulum has been performed in the past, but the necessity for cardiac catheterization and better visualization of intracardiac defects is obvious and would preclude the need for direct puncture. In addition, the diverticular wall may be thin, and the possibility of leakage or other hazardous complications at the puncture site is possible.

PROGNOSIS AND TREATMENT

Patients with congenital cardiac diverticula may expire in several ways. Not only are they susceptible to trauma to this exposed segment of the heart, but several cases of spontaneous rupture of a diverticulum have been reported.[9,14] Lowe *et al.*[9] postulate the mechanism of spontaneous rupture to be caused by a slight delay in diverticular contraction with respect to systole of the ventricle itself. This results in elevated pressure within the diverticulum as it prepares to contract against its narrowing neck, while the left ventricle itself is already in systole. Serial roentgenograms in our cases show this to be possible even though direct diverticular pressure measurements were not obtained. More commonly, however, fatalities are due to congestive heart failure and associated congenital heart lesions. Fatal cerebral embolization from a left ventricular diverticulum has been reported.[1] This patient, however, did not have associated anterior midline defects. Significant anomalies aside from those within the realm of this syndrome are apparently rare.[3]

Because of the potentially lethal complications of the diverticulum and associated anomalies, early surgical correction, when possible, would appear to be the treatment of choice.

SUMMARY

Two cases of congenital left ventricular diverticulum are reported.

They are associated with several unusual

anomalies including: (1) midline, supraumbilical abdominal wall defect; (2) a defect of the lower sternum; (3) a deficiency of the anterior diaphragm; (4) a defect of the diaphragmatic pericardium; and (5) congenital intracardiac defects.[3] Malposition of the heart due to incomplete rotation (dextrocardia) is a constant feature as well.

Because the lesion is potentially curable and often fatal, surgical correction when feasible should be instituted.

The roentgenologic features are discussed and some of the underlying embryologic abnormalities are considered.

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REFERENCES

1. Bell, W. E., and Ehmke, D. A. Diverticulum of left ventricle in child with fatal cerebral embolization. *South. M. J.*, 1971, *64*, 537–540.
2. Byron, S. Ectopia cordis: report of case with attempted operative correction. *J. Thoracic Surg.*, 1948, *17*, 717–722.
3. Cantrell, G. R., Haller, J. A., and Ravitch, M. N. Syndrome of congenital defects involving abdominal wall, sternum, diaphragm, pericardium and heart. *Surg., Gynec. & Obst.*, 1958, *107*, 602–614.
4. Carter, J. B., Van Tassel, R. A., Moller, J. H., Amplatz, K., and Edwards, J. E. Congenital diverticulum of right ventricle: association with pulmonary stenosis and ventricular septal defect. *Am. J. Cardiol.*, 1971, *28*, 478–482.
5. Crittenden, I. H., Adams, F. H., and Mulder, D. G. Syndrome featuring defects of heart, sternum, diaphragm, and anterior abdominal wall. *Circulation*, 1959, *20*, 396–404.
6. Cumming, G. R. Congenital diverticulum of right ventricle. *Am. J. Cardiol.*, 1969, *23*, 294–297.
7. Edgett, J. W., Jr., Nelson, W. P., Hall, R. J., Fishback, M. E., and Jahnke, E. J. Diverticulum of heart: part of syndrome of congenital cardiac and midline thoracic and abdominal defects. *Am. J. Cardiol.*, 1969, *24*, 580–583.
8. Galioto, F. M., Jr., Reitman, M. J., Vargo, T. A., Gillete, P. C., and McNamara, D. G. Congenital diverticulum of left ventricle. *Am. Heart J.*, 1974, *87*, 109–112.
9. Lowe, J. D., Williams, J. C. P., Robb, D., and Cole, D. Congenital diverticulum of left ventricle. *Brit. Heart J.*, 1959, *21*, 101–106.
10. Lurie, A. O. Left ventricular aneurysm in African. *Brit. Heart J.*, 1960, *22*, 181–188.
11. Mulder, D. G., Crittenden, I. H., and Adams, F. H. Complete repair of syndrome of congenital defects involving abdominal wall, sternum, diaphragm, pericardium and heart; excision of left ventricular diverticulum. *Ann. Surg.*, 1960, *151*, 113–122.
12. Murphy, D. A., Aberdeen, E., Dobbs, R. H., and Waterston, D. J. Surgical treatment of syndrome consisting of thoraco-abdominal wall; diaphragmatic, pericardial, and ventricular septal defects; and left ventricular diverticulum. *Ann. Thoracic Surg.*, 1968, *6*, 528–534.
13. Perez-Alvarez, J. J., Perez-Trevino, C., and Domene-Flor, R. Successful total correction of ventricular septal defect, diverticula of left ventricle, and anomaly of thoracoabdominal wall in 11 year old boy with cardiac dextroversion. *J. Thoracic & Cardiovasc. Surg.*, 1966, *52*, 386–394.
14. Potts, W. J., DeBoer, A., and Johnson, F. R. Congenital diverticulum of left ventricle: case report. *Surgery*, 1953, *33*, 301–307.
15. Skapinker, S. Diverticulum of left ventricle of heart: review of literature and report of successful removal of diverticulum. *Arch. Surg.*, 1951, *63*, 629–634.
16. Symbas, P. N., and Ware, R. E. Syndrome of defects of thoracoabdominal wall, diaphragm, pericardium and heart: one-stage surgical repair and analysis of syndrome. *J. Thoracic & Cardiovasc. Surg.*, 1973, *65*, 914–919.
17. Toyama, W. M. Combined congenital defects of anterior abdominal wall, sternum, diaphragm, pericardium and heart: case report and review of syndrome. *Pediatrics*, 1972, *50*, 778–792.
18. Vazquez-Perez, J., Gautier, M., Mertier, J. N., Belaisch, G., and Nouaille, J. Diverticulum of left ventricle: à propos of three cases. *Arch. mal. coeur*, 1969, *62*, 922–940.
19. Verger, P., Bricand, H., Fontan, F., Guillard, J. M., Mounicot, F. B., Sandler, B., Sagardiluz, J., and Bronstet, P. Congenital diverticulum of left ventricle: report of case treated successfully by surgery. *Ann. Pediat.*, 1970, *17*, 24–30.

NEUROFIBROMATOSIS AND CONGENITAL HEART DISEASE*

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THE cutaneous, osseous and central nervous system manifestations of neurofibromatosis have been widely recognized for many years.[4,5,10,11,15] Less attention has been given to the cardiovascular abnormalities which may also occur. The most frequently described abnormality is renal artery stenosis and secondary hypertension, especially in children.[1,9,14] Hypertension secondary to pheochromocytoma has been described in adults.[8] Cardiac anomalies in neurofibromatosis, however, have only recently been described.[12]

A review of all cases of neurofibromatosis seen at the University of Michigan Medical Center during the past 20 years (1951–1971) disclosed 78 children with this condition, of whom 6 also had congenital heart disease. The purpose of this report is to describe these 6 cases and to emphasize the increased incidence of congenital heart disease in patients with neurofibromatosis.

REPORT OF CASES

CASE I. (HG) This 9 month old male with café au lait spots and a family history of neurofibromatosis was noted to have a Grade 3/6 systolic murmur at the left sternal border and mild cyanosis. A cardiac catheterization revealed a high ventricular septal defect. At the age of 5 years he underwent repair of the defect. However, 3 years later he was re-operated on for closure of a residual defect.

CASE II. (RLR) This 21 month old male with café au lait spots, xanthoma of the scalp, mental retardation and velopharyngeal insufficiency was noted to have a Grade 4/6 systolic murmur radiating to the left axilla. Cardiac catheterization revealed pulmonic valve stenosis (26 mm. Hg systolic gradient) and peripheral pulmonic stenosis at the origin of the right and left pulmonary arteries (10 mm. Hg gradients).

CASE III. (PJR) This 4½ year old asymptomatic girl with multiple café au lait spots on the abdomen and chest was the sister of Case II. Blood pressure in the right arm was 160/90 and right leg 90/84 mm. Hg. The femoral pulses were delayed and weaker than the radial pulse. A Grade 2/6 harsh systolic ejection murmur was best heard at the right sternal border in the second interspace. Chest roentgenogram demonstrated rib notching. Cardiac catheterization revealed a high grade coarctation of the aorta just distal to the origin of the left subclavian artery with prominent collaterals (Fig. 1, *A* and *B*). She also has mental retardation and a Turner phenotype.

CASE IV. (LLR) This patient is the mother of Case II and Case III (Fig. 2). She was 12 years old when first seen with a history of easy fatigability, multiple café au lait spots, mental retardation, velopharyngeal insufficiency, a Turner phenotype and a harsh systolic murmur best heard in the pulmonic area. Cardiac catheterization in 1953 demonstrated pulmonic valvular stenosis. A transventricular pulmonary valvulotomy was carried out. Six years later she had recurrent symptoms and subsequently had a pulmonary valvulotomy using cardiopulmonary bypass.

CASE V. (RC) This 6 year old boy with multiple café au lait spots over the trunk and extremities and probably a left forehead subcutaneous neurofibroma was evaluated because of progressive easy fatigability and a Grade 3/6 systolic murmur heard over the entire precordium. Cardiac catheterization revealed an atrial septal defect which was subsequently repaired.

CASE VI. (BC) This patient with multiple café au lait spots over the abdomen and extremities and multiple cutaneous neurofibromata was first seen at the age of 5 years, when she was noted to have sinus bradycardia with

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second degree A-V block. At the age of 17 years she was found to have complete heart block. Cardiac catheterization demonstrated no abnormality except generalized cardiac enlargement.

DISCUSSION

Neurofibromatosis is an uncommon but certainly not rare disease, probably due to a mesodermal and ectodermal dysplasia that can affect any system of the body. The protean manifestations have generally been well documented.[4,5,10,11,15] Cardiovascular system manifestations are less frequently observed, however, and it was only in 1965 that the relationship of renal artery stenosis and hypertension in children with neurofibromatosis was described.[9] The association of pheochromocytoma with neurofibromatosis or other of the neurocutaneous syndromes is well known, with some estimates of a 10 to 20 per cent incidence.[8] Carol *et al.*,[3] and Glenn *et al.*[7] have each described a case of coarctation of the aorta and neurofibromatosis.

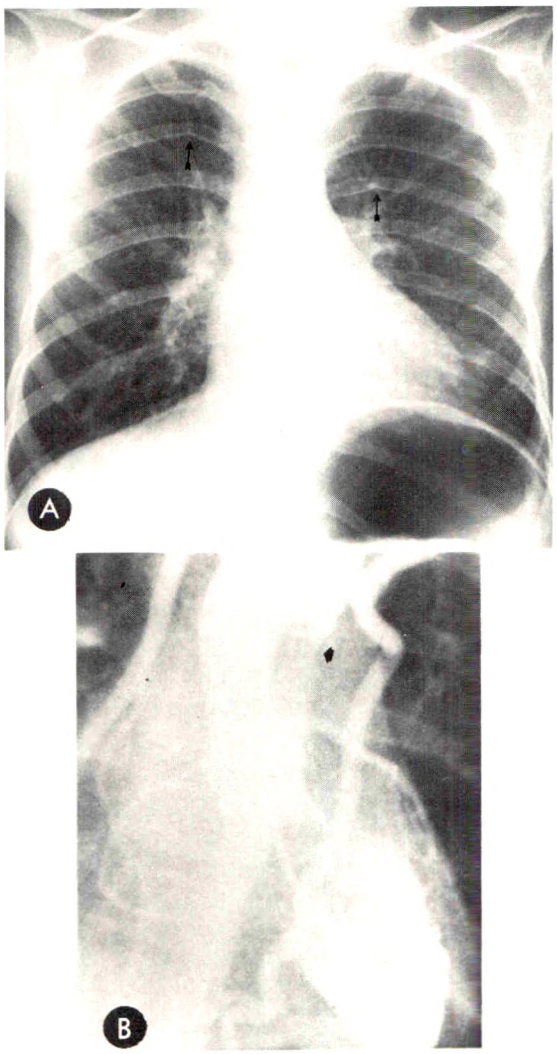


FIG. 1. Case III. (*A*) Chest roentgenogram demonstrates prominent rib notching shown by cinecardiography to be due to collateral circulation rather than to neurofibromas of intercostal nerves. (*B*) Cinecardiogram demonstrates coarctation of the aorta (arrow) with markedly enlarged internal mammary arteries.

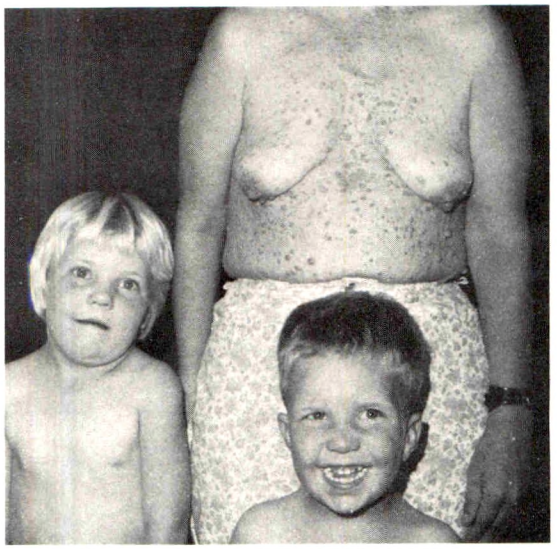


FIG. 2. Photograph of Cases II, III, and IV. Multiple neurofibromata are visible on LLR (Case IV).

Kaufman *et al.*[12] recently presented evidence that congenital heart disease, especially pulmonic valvular stenosis, may be part of the vascular manifestations of neurofibromatosis. They found 5 cases of pulmonic valvular stenosis among 19 families affected with this condition. An additional member of one family had supravalvular aortic stenosis. Crowe *et al.*,[4] however, found in 223 cases only 1 patient with possible congenital heart disease; presumably an atrial septal defect.

We have reviewed all the cases of neurofibromatosis seen at the University of Michigan Medical Center in the years

TABLE I

SUMMARY OF CASES

Name	Sex	Age at Time of Evaluation	Congenital Heart Disease	Associated Abnormalities
HG	M	9 mo.	Ventricular septal defect	—
RLR	M	21 mo.	Pulmonic valvular stenosis	Mental retardation Velopharyngeal insufficiency
PJR	F	4 yr. 6 mo.	Coarctation of upper thoracic aorta Bicuspid aortic valve	Mental retardation Turner phenotype
LLR	F	12 yr. 4 mo.	Pulmonic valvular stenosis	Mental retardation Turner phenotype
RC	M	6 yr. 2 mo.	Atrial septal defect	—
BC	F	5 yr.	Complete heart block	—

1951–1971 and found 78 children with this diagnosis. In this group there were 6 cases with congenital heart disease. Three were males and 3 females, ranging in age from 9 months to 12 years. Generally, the patient presented because of the cardiac symptomatology (Table I).

The incidence of congenital heart disease has been found to be from 0.32 to 0.64 per cent of live births.[2,13] Our series, therefore, indicates a definite increased incidence of congenital heart disease and neurofibromatosis (7.7 per cent). The exact incidence, however, is uncertain since 3 of our cases are from the same family and since the diagnosis of neurofibromatosis is often not made until the second or third decade. Particularly, there is an increased incidence of pulmonic valvular stenosis and neurofibromatosis in view of the 5 cases of Kaufman *et al.*[12] and 2 of our 6 cases. Additionally, Rosenquist *et al.*[16] described a child with neurofibromatosis who developed stenosis of the pulmonary outflow tract, but who had a normal appearing pulmonic valve. It is of particular interest that the association of pigmentary cutaneous disorders and pulmonic valvular stenosis has also been reported to occur in the multiple lentigines syndrome and the Watson syndrome.[12]

A previous report in 1940 described the association of coarctation of the upper thoracic aorta and neurofibromatosis in a 7 year old boy.[3] Glenn *et al.*[7] described a case of coarctation of the lower thoracic aorta in a patient with neurofibromatosis. Therefore, our case is only the third demonstration of this association. These cases are particularly interesting from a roentgenographic standpoint in that both coarctation and neurofibromatosis can produce rib notching.[10] In our case, however, collateral vessels secondary to the coarctation are the proven cause for the notching. Congenital heart block has not been previously reported in neurofibromatosis.

The increased incidence of congenital anomalies of the heart in patients with neurofibromatosis may be based on the fact that the heart embryologically has a mesodermal origin and therefore is likely to be involved in this dysplasia.

SUMMARY

A review of 78 cases of neurofibromatosis in children disclosed 6 with congenital heart disease. This included 2 cases of pulmonic valvular stenosis and 1 case each of coarctation of the thoracic aorta, atrial septal defect, ventricular septal defect and congenital heart block.

This material, therefore, further emphasizes the increased incidence of congenital heart disease in patients with neurofibromatosis.

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REFERENCES

1. Allan, T. N., and Davies, E. R. Neurofibromatosis of renal artery. *Brit. J. Radiol.*, 1970, *43*, 906–908.
2. Carlgren, L. Incidence of congenital heart disease in children in Gothenburg 1941–1950. *Brit. Heart J.*, 1959, *21*, 40–50.
3. Carol, Von W. L. L., Godfried, E. G., Prakken, J. R., and Prick, J. J. G. v. Recklinghausensche neurofibromatosis atrophodermia vermiculata und kongenitale Herzanomalie als Hauptkennzeichen eines familien hereditären Syndroms. *Dermatologica* (Basel), 1940, *81*, 345–365.
4. Crowe, F. W., Schull, W. J., and Neel, J. V. A Clinical, Pathological and Genetic Study of Multiple Neurofibromatosis. Charles C Thomas, Publisher, Springfield, Ill., 1956.
5. Fienman, N. L., and Yankovac, W. C. Neurofibromatosis in childhood. *J. Pediat.* 1970, *76*, 339–346.
6. Fleming, M. P., and Miller, W. E. Renovascular hypertension due to neurofibromatosis: report of case. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *112*, 452–454.
7. Glenn, F., Keefer, E. B. C., Speer, D. S., and Dotter, C. T. Coarctation of lower thoracic and abdominal aorta immediately proximal to celiac axis. *Surg., Gynec. & Obst.*, 1952, *94*, 561–569.
8. Glushein, A. S., Mansuy, M. M., and Littman, D. S. Pheochromocytoma: its relationship to neurocutaneous syndromes. *Am. J. Med.*, 1953, *14*, 318–327.
9. Halpern, M., and Currarino, G. Vascular lesions causing hypertension in neurofibromatosis. *New England J. Med.*, 1965, *273*, 248–252.
10. Holt, J. F., and Wright, E. M. Radiologic features of neurofibromatosis. *Radiology*, 1948, *51*, 647–664.
11. Hunt, J. C., and Pugh, D. G. Skeletal lesions in neurofibromatosis. *Radiology*, 1961, *76*, 1–20.
12. Kaufman, R. L., Hartmann, A. F., and McAlister, W. H. Family studies in congenital heart disease. IV. Congenital heart disease associated with neurofibromatosis. In: Birth Defects: Original Article Series. Volume VIII. Number 5. Pp. 92–95.
13. MacMahon, B., McKeown, T., and Record, R. G. Incidence and life expectation of children with congenital heart disease. *Brit. Heart J.*, 1953, *15*, 121–129.
14. Mena, E., Bookstein, J. J., Holt, J. F., and Fry, W. J. Neurofibromatosis and renovascular hypertension in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 39–45.
15. Meszaros, W. T., Guzzo, F., and Schorsch, H. Neurofibromatosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 557–569.
16. Rosenquist, G. C., Krovetz, L. J., Holler, J. A., Simon, A. L., and Bonnayan, G. A. Acquired right ventricular outflow obstruction in child with neurofibromatosis. *Am. Heart J.*, 1970, *79*, 103–108.

BENIGN PNEUMATOSIS INTESTINALIS IN CHILDHOOD LEUKEMIA*

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PNEUMATOSIS intestinalis is a well recognized manifestation of necrotizing enterocolitis in children. One of the serious complications of childhood leukemia is necrotizing enteritis of the terminal ileum and right side of the colon.[1] The roentgen demonstration of pneumatosis intestinalis in a leukemic child might then lead to an immediate and erroneous diagnosis of necrotizing enterocolitis if the radiologist is not aware of a benign form of pneumatosis intestinalis which may be seen in children with leukemia. This entity was described in the European literature in 1973.[2]

It is the purpose of this report to document this entity in the American literature, to emphasize the importance of its occurrence, and to correlate this event with body gas leakage in other locations.

REPORT OF A CASE

The patient is a 14 year old white girl, who was well until February of 1973, when she was found to have acute myelogenous leukemia. She was treated with cytosine arabinoside and prednisone with poor response. Adriomycin was begun with clinical improvement. Blood transfusions were required. In late March, an acute febrile episode led to a cavitating abscess in the right middle lobe and positive blood cultures for *Klebsiella*. This slowly resolved after massive, intravenous antibiotic therapy. On April 8, 1973 a chest roentgenogram showed the presence of gas in the wall of her colon (Fig. 1, *A* and *B*). Scout roentgenogram of the abdomen showed the gas to be colonic in origin and largely subserosal in location (Fig. 2, *A* and *B*). She had no gastrointestinal symptoms at this time. Her blood cultures subsequently became negative. She was followed clinically and roentgenographically with considerable resolution of the intramural air after 2 weeks (Fig. 3). She was discharged in remission and without gastrointestinal symptoms.

DISCUSSION

Childhood cases of benign pneumatosis intestinalis appear as an incidental asymptomatic finding during steroid therapy. In the series of 15 cases reported by Borns and Johnston,[1] 3 consisted of gastric emphysema with pyloric stenosis. None of the others had structural bowel disease, and all who did not have immune deficiency were presumably receiving steroid therapy. None had pneumomediastinum or chronic lung disease. The 10 steroid patients included 7 leukemic children also receiving other chemotherapy, all of whom showed right colon pneumatosis in which the colon remained mobile and without evidence of inflammatory disease. The pneumatosis, which was submucosal as well as subserosal, persisted for several months, then resolved spontaneously. In the other patients, steroids were given for such conditions as acute rheumatic fever and myocardiopathy. Since children normally tend to have more lymphoid tissue in the right colon than adults, Borns and Johnston[1] hypothesized that the patients with pneumatosis have severe lymphoid depletion and that the denuded Peyers patches act as defects of the mucosa permitting air to dissect into noninflamed bowel wall.

A similar course of events is suggested in a recent report by McSweeny and Stempel[2] on the subject of spontaneous pneumomediastinum and pneumopericardium in children. These authors indicated that in 2 children, spontaneous gas leakage occurred in the presence of lymphoblastic leukemia, while both patients were presumably receiving steroid therapy.

It would appear that the common denominator in all these events, including that of our patient, is steroid therapy,

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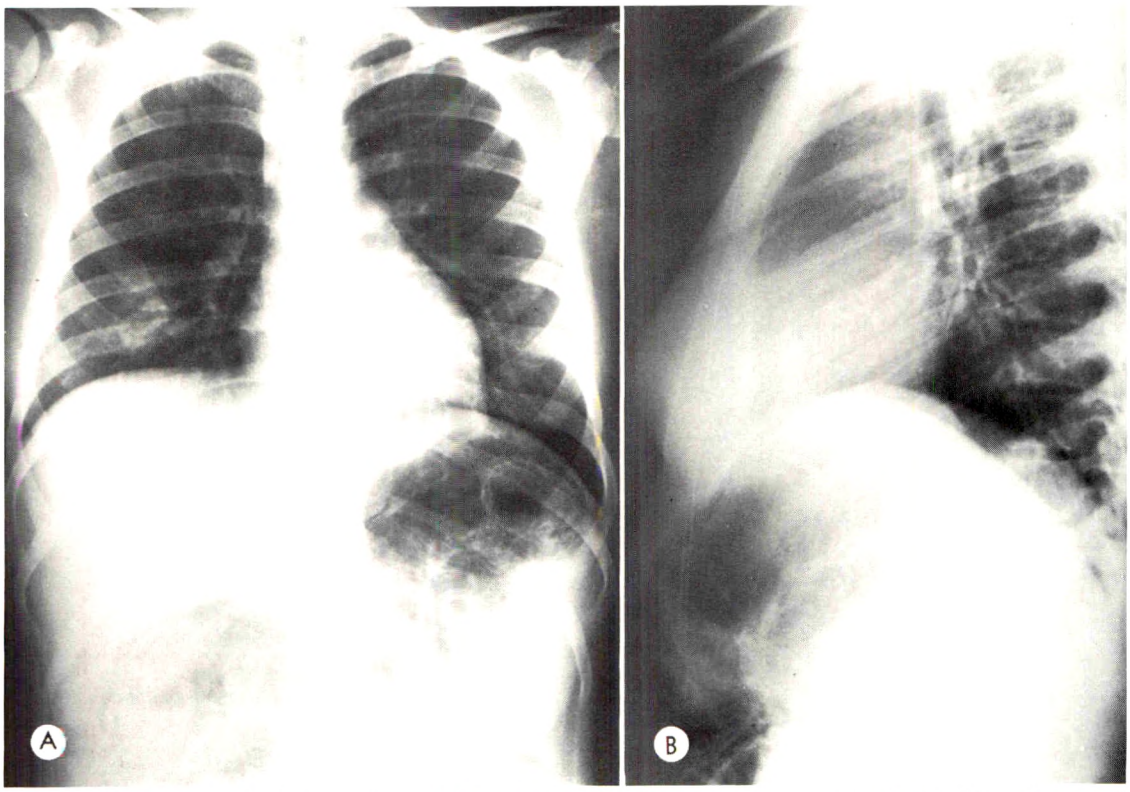


FIG. 1. (*A*) Frontal and (*B*) lateral projections of the chest show pneumatosis intestinalis. There is a small area of pneumonitis in the right middle lobe. No pneumomediastinum is present.

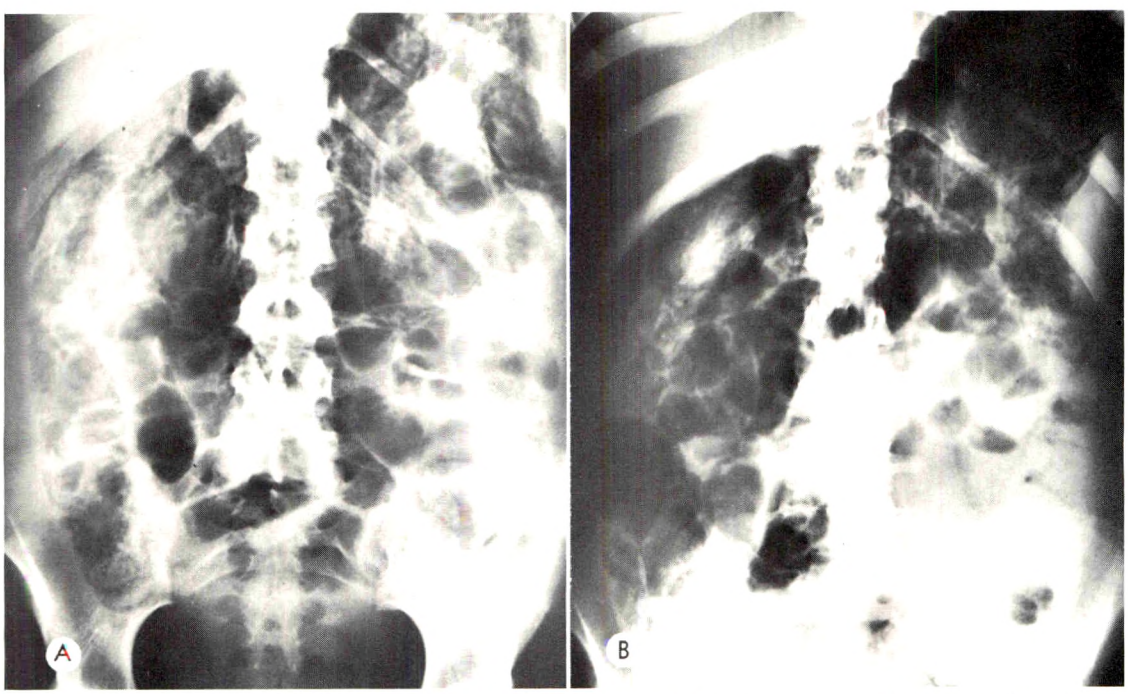


FIG. 2. (*A*) Supine roentgenogram of the abdomen showing extensive pneumatosis intestinalis which is largely subserosal. (*B*) Upright roentgenogram of the abdomen indicating that the pneumatosis is confined to the ascending and transverse colons.

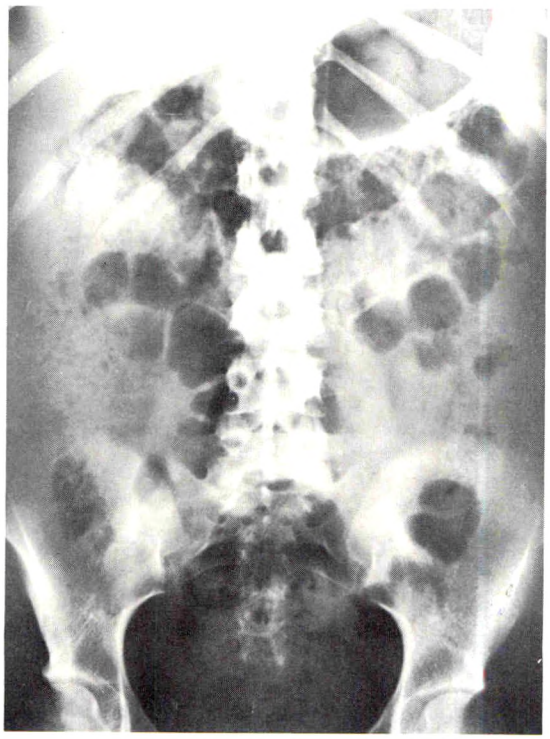


FIG. 3. Scout roentgenogram of the abdomen made 2 weeks later showing partial clearing of the pneumatosis.

rather than the leukemic process *per se*.

The recognition of the spontaneous leakage of gas in steroid treated leukemic children as a complication of therapy is of some clinical importance, particularly in that benign pneumatosis intestinalis not be mistaken for a manifestation of necrotizing enterocolitis.

SUMMARY

A case of benign pneumatosis in a child with myelogenous leukemia is reported.

The occurrence of gas leakage in the chest and abdomen appear to be related to steroid therapy, rather than to the leukemic process.

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REFERENCES

1. BORNS, P. F., and JOHNSTON, T. A. Indolent pneumatosis of bowel wall associated with immune suppressive therapy. *Ann. Radiol.*, 1973, *16*, 163–166.
2. MCSWEENEY, W. J., and STEMPEL, D. A. Non-iatrogenic pneumomediastinum in infancy and childhood. *Pediatric Radiol.*, 1973, *1*, 139–144.
3. WAGNER, M. L., ROSENBERG, H. S., FERNBACH, D. J., and SINGLETON, E. B. Typhlitis: complication of leukemia in childhood. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 341–350.

VOL. 122, No. 1

THE VALUE OF THE LATERAL ABDOMINAL ROENTGENOGRAM IN THE DIAGNOSIS OF NEONATAL HEPATIC PORTAL VENOUS GAS (HPVG)*

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ROENTGENOLOGIC recognition of gas within the hepatic portal venous system of a neonate is extremely important. The use of the lateral abdominal roentgenogram in detecting this abnormality has been illustrated and briefly discussed.[1,2,17,19]

Three recent cases suggest that the value of the lateral abdominal roentgenogram for the diagnosis of neonatal hepatic portal venous gas (HPVG) should be stressed.

REPORT OF CASES

CASE I. F.D., a 1,786 gram female, was born after a 37 week gestation to a 20 year old female. The initial physical examination revealed low-set ears, micro-ophthalmia, micrognathia, systolic heart murmur, and hypertelorism. At 13 days of age, the infant was transferred to Brooke Army Medical Center because of poor feeding and lack of weight gain. Five days after admission, there was further difficulty with feeding and a large gastric residual was noted. The infant began to pass guaiac-positive stools, and bile-stained gastric secretions were observed on the following day. Anteroposterior supine abdominal roentgenogram demonstrated bowel distention, probable air within bowel wall, and possible HPVG (Fig. 1*A*). A lateral abdominal roentgenogram confirmed the presence of pneumatosis intestinalis and HPVG (Fig. 1*B*). Despite nasogastric suction, transfusion, and antibiotics, the infant died at 20 days of age. Autopsy examination revealed necrosis of the entire ileum without evidence of perforation. Culture of peritoneal fluid grew *Escherichia coli*. Chromosomal analysis was normal.

CASE II. J.A., a 2,052 gram male, was the product of a spontaneous delivery by a 27 year old female. At 1 hour of age, a transient episode of tachypnea associated with retraction and cyanosis was noted. A chest roentgenogram at this time was normal. Abdominal distention, bile emesis, jaundice, and bloody stools were observed at 2 days of age. Anteroposterior and lateral abdominal roentgenograms demonstrated bowel distention, pneumatosis intestinalis, and HPVG (Fig. 2, *A* and *B*). The HPVG was appreciated only on the lateral roentgenogram. Because of progressive clinical deterioration, surgery was performed at 54 hours of age. Extensive necrosis of the cecum and appendix was identified. Segmental resection of the involved colon and a diverting ileostomy were performed. The infant became acidotic, suffered a cardiac arrest, and died on the morning after surgery. Pathologic examination of the resected colon showed interstitial gas, edema, inflammatory response, and mucosal sloughing. Cultures of blood, stool, and colonic mucosa all grew *Pseudomonas aeruginosa*.

CASE III. B.T., a premature 1,068 gram male, was born spontaneously after a 30 week gestation to a 28 year old female. Roentgenograms of the chest obtained at 3 hours of age demonstrated classic changes of the respiratory distress syndrome. An anteroposterior abdominal roentgenogram showed a rounded lucency just medial to the stomach (Fig. 3*A*). The lateral roentgenogram confirmed the presence of HPVG (Fig. 3*B*). Despite supportive therapy, the infant died at 70 hours of age. Autopsy demonstrated prematurity, severe respiratory distress syndrome, and no evidence of necrotizing enterocolitis.

DISCUSSION

Roentgenologic identification of hepatic portal venous gas (HPVG) was initially described by Wolfe and Evans in 1955.[23] Linear lucencies overlying the liver may be

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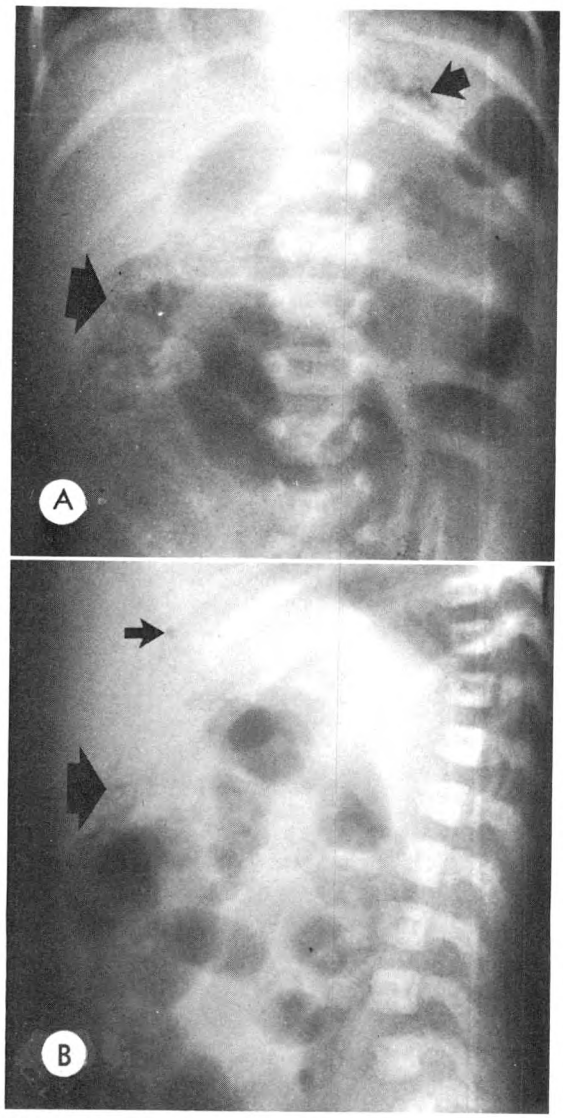


FIG. 1. Case 1. (*A*) Anteroposterior supine roentgenogram of the abdomen demonstrates bowel distention, mottled density in the right midabdomen (large arrow), and questionable linear lucencies in the left upper quadrant (small arrow). (*B*) Right lateral abdominal roentgenogram confirms the presence of extensive pneumatosis intestinalis (large arrow) as well as hepatic portal venous gas (HPVG) (small arrow).

due either to HPVG or air within the biliary tract. Biliary gas is usually located in large ducts in the region of the porta hepatis because of the centripetal flow of bile.[18] Conversely, the centrifugal flow of portal venous blood carries HPVG into finer peripheral radicles usually within 2 centimeters of the liver capsule.[16,18] The application of these classic distinguishing features may be extremely difficult in the infant.[6]

Susman and Senturia[18] noted that injection of air into a mesenteric vein permitted the initial recognition of HPVG in the relatively large left hepatic lobe of the dog. The human neonatal liver is a transverse structure with a prominent left lobe, and calcified portal vein thromboemboli occur most frequently in this left lobe.[3] This distribution of thromboemboli suggests the importance of portal vein blood flow in the relatively large neonatal left hepatic lobe. HPVG is not limited to the right upper quadrant in the infant. However, HPVG limited to the left lobe of the liver may be extremely difficult to identify on an anteroposterior roentgenogram of the abdomen because of superimposed gas in the stomach. Two of our 3 cases demonstrated possible lucencies in the left upper quadrant which were confirmed as HPVG on the lateral roentgenogram. The third case showed evidence of HPVG only on the lateral view.

The lateral abdominal roentgenogram may be obtained with either a vertical or horizontal beam. HPVG is demonstrated by both methods. Associated free intraperitoneal air in cases of necrotizing enterocolitis would be readily demonstrated by a cross-table lateral study (horizontal beam) and is suggested if an erect anteroposterior abdominal roentgenogram is not obtained. The vertical beam lateral roentgenogram seems better for demonstrating the extent of associated pneumatosis intestinalis.

Bell and co-workers[1] noted that the lateral view of the abdomen was the most sensitive detector of HPVG in cases of neonatal necrotizing enterocolitis. They recommend anteroposterior supine, anteroposterior erect, and either a left recumbent or cross-table lateral examination of the abdomen if this entity is suspected. Once the diagnosis of necrotizing enterocolitis has been confirmed, serial roentgenograms at

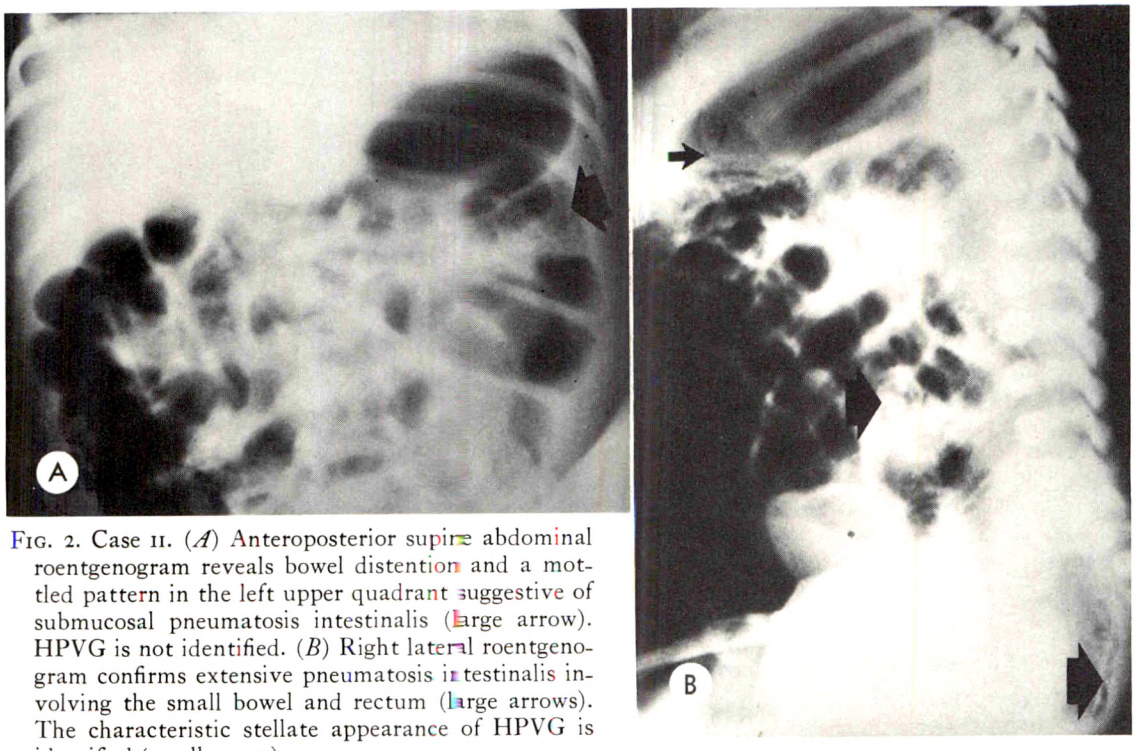


Fig. 2. Case II. (*A*) Anteroposterior supine abdominal roentgenogram reveals bowel distention and a mottled pattern in the left upper quadrant suggestive of submucosal pneumatosis intestinalis (large arrow). HPVG is not identified. (*B*) Right lateral roentgenogram confirms extensive pneumatosis intestinalis involving the small bowel and rectum (large arrows). The characteristic stellate appearance of HPVG is identified (small arrow).

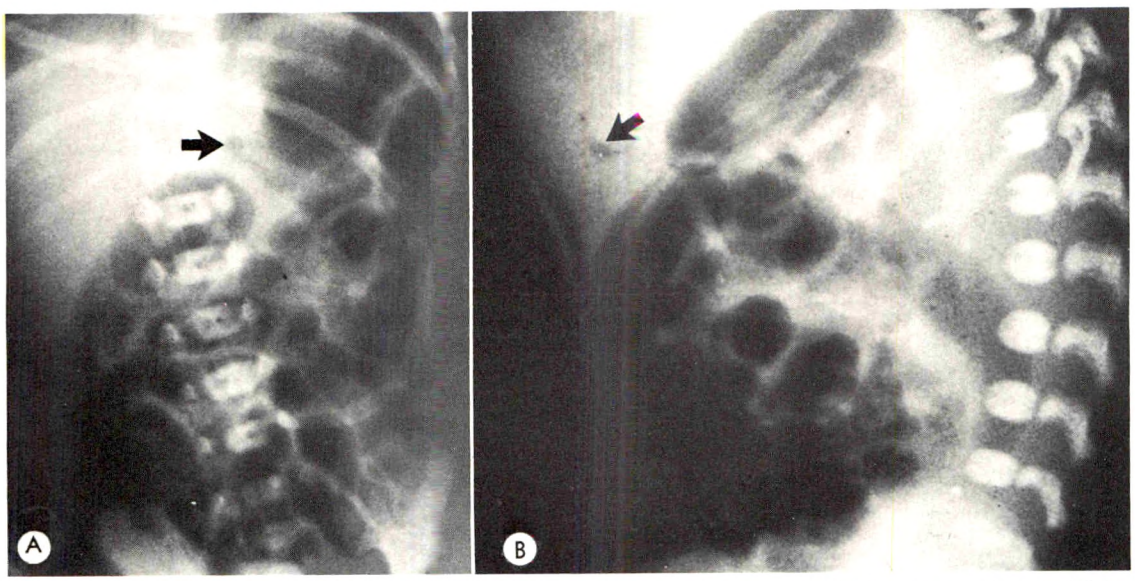


Fig. 3. Case III. (*A*) Anteroposterior abdominal roentgenogram shows an umbilical venous catheter with its tip overlying the hepatic shadow. A rounded lucency (arrow) is noted just medial to the gastric air bubble. (*B*) Right lateral roentgenogram of the abdomen confirms that the tip of the umbilical venous catheter is in the region of the ductus venosus. The stellate lucency due to HPVG is seen anterior to the stomach (arrow).

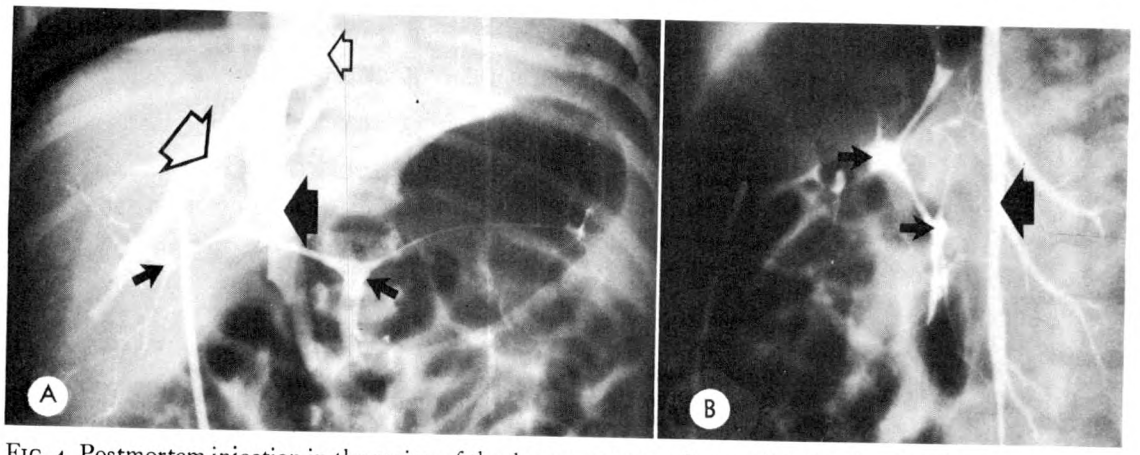


FIG. 4. Postmortem injection in the region of the ductus venosus of 1 cc. of meglumine diatrizoate through an umbilical venous catheter. (*A*) Anteroposterior abdominal roentgenogram demonstrates contrast medium within hepatic portal venous system (small arrows), hepatic vein (large open arrow), inferior vena cava (large arrow), and right atrium (small open arrow). Note the branching pattern of the portal venous system as well as the transverse position of the liver and prominent left hepatic lobe. (*B*) Cross-table lateral roentgenogram demonstrates the "stellate" appearance of contrast medium within hepatic portal veins (small arrows). Contrast material is again noted in the inferior vena cava (large arrow).

least every 12 hours should be obtained and correlated with continuous clinical evaluation.[17] Anteroposterior supine and cross-table lateral roentgenograms of the abdomen are suggested for these follow-up examinations.

The roentgenologic appearance of HPVG on the lateral abdominal roentgenogram has a fairly characteristic appearance (Fig. 1–3). A "stellate" lucency is noted in the liver. Postmortem injection of contrast material into the neonatal portal venous system demonstrates this same appearance (Fig. 4, *A* and *B*). The stellate appearance is believed to be due to contrast material seen end-on in large hepatic portal vein branches with the surrounding and radiating linear densities due to smaller peripheral branches.

Gas within the biliary tract of neonates is extremely rare.[10] A similar stellate appearance could theoretically be present on the lateral abdominal roentgenogram in patients with this entity. However, caudal flow of bile toward the duodenum should cause biliary tract gas to localize near the porta hepatis inferiorly. Experimental injections of the neonatal biliary tract and portal venous system should permit further anatomic clarification.

There are numerous causes of HPVG[4,5,7,9,11,14–16,19,22] (Table 1). Necrotizing enterocolitis and umbilical venous catheter malposition are the primary differential diagnostic considerations in the neonate. Many of the other listed causes of HPVG have

TABLE I

CAUSES OF HEPATIC PORTAL VENOUS GAS (HPVG)

Common
Ischemic bowel disease—adult
Neonatal necrotizing enterocolitis
Umbilical venous catheter malposition
Rare
Adynamic ileus
Barium enema examination in patient with Hirschsprung's disease or ulcerative colitis
Corrosive ingestion
Diarrhea and pneumatosis intestinalis
Carbohydrate intolerance
Gastroenteritis
Gastric distention
Gastric ulcer eroding mesenteric vein
Hydrogen peroxide enema examination
Jugular vein catheterization
Mechanical bowel obstruction with intestinal necrosis
Sepsis
Umbilical vein portography

been described in adults and are simply theoretic considerations in the newborn infant.

HPVG is identified in 30 per cent of neonates with necrotizing enterocolitis.[1] Since the original description by Berdon and coworkers,[2] the clinical recognition and pathologic incidence of this entity have increased.[1,8,13,14,20,21] Current indications for surgery are: increasing signs of sepsis; clinical evidence of peritonitis; and pneumoperitoneum.[17] Leonidas *et al.*[12] have stressed that roentgenographic evidence of free intraperitoneal fluid in infants with necrotizing enterocolitis indicates peritonitis with possible perforation. They feel that these patients should have surgery as soon as possible. The presence of portal vein gas in neonatal necrotizing enterocolitis can also be correlated with poor prognosis.[1,12,20,21,23] Although the presence of HPVG has not been used as an indication for surgery, its presence should warrant even closer clinical and roentgenologic evaluation. Further delineation of strict clinical and roentgenographic criteria for early surgical intervention in neonatal necrotizing enterocolitis is imperative.

SUMMARY

The roentgenologic appearance of neonatal hepatic portal venous gas (HPVG) on the lateral abdominal roentgenogram is described. The value of this view for the recognition of HPVG is discussed. A differential diagnosis of HPVG is presented.

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REFERENCES

1. Bell, R. S., Graham, C. B., and Stevenson, J. K. Roentgenologic and clinical manifestations of neonatal necrotizing enterocolitis: experience with 43 cases. Am. J. Roentgeno Rad. Therapy & Nuclear Med., 1971, *1*. 123–134.
2. Berdon, W. E., Grossman, H., Baker, D. I Mizrahi, A., Barlow, O., and Blanc, W. Necrotizing enterocolitis in premature infa *Radiology*, 1964, *83*, 879–887.
3. Blanc, W. A., Berdon, W. E., Baker, D. I and Wigger, H. J. Calcified portal v thromboemboli in newborn and stillbc infants. *Radiology*, 1967, *88*, 287–292.
4. Dell, J. M., Jr. Gas in portal vein. Am. Roentgenol., Rad. Therapy & Nucle Med., 1967, *100*, 424–425.
5. Fink, D. W., and Boyden, F. M. Gas in por veins: report of two cases due to ingestion corrosive substances. *Radiology*, 1966, *87*, 74 743.
6. Frates, R. E. Incompetence of sphincter Oddi in newborn. *Radiology*, 1965, *85*, 87 879.
7. Fred, H. W., Mayhall, C. G., and Harle, T. Hepatic portal venous gas: review and rep on six new cases. *Am. J. Med.*, 1968, *44*, 55 565.
8. Hopkins, G. B., Gould, V. E., Stevensc J. K., and Oliver, T. K., Jr. Necrotizi enterocolitis in premature infants: clinical a pathologic evaluation of autopsy materi *A.M.A. Am. J. Dis. Child.*, 1970, *120*, 22 232.
9. Kees, C. J., and Hester, C. L., Jr. Portal ve gas following barium enema examinatio *Radiology*, 1972, *102*, 525–526.
10. Kirks, D. R., and Baden, M. Incompetence sphincter of Oddi associated with duoden stenosis. *J. Pediat.*, 1973, *83*, 838–840.
11. Lazar, H. P. Survival following portal venous a embolization: report of case. *Am. J. Dige Dis.*, 1965, *10*, 259–264.
12. Leonidas, J. C., Krasna, I. H., Fox, H. A and Broder, M. S. Peritoneal fluid in necr tizing enterocolitis: radiologic sign of clinic deterioration. *J. Pediat.*, 1973, *82*, 672–675.
13. Mizrahi, A., Barlow, O., Berdon, W., Blanc W. A., and Silverman, W. A. Necrotizin enterocolitis in premature infants. *J. Pedia* 1965, *66*, 697–706.
14. Pochaczevsky, R., and Kassner, E. G. Necr tizing enterocolitis of infancy. Am. J. Roen genol., Rad. Therapy & Nuclear Med 1971, *113*, 283–296.
15. Rigler, L. G., and Pogue, W. L. Roentgen sign of intestinal necrosis. Am. J. Roentgenol. Rad. Therapy & Nuclear Med., 1965, *94* 402–409.
16. Sisk, P. B. Gas in portal venous system. *Radiology*, 1961, *77*, 103–107.
17. Stevenson, J. K., Oliver, T. K., Jr., Graham, C. B., Bell, R. S., and Gould, V. E. Aggres-

sive treatment of neonatal necrotizing enterocolitis: 38 patients with 25 survivors. *J. Pediat. Surg.*, 1971, *6*, 28–35.
18. SUSMAN, N., and SENTURIA, H. R. Gas embolization of portal venous system. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 847–850.
19. SWAIM, T. J., and GERALD, B. Hepatic portal venous gas in infants without subsequent death. *Radiology*, 1970, *94*, 343–345.
20. TOULOUKIAN, R. J., BERDON, W. E., AMOURY, R. A., and SANTULLI, T. V. Surgical experience with necrotizing enterocolitis in infant. *J. Pediat. Surg.*, 1967, *2*, 389–401.
21. WILSON, S. E., and WOOLLEY, M. M. Primary necrotizing enterocolitis in infants. *A.M.A. Arch. Surg.*, 1969, *99*, 563–566.
22. WIOT, J. F., and FELSON, B. Gas in portal venous system. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 920–929.
23. WOLFE, J. N., and EVANS, W. A. Gas in portal veins of liver in infants: roentgenographic demonstration with postmortem anatomical correlation. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1955, *74*, 486–489.

Vol. 122, No. 1

A NEW AND UNUSUAL ROENTGENOGRAPHIC FINDING OF FATTY LIVER IN INFANTS*

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TWO patients with the somewhat unusual and surprising plain film roentgenographic finding of "radiolucent liver," secondary to extensive fatty replacement, have recently come to our attention. The fact that "fatty liver" was determined roentgenographically, before any substantial clinical work-up or histologic examination of the liver was available, prompted us to report this interesting and uncommon roentgenographic manifestation of advanced fatty degeneration of the liver.

REPORT OF CASES

CASE 1. L.G. This 3 month old Latin American girl was admitted to the John Sealy Hospital for evaluation of hepatomegaly. She was delivered as a normal 8 pound, 2 ounce baby of a normal 32 year old woman. The only significant gestational history centered around the fact that the mother had inhalation anesthesia (at 3 months' gestation) for attempted corneal transplant. Otherwise, the pregnancy was uncomplicated and the immediate postnatal history unremarkable.

The baby, after discharge from hospital, was fed similac at home, 2 to 4 ounces, 6 times daily to a total of 21 ounces per day. During the third month rice and oat cereal were introduced into the diet (twice a day), but the infant was never fed vegetables, meat, or eggs. Two weeks prior to the admission to our hospital, this infant was hospitalized in an outlying hospital for vomiting and watery stools. At that time she was lethargic, seemed to "sleep too much," and was eventually diagnosed as having a viral infection. However, recovery was incomplete, the infant remained lethargic, and her abdomen remained distended. The family history was unremarkable, and specifically there was no history of liver disease. Five other siblings were in good health.

Physical examination on this admission showed the baby to be in no acute distress, but she was observed to be hypotonic and possibly retarded in terms of motor development. Apart from this, neurologic examination was normal. Abdominal examination showed the liver to be enlarged and to extend 9 cm. below the right costal margin. It was hard and smooth. The spleen was also palpable, just under the left costal margin, but was not grossly enlarged.

Laboratory investigation showed a blood urea nitrogen of 35 mg. per cent, calcium 8.5 mEq./l., phosphorus 2.8 mEq./l., SGPT 185 mU/ml., SGOT 140 mU/ml., LDH 485 mU/ml., and alkaline phosphatase 285 mU/ml. All of these latter 4 determinations were abnormally elevated. Hemoglobin and white blood cell count differential were normal, and a VDRL was nonreactive. Prothrombin time was 100 per cent of normal, while the cerebrospinal fluid protein was 170 mg. per cent. Serum cholesterol was 243 mg. per cent, while triglycerides were in excess of 600 mg. per cent. No lipoprotein was present on multiple serum protein electrophoreses, but pre-beta lipoprotein appeared elevated. Total protein was 5.8 gm. per cent, while serum albumin was 3.4 gm. per cent. Total bilirubin was 1.0 mg. per cent.

Chest and abdominal roentgenograms, obtained before any of the laboratory values were available, and before any definite clinical formulation was made, showed an unusually radiolucent, enlarged liver (Fig. 1, *A–C*). This finding suggested the presence of excess fat in the liver and the diagnosis of fatty liver was proposed. No calcifications were seen and apart from this finding, no abnormality was demonstrated in the abdomen or chest. Subsequent to this observation a liver biopsy was obtained and this demonstrated severe fatty metamorphosis of the liver (Fig. 2). A bone marrow aspiration was also obtained and showed peculiar vacuolated, fat containing cells and vacuolated erythroid and myeloid precursors. Renal and rectal biopsies were normal and the final pathologic diagnosis was fatty metamorphosis of the liver.

The infant's diet was adjusted to a more normal regimen, but before any significant

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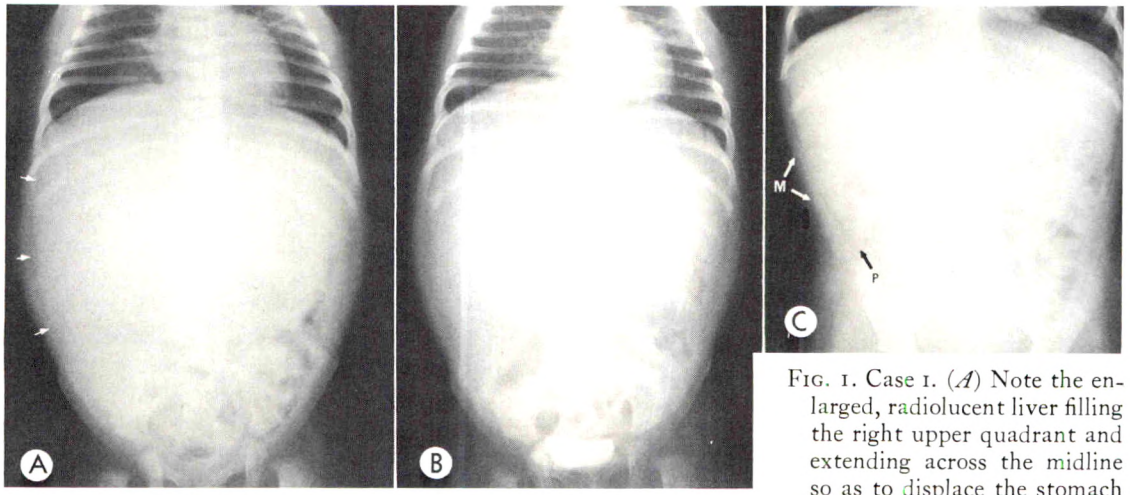


Fig. 1. Case 1. (*A*) Note the enlarged, radiolucent liver filling the right upper quadrant and extending across the midline so as to displace the stomach and intestines to the left and downward. The right liver edge (arrows) extends to the abdominal muscles. The muscles are more opaque than the fatty liver, whereas normally they are of equal density. (*B*) Roentgenogram obtained during intravenous pyelography enhances the radiolucent appearance of the liver, probably by a minimal residual total body opacification effect. (*C*) Normal liver for comparison. Note the normal degree of opacity of the liver which is separated from the equally opaque abdominal muscles (M) by the normally radiolucent properitoneal fat stripe (P). In *A* and *B* the fat stripe is not visualized.

follow-up could be obtained, the infant's parents removed the infant from hospital care and took her home. She died within 2 weeks, but unfortunately postmortem examination was not permitted. However, it was postulated that the infant suffered from extensive fatty metamorphosis of the liver either from; (1) accidental ingestion of a toxin, unknown to the parents and physicians in charge, or (2) from a complication of the incompletely documented viral infection. The possibility that the mother's general anesthesia at 3 months' gestation had produced these liver changes seemed to be a very remote etiologic possibility. Indeed, it seemed doubtful that this event had anything to do with the infant's liver disease.

Case II. B.M. This baby boy was born on September 26, 1972, a normal product of a spontaneous cephalic vaginal delivery. Birth weight was 7 pounds, 2½ ounces and the infant was discharged as a normal infant on a regular diet. He was readmitted March 22, 1973, at the age of 6 months, with the presenting complaint of "yellow eyes" for 1 week. Three months prior to this admission the infant had episodes of projectile vomiting after feeding. Because of this problem, he was placed, by his parents, on a diet of "kool-aid," bean soup, and rice gruel. In essence there was no animal protein or milk intake since December, 1972. Prior to this date he had been receiving regular feedings.

On arrival to our hospital the infant was noted to be extremely edematous and jaundiced. Ecchymotic areas were present over the entire body and scalp. The liver was enlarged and palpable to approximately 6 cm. below the right costal margin. It was smooth and firm.

Roentgenograms of the chest and abdomen were obtained and demonstrated a large, abnormally and strikingly radiolucent liver (Fig. 3). In addition, the skeleton was extremely demineralized and numerous pathologic fractures

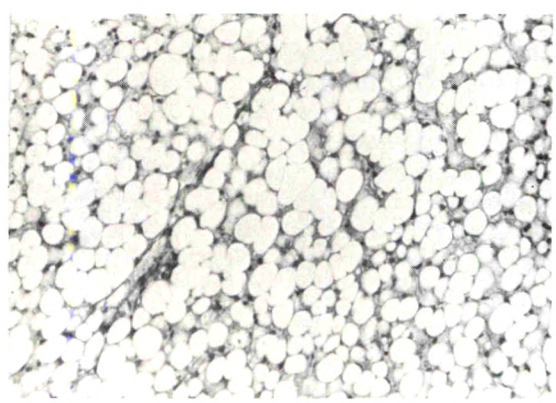


Fig. 2. Case 1. Biopsy material showing extensive fatty metamorphosis of the liver. Note the large lipid-containing cells, and virtually no normal liver parenchyma.

throughout the metaphyses were present (Fig. 4). The soft tissues were markedly edematous. On the basis of the unusual radiolucency of the enlarged liver, the diagnosis of fatty liver was suggested. The bone and soft tissue changes were believed secondary to severe malnutrition.

Laboratory data revealed a total serum bilirubin of 14.8 mg. per cent (11.4 mg. per cent direct). Prothrombin time was 19.9 seconds (control 11 seconds), while serum electrolytes were normal. Alkaline phosphatase was 144 mU/ml., SGOT 293 mU/ml., and SGPT 71 mU/ml. Hemoglobin was 8.4 gm. per cent, while the white blood cell count was 36,100. Repeat prothrombin time rose to 30.8 seconds (control 12.9 seconds), and serum protein determination showed a total serum protein of 3.5 gm. per cent and a serum albumin of 1.7 gm. per cent. Serum calcium was 5.7 mEq./l. and serum phosphate 9.2 mEq./l. In spite of peritoneal dialysis for a profound acid-base imbalance, the infant died shortly after admission.

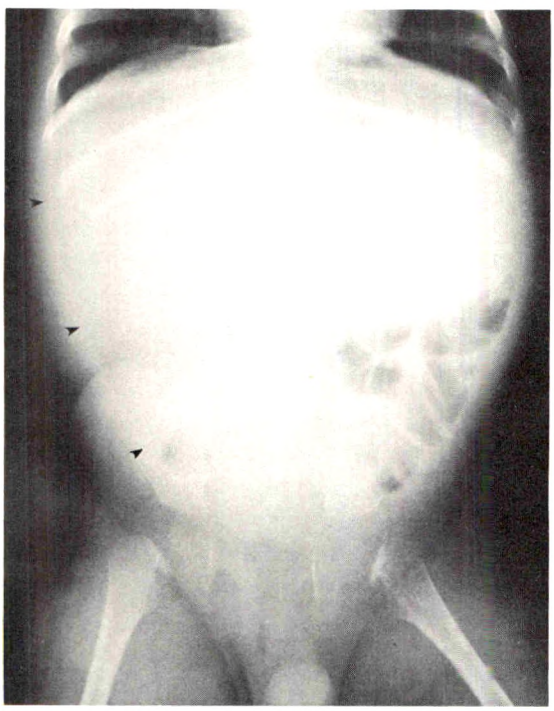


Fig. 3. Case 11. Note the enlarged, strikingly radiolucent liver which displaces the intestines down and to the left. The left lobe of the liver is also radiolucent and enlarged and occupies the left upper quadrant. Note that the right edge of the liver (arrows) extends to the adjacent abdominal muscles. The muscles appear more dense than the liver. Normally they are of equal density and are usually separated from the liver by the normally radiolucent, but thin properitoneal fat stripe (see Fig. 1*C*). Also note that the bones in the infant are markedly demineralized and that there are numerous metaphyseal pathologic fractures.



Fig. 4. Case 11. The long bones show extensive demineralization and changes not unlike scurvy, including a Wimberger's ring. Scurvy was not biochemically documented in this infant, and the changes were believed secondary to the severe degree of liver disease. Also note extensive soft tissue edema and an indwelling catheter in the soft tissues of the right lower leg.

Postmortem examination revealed the liver to be totally replaced by fat (Fig. 5*A*). It had the texture and appearance of butter when cut with a knife and weighed 585 gm. (normal 200 gm.). No other abnormalities of the abdominal viscera were noted and on histologic examination the liver hepatocytes were completely replaced by fat (Fig. 5*B*). Many of the cells were of the signet ring variety and most nuclei were pyknotic. No fibrosis or infiltration was seen. Final diagnosis was extreme fatty metamorphosis of the liver.

On the basis of the inadequate diet fed to this infant, and the striking postmortem appearance of the liver, it was determined that the most likely diagnosis was severe malnutrition with associated kwashiorkor.

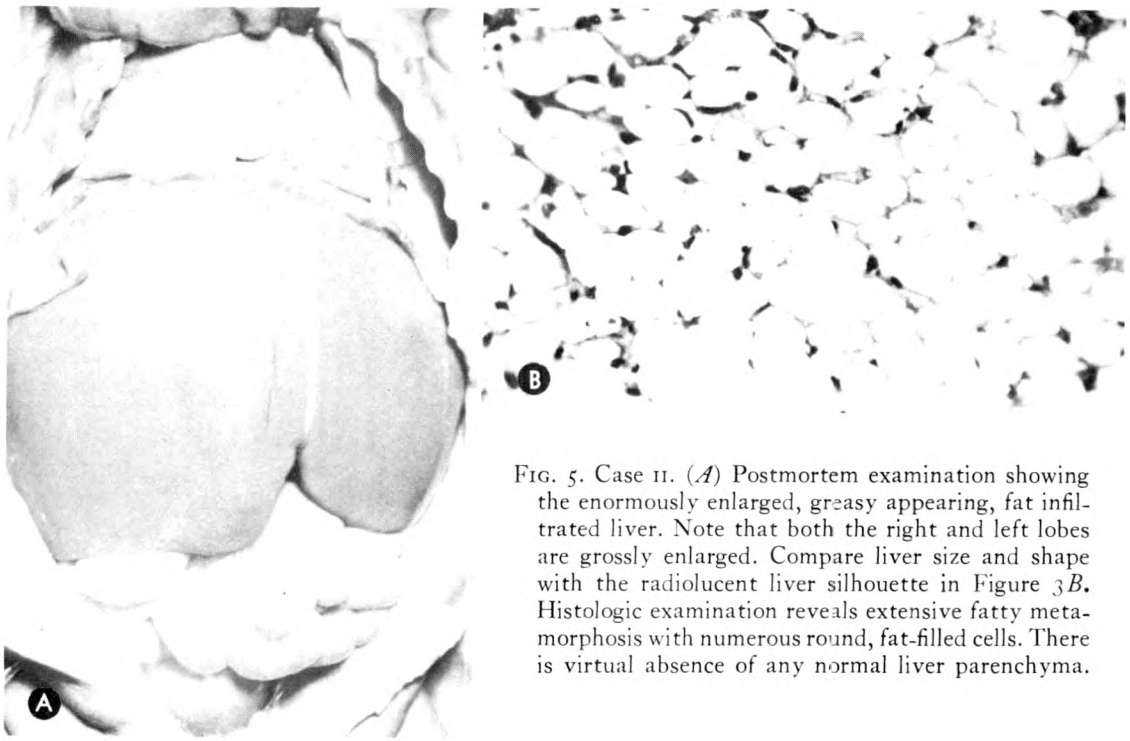


FIG. 5. Case II. (*A*) Postmortem examination showing the enormously enlarged, greasy appearing, fat infiltrated liver. Note that both the right and left lobes are grossly enlarged. Compare liver size and shape with the radiolucent liver silhouette in Figure 3*B*. Histologic examination reveals extensive fatty metamorphosis with numerous round, fat-filled cells. There is virtual absence of any normal liver parenchyma.

DISCUSSION AND CONCLUSIONS

Fatty replacement of the liver can occur with a number of conditions. These include infectious hepatitis, viral hepatitis in the newborn,[12] toxic hepatitis, profound malnutrition with protein deficiency in kwashiorkor,[3,4,6,9] encephalopathy and fatty degeneration of the viscera or Reye's syndrome,[2,8] a number of lipid storage diseases, and galactosemia.[10] However, in most such cases the diagnosis of fatty liver is made on the basis of biochemical findings and/or liver biopsy. In our experience, it is unusual to diagnose the condition from abdominal roentgenograms, and yet in our 2 cases the enlarged liver silhouette was so radiolucent (characteristic fatty density) that one had no choice but to suggest the diagnosis of "fatty liver."

Both of our infants died shortly after their condition was diagnosed and clearly were in the advanced stages of liver disease with extreme fatty metamorphosis. The first infant died of undetermined causes, but the serious degree of liver abnormality was presumed to be primarily contributory to the infant's demise. It was interesting, however, that even though the liver was almost entirely replaced by fat, the patient was not jaundiced. This was not true of our second infant who was clearly jaundiced at the time of admission. This infant was finally shown to be suffering from severe malnutrition with no protein in the diet and resultant kwashiorkor. In the first patient the exact cause of extensive fatty metamorphosis of the liver was not determined, although infectious (viral) hepatitis or toxic hepatitis was considered.

It has been known for many years that lipomatous tissue, when present in sufficient quantities, casts a characteristic radiolucent density on the roentgenogram. The degree of radiolucency is less than that seen with air, but still marked enough so that fatty tissue is easily distinguished from the other soft tissues. This phenomenon occurs in various portions of the body, some as normal manifestations (*i.e.*, properitoneal fat stripes, the fat line around normal kidneys, normal fat in the soft tissues of the extremities, etc.), and some

with lipomatous tumors (*i.e.*, soft tissue lipomas, lipoma of the corpus callosum, teratoma, etc.). However, the roentgenographic documentation of advanced fatty replacement of the liver, by virtue of an unusually radiolucent appearance to the enlarged liver silhouette has not, to our knowledge, been previously documented, even in kwashiorkor. Even if its documentation has eluded us in our survey of the literature, it surely is not a well known roentgenographic finding. On the other hand, it is also quite possible that since abdominal roentgenograms are not routinely obtained in many of these cases, the finding has been overlooked.

It is of interest that both our infants died shortly after the finding of radiolucent liver became apparent and clearly these infants were in the end stages of severe liver disease. Certainly it would seem that almost total replacement of the liver must be present before the roentgenograms become positive for the finding of "radiolucent liver."

The marked degree of bone demineralization, and metaphyseal fragmentation (pathologic fracture), is an interesting additional finding in our second infant. Such bone change has been noted with severe liver disease, especially liver tumor,[7,11–13] and cortical and trabecular thinning has been specifically noted with kwashiorkor.[1] In such cases bone changes are presumed secondary to protein deficiency.[2] Other bone changes noted with kwashiorkor include a delay in bone maturation.[1] Alimentary tract changes seen with kwashiorkor include a roentgenographically abnormal small bowel pattern, somewhat similar to that seen with gluten enteropathy.[5]

SUMMARY

A new roentgenographic sign, the "radiolucent liver," secondary to extensive fatty degeneration of the liver is presented in 2 infants.

In 1 infant the cause of liver damage was not completely ascertained (although infectious or toxic hepatitis was postulated) while kwashiorkor was the problem in our second infant.

The radiolucent liver sign is quite striking roentgenographically and is probably specific for extensive fatty replacement of the liver.

Both our infants died shortly after the roentgenographic and clinical findings were documented, and although it is not believed that one can unequivocally attach prognostic significance to this sign, it does seem to be associated with very extensive end stage fatty replacement of the liver, probably well beyond significant recovery.

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REFERENCES

1. Adams, P., and Berridge, F. R. Effects of kwashiorkor on cortical and trabecular bone. *Arch. Dis. Childhood*, 1969, *44*, 705–709.
2. Bourgeois, C., Olson, L., Comer, D., Evans, H., Keschamras, N., Cotton, R., Grossman, R., and Smith, T. Encephalopathy and fatty degeneration of viscera: clinicopathologic analysis of 40 cases. *Am. J. Clin. Path.*, 1971, *56*, 558–571.
3. Chastel, C., Brottes, H., Goasguen, J., LeMao, G., Favre, C., Thomas, J., and Sagnet, H. États de malnutrition protidique et de dénutrition des enfants biafrais: Étude anatomo-pathologique. *Bull. Soc. Path. Exot.*, 1969, *6*, 941–955.
4. Chaudhuri, A. D., Bhattacharyya, A. K., and Mukherjee, A. M. Liver in pre-kwashiorkor and kwashiorkor-marasmus syndromes. *Tr. Roy. Soc. Trop. Med. & Hyg.*, 1972, *66*, 258–262.
5. Kowalski, R. Roentgenologic studies of alimentary tract in kwashiorkor. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 100–112.
6. Kumar, V., Deo, M. G., and Ramalingaswami, V. Mechanism of fatty liver in protein deficiency: experimental study in rhesus monkey. *Gastroenterology*, 1972, *62*, 445–451.
7. McDonald, P. Hepatic tumors in childhood. *Clin. Radiol.*, 1967, *18*, 74–82.

8. REYE, R. D., MORGAN, G., and BARAL, J. Encephalopathy and fatty degeneration of viscera: disease entity in childhood. *Lancet*, 1963, *2*, 749–752.
9. THOMAS, J., SAGNET, H., CHASTEL, C., and LEVOURCH, C. Le kwashiorkor de famine (ou de guerre): réflexions à propos de 1900 observations. *Presse méd.*, 1971, *79*, 1681–1684.
10. SCHAFFER, A. J., and AVERY, M. E. Diseases of the Newborn. Third edition. W. B. Saunders Company, Philadelphia, 1971, p. 826.
11. SORSDAHL, O. A., and GAY, B. B., JR. Roentgenologic features of primary carcinoma of liver in infants and children AM J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 117–127.
12. SWISCHUK, L. E. Radiology of Newborn and Young Infants. Williams & Wilkins Company, Baltimore, 1973, pp. 327–328.
13. TENG, C. T., DAESCHNER, C. W., JR., SINGLETON, E. B., ROSENBERG, H. S., COLE, V. W., HILL, L. L., and BRENNAN, J. C. Liver diseases in osteoporosis in children: clinical observations. *J. Pediat.*, 1961, *59*, 684–702.

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VALUE OF ARTERIOGRAPHY BEFORE CLOSURE OF ARTERIOVENOUS FISTULAE PERFORMED FOR LOWER LIMB DISCREPANCY*

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JANES and Musgrove in 1950[2] were first to report the use of surgical arteriovenous fistulae in the correction of congenital short limbs in children. Since then there have been other communications on the value of the operation.[1,3,4] Recently, Kinmonth and Negus[5] have reviewed their experience of 10 patients followed for 12 years.

This paper describes in detail the findings on arteriography in 8 of these patients and discusses the value of arteriography before operative closure of the fistulae.

MATERIAL AND METHOD

Of the 8 patients, 6 had congenital short limbs, 1 had had osteomyelitis of the femur which destroyed the lower femoral and upper tibial epiphyses, and 1 poliomyelitis.

All the angiograms were obtained by translumbar aortography using a standard technique.[6] The contrast medium agent, meglumine iothalamate 60 per cent, 1.5 ml./kg. body weight, was injected and films were exposed at the rate of 2/sec. using an AOT serial film changer with a programmed moving table top. The arteries from the lower abdominal aorta to the ankles were included in the films.

RESULTS

The angiographic findings are summarized in Table 1.

DISCUSSION

Translumbar aortography was performed in these patients in preference to bilateral femoral arteriography to show the lower abdominal aorta and the arterial supply to both lower limbs, so that a comparison of the size of the main arteries could be made. Arterial catheterization of the lower abdominal aorta was not employed in order to avoid possible trauma to the normal femoral artery. In addition, translumbar aortography can be rapidly performed, an important factor when examining patients liable to high output cardiac failure, an occasional complication of the arteriovenous fistula.

Using a rapid series of films, the site and size of the arteriovenous fistula were demonstrated in all cases. In addition, the presence of any arterial collaterals and enlarged venous tributaries in the region of the fistula which could represent an operative hazard was also shown. The angiographic findings were subsequently confirmed in those patients who came to surgery.

The mechanism by which surgical arteriovenous fistula formation stimulates limb growth is not understood. In none of the arteriograms of our patients were the arteries below the fistula increased in size, although in 2 patients there were arterial collaterals in the region of the fistula. Although the size of arteries is not a direct index of increased flow, it is related to it and these arteriographic findings suggest that increased blood flow is not the primary factor in producing limb growth. This is in agreement with Kinmonth and Negus' findings[5] of the blood flow measured by plethysmography in which it was usually less in the limb with the fistula than in the normal limb. These authors postulate that there is an increased turnover rate of tissue

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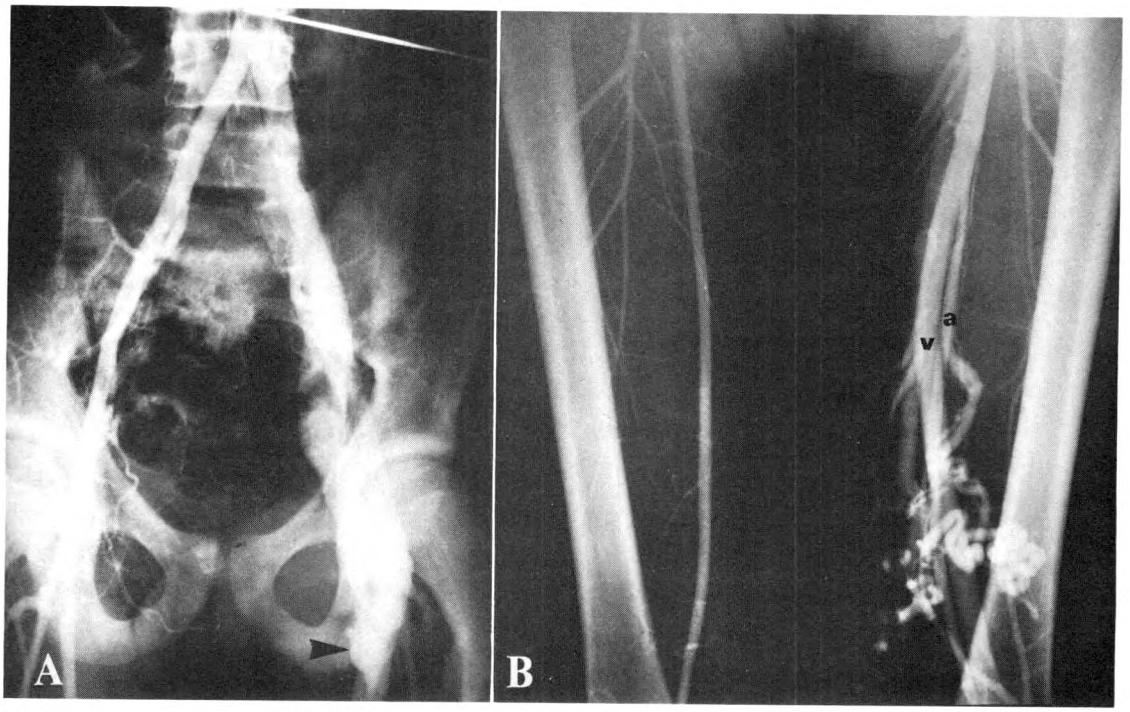


FIG. 1. Case 1. (*A*) Note site of fistula between left long saphenous vein and the superficial femoral artery (arrow). (*B*) The femoral arteries (a) are of equal size. The left femoral vein is considerably enlarged (v). There are enlarged tributaries in the adductor canal.

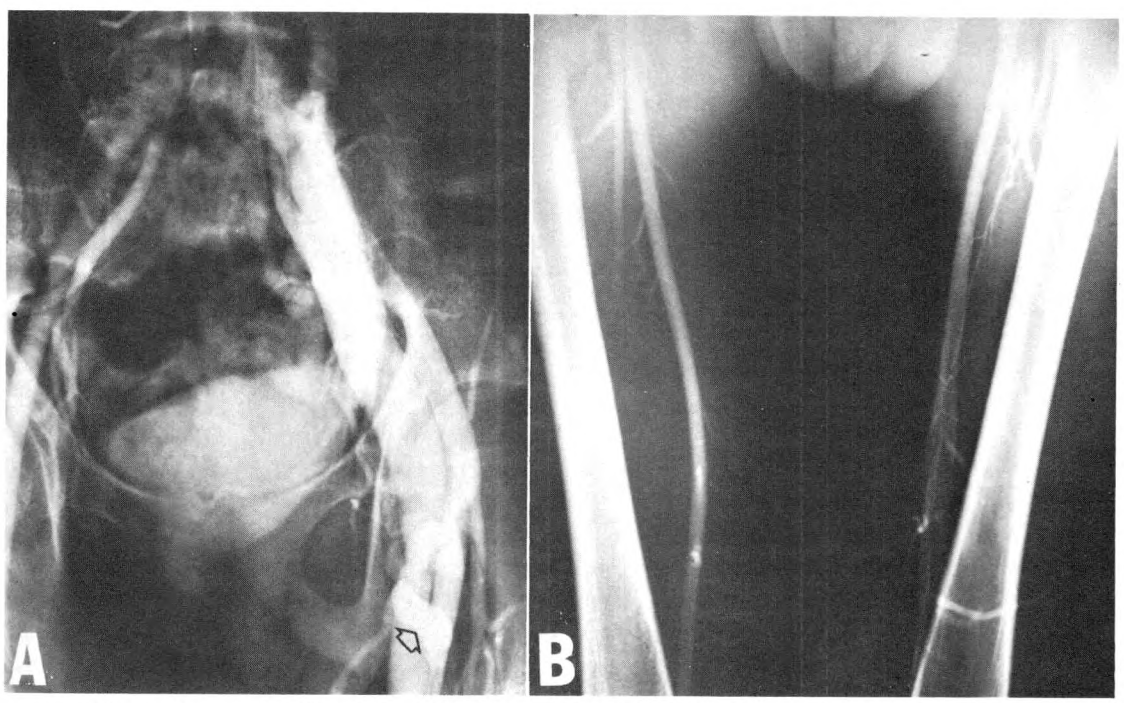


FIG. 2. Case 2. (*A*) Note site of fistula (arrow). (*B*) The femoral arteries are of equal size.

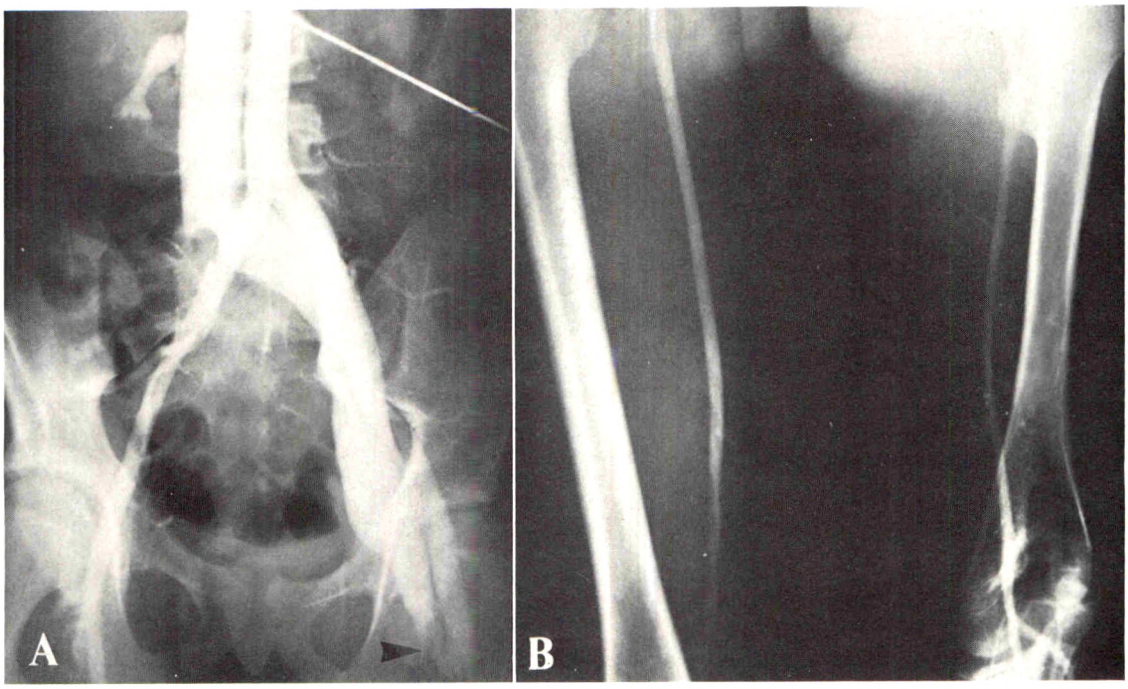
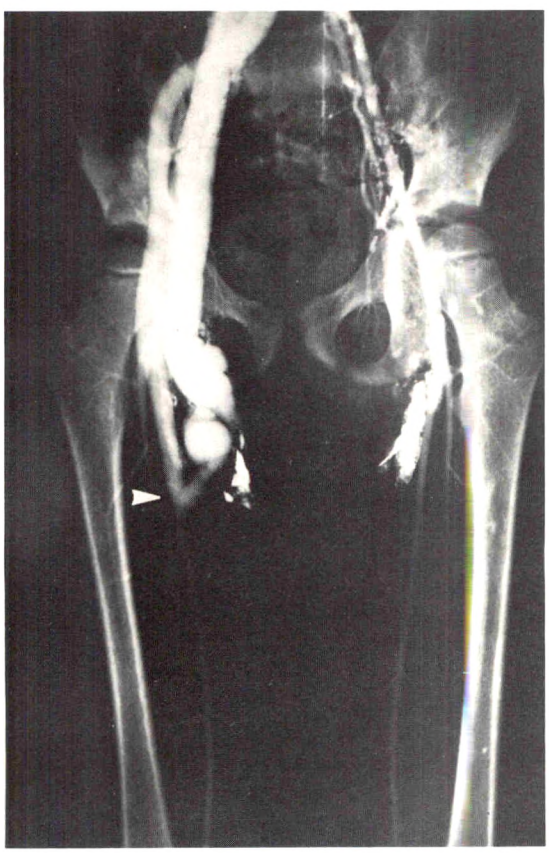


Fig. 3. Case 3. (*A*) Note site of fistula (arrow). (*B*) The left femoral artery is smaller than the right.



fluid and lymph flow and found a slight increase in the size of the lymphatic pathways in the operated limb.

Whatever the exact mechanism, the creation of a surgical arteriovenous fistula in patients with congenitally short limbs appears to be of value. Thus, in the 8 patients reported here, there was no increase in limb discrepancy in 3 patients, and in 3 others the limb discrepancy was only slight. In the patient in whom the limb discrepancy remained unchanged (Case 3), the patient had had osteomyelitis destroying the epiphyses. In the other patient (Case 8) the length of the limb before the fistula was created was not known.

Once the epiphyses have fused, no further bone growth can occur and as the fistulae gradually increase in size this may eventually lead to high output cardiac failure. For these reasons, once maximal growth has been achieved, the fistula is closed. To

Fig. 4. Case 4. Note site of fistula (arrow). The femoral arteries are of equal size

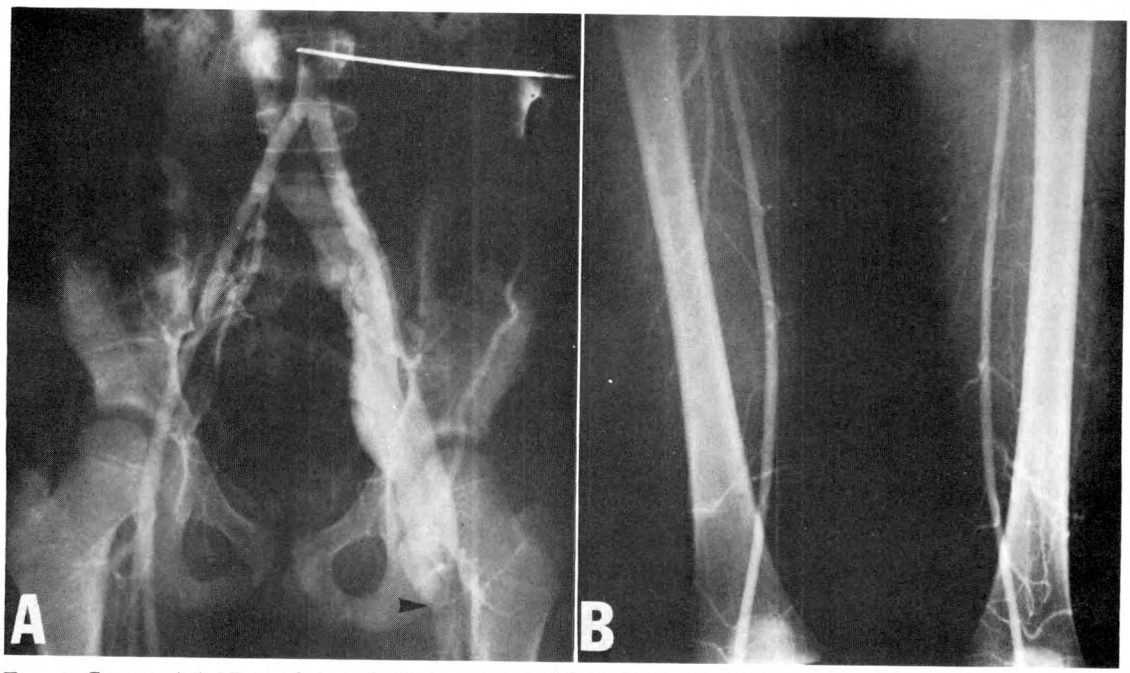


FIG. 5. Case 5. (*A*) Note of site of fistula (arrow). The left superficial circumflex iliac artery is enlarged. (*B*) The femoral arteries are of equal size.

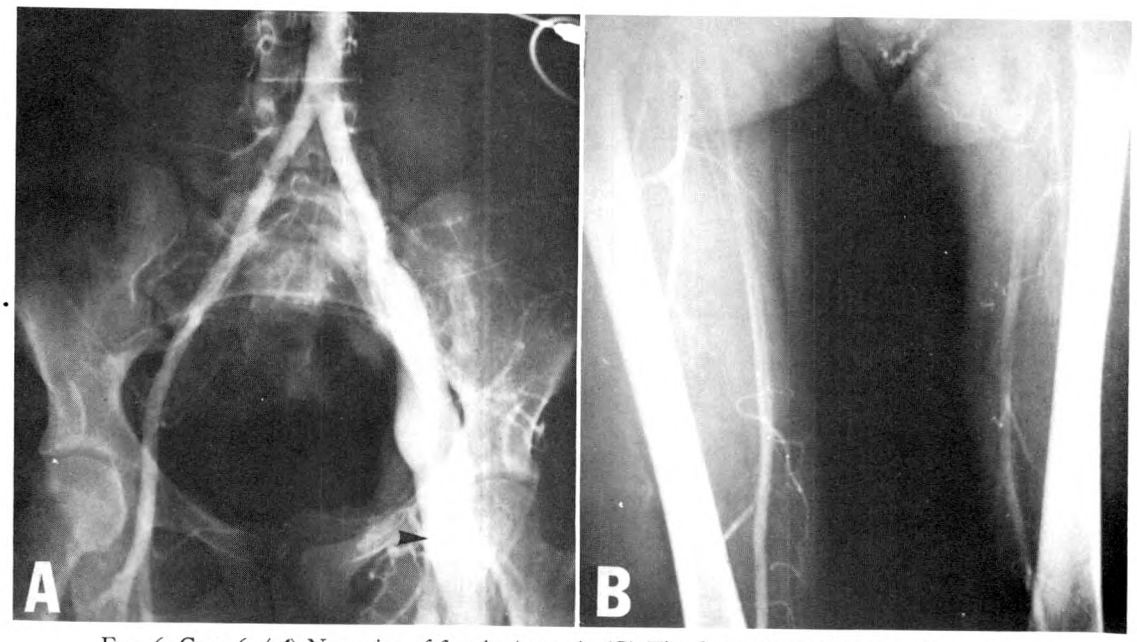


FIG. 6. Case 6. (*A*) Note site of fistula (arrow). (*B*) The femoral arteries are of equal size.

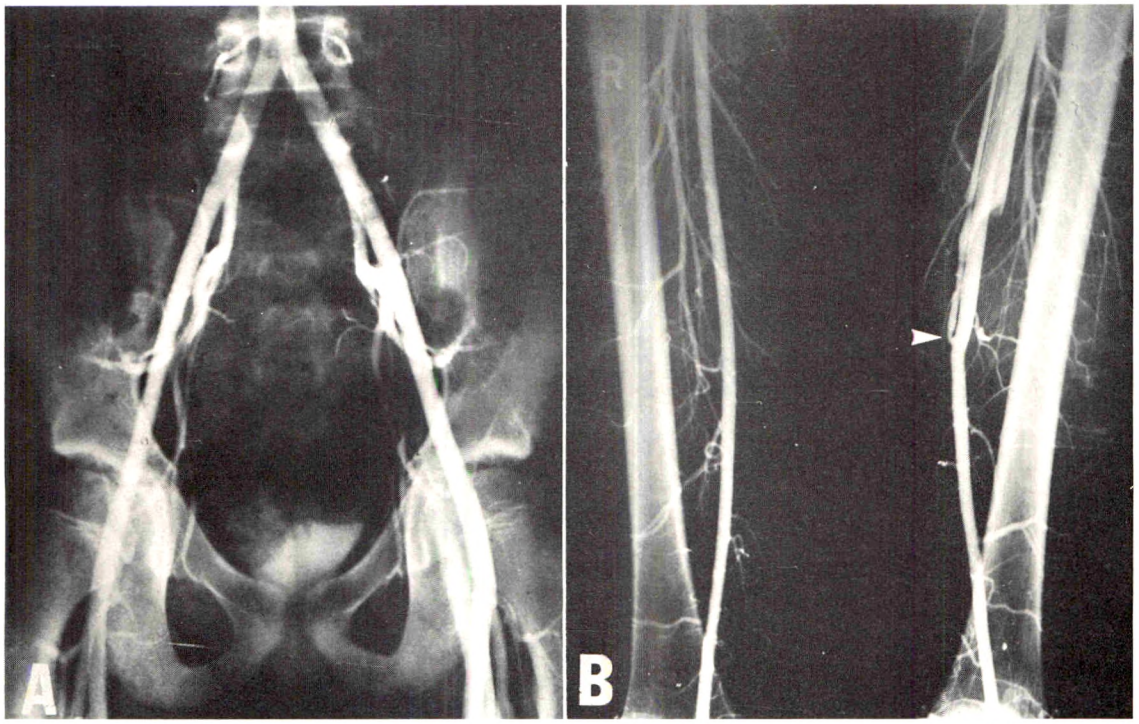


Fig. 7. Case 7. (*A*) The arteries on the left proximal to the fistula are slightly enlarged. (*B*) Note site of fistula (arrow). The femoral arteries below the fistula are of equal size.

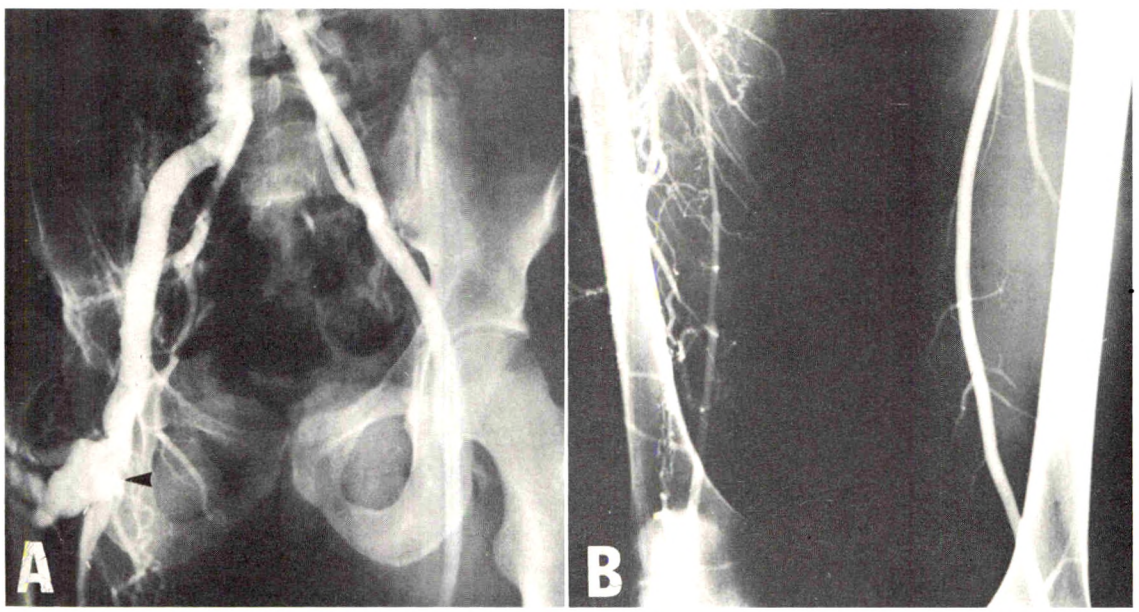


Fig. 8. Case 8. (*A*) Note site of fistula on right between the profunda artery and vein (arrow). (*B*) The femoral artery on the right side is smaller than on the left.

TABLE I

ANGIOGRAPHIC FINDINGS

Patient	Site of Fistula	Size of Arteries Proximal to Fistula Compared with Other Side	Size of Veins Proximal to Fistula Compared with Other Side	Size of Artery below Compared with Other Side	Arterial Collaterals	Venous Tributaries	Value of Fistula	Age at which Fistula Was Created	Age at which Fistula Was Closed	Figures
1. (V.H.)	Below left saphenous opening	Enlarged	Enlarged	Equal	None	Superficial femoral vein greatly enlarged. Tributaries in adductor canal	No increase in limb discrepancy	13 years	15 years	1, *A* and *B*
2. (S.M.)	Below left saphenous opening	Enlarged	Enlarged	Equal	None	Few in region of fistula	No increase in limb discrepancy	12 years	16 years	2, *A* and *B*
3. (T.I.)	Below left saphenous opening	Enlarged	Enlarged	Smaller	None	None	No improvement	8 years	10 years	3, *A* and *B*
4. (M.B.)	Below right saphenous opening	Enlarged	Enlarged	Equal	None	None	No increase in limb discrepancy	2 years	4 years	4
5. (K.B.)	Below left saphenous opening	Enlarged	Enlarged	Smaller	None	Around and proximal to fistula	Discrepancy less than 2 cm.	10 years	Present	5, *A* and *B*
6. (Y.E.)	Below left saphenous opening	Enlarged	Enlarged	Equal	None	Around and proximal to fistula	Discrepancy less than 1 cm.	11 years	18 years	6, *A* and *B*
7. (S.E.)	Upper third of the thigh	Slightly enlarged	Slightly enlarged	Equal	In mid thigh	None	Discrepancy less than 0.5 cm.	9 years	13 years	7, *A* and *B*
8. (P.K.)	Profunda femoris artery	Enlarged	Enlarged	Smaller femoral artery. Larger profunda artery	In upper thigh	At site of fistula and proximal to it	Not known	12 years	22 years	8, *A* and *B*

facilitate this an end-to-side anastomosis of the proximal saphenous vein to the superficial femoral artery[5] appears preferable to a side-to-side anastomosis.[2]

While the original site of the fistula is usually known, and physical examination will reveal a bruit, only arteriography will show the exact site and size of the fistula. In addition, any arterial collaterals and large venous tributaries in the operative field are demonstrated. We, therefore, suggest that arteriography is clearly indicated in the preoperative assessment prior to closure of the arteriovenous fistula..

SUMMARY

The angiographic findings in 8 patients with surgically produced arteriovenous fistulae performed for lower limb discrepancy are described. The exact site and size of the fistulae were shown in all patients. The arteries below the fistulae are usually of the same size as the normal limb or smaller, suggesting that increased blood flow is not the principal mechanism of the bone growth which may result from fistula formation.

Additional information concerning arterial collaterals and enlarged venous tributaries is also obtained.

It is suggested that arteriography should be performed immediately before closure of the fistulae.

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All the patients were under the care and operated upon by Professor J. B. Kinmonth, to whom we are grateful for permission to publish.

REFERENCES

1. Breslaw, R. C. Complications of arteriovenous fistula induced for augmentation of limb growth. *Surgery*, 1968, *63*, 1012–1018.
2. Janes, J. M., and Musgrove, J. E. Effect of arteriovenous fistula on growth of bone: experimental study. *Surg. Clin. North America*, 1950, *30*, 1191–1200.
3. Janes, J. M., and Jennings, W. K. Effect of induced arteriovenous fistula on leg length: 10 year observations. *Proc. Mayo Clin.*, 1961, *36*, 1–11.
4. Janes, J. M., and Sweeting, R. C. Experiences with discrepancy in length of lower limbs. *Proc. Mayo Clin.*, 1971, *46*, 489–491.
5. Kinmonth, J. B., and Negus, D. Arteriovenous fistulae in management of lower limb discrepancy. To be published.
6. Thomas, M. L., and Lintott, D. Large volume aortography in atherosclerotic peripheral vascular disease. *Acta radiol.*, 1973, *4*, 56–64.
7. Treleaven, R. B., and Copestake, G. G. Experiences with Seldinger catheterisation. *J. Canad. A. Radiologists*, 1962, *13*, 58–64.

THE TAPERED DILATOR IN PERCUTANEOUS PEDIATRIC NEUROANGIOGRAPHY*

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THE increased reliance by present day Neuroradiologists upon adult percutaneous femoral angiography has only more recently been applicable to pediatric practice.

In the infantile group, the minute size of the femoral artery and the ease with which this vessel undergoes vasospasm, makes it essential that the smallest possible guidewires and catheters be employed by the angiographer.

Optimal flow rate studies in this age group can be obtained with No. 3 or No. 4 selective polyethylene catheters. However, the soft tip of such catheters may cause them to fray or buckle upon entering the vasospastic artery. Even if vascular penetration is made by such a catheter, it is of definite risk to pass the damaged tip into a cerebral blood vessel.

Teflon is a more rigid material than polyethylene and will not usually splinter.

METHOD

A 20 gauge thin-walled Potts needle is used to enter the femoral artery followed by the passage of a .021 inch stainless steel guidewire with flexible tip through the needle. Subsequently we pass a tapered dilator* (Fig. 1). This dilator is made of teflon with the 1.5 cm. tapered portion size No. 3 and the remainder size No. 4. The tapered portion fits snugly over the guidewire enabling easy passage into the spastic femoral artery. By advancing the dilator further into the artery, the caliber of the opening in this vessel will be sufficient to subsequently permit the passage of any No. 4 selective polyethylene catheter.

* Cook, Inc., Bloomington, Indiana.

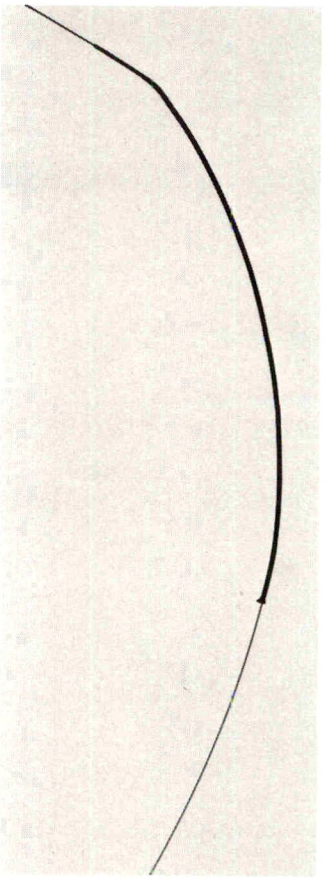


FIG. 1. Photograph of 0.021 inch stainless steel guidewire passing through the tapered dilator.

SUMMARY

In over 400 pediatric cases, we have successfully used this percutaneous technique without any thrombosis at the site of the arterial puncture and without any damage to the tip of the polyethylene selective catheter.

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A "SLIDE RULE" FOR ANGIOGRAPHERS*

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THERE is a boom in vascular radiology. Now that ready-to-use material is available, percutaneous catheterization techniques have become more accessible and the "market" for these procedures is growing. They are employed not only at large hospitals but at many small ones, with a resulting two-way traffic of x-ray films of patients treated at several centers. This affords an opportunity to see a large quantity of material of undoubted interest. However, a fair number of poorly performed investigations are seen and the accident rate is certainly rising.

The level of competence, often inversely correlated with confidence, varies widely in angiography rooms, where the procedures are performed by a variety of specialists: cardiologists, radiologists, surgeons and nephrologists. Rarely is each one of these, taken individually, perfectly familiar with all the ins and outs of vascular radiology. The specialist literature is full of advice, myths that die hard and passing fads. Everything or nearly everything is known about what should be done, but rarely is a high level of reproducibility found in this investigation, which is one of the most dangerous procedures of instrumental diagnosis.

As this—for want of a better term—semi-specialist practice of angiography has come to stay and there is little point in lamenting the fact, the situation seems to call for a simple mnemonic aid so that all the essentials are remembered. Our Department has developed a "slide rule," which we describe in case it might be of general interest.

SLIDE RULE DEVICE

This device is simply a square of ordinary perspex (Fig. 1) on which have been engraved and lead-painted the following key data:

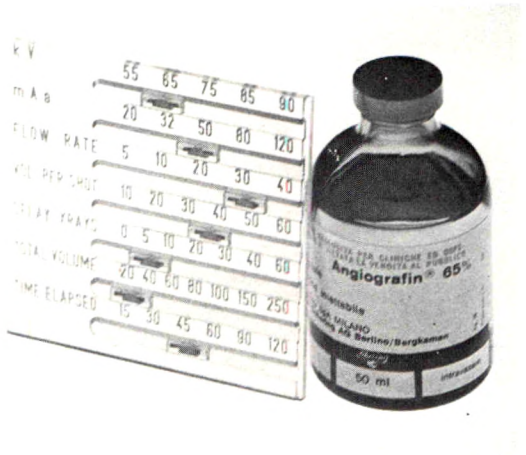


FIG. 1. The radiotransparent plastic slide rule alongside a 50 ml. bottle of contrast medium.

KV AND MAS

Memorization of the roentgenographic constants on x-ray film facilitates the search for the optimal exposure in the course of the investigation or in a subsequent evaluation of the patient. The degree of contrast on the angiograms gives an idea of the concentration of contrast medium used and guidance on whether a given method of injection should be done slower or faster or with more or less contrast material.

FLOW RATE

The flow rate is expressed in ml./sec. Exact memorization of the rate enables one at any time to assess the efficiency of a catheter or the velocity of circulation of a given organ and to locate the roentgenograms accurately in a sequence. This is calculated from the volume per shot and the delayed roentgenograms.

* From the Department of Radiology, National Cancer Institute of Milan, Italy.

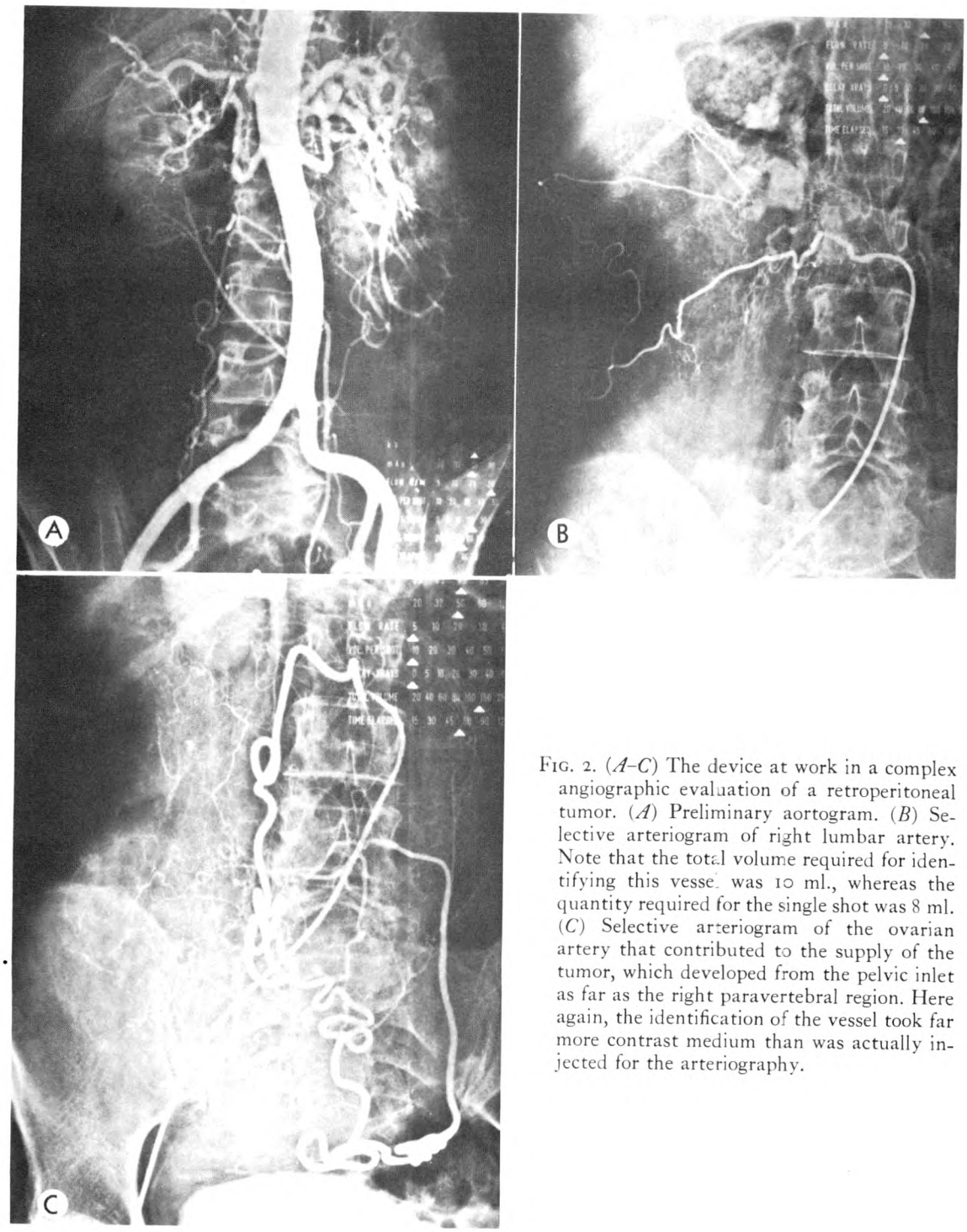


FIG. 2. (*A–C*) The device at work in a complex angiographic evaluation of a retroperitoneal tumor. (*A*) Preliminary aortogram. (*B*) Selective arteriogram of right lumbar artery. Note that the total volume required for identifying this vessel was 10 ml., whereas the quantity required for the single shot was 8 ml. (*C*) Selective arteriogram of the ovarian artery that contributed to the supply of the tumor, which developed from the pelvic inlet as far as the right paravertebral region. Here again, the identification of the vessel took far more contrast medium than was actually injected for the arteriography.

VOLUME PER SHOT

The maximum volume per shot on our slide rule is 60 ml./sec. If the flow rate is exact and the catheter is correctly positioned, a larger dose is needed only in exceptional cases.

DELAYED X-RAYS FILMS

The interval between injection of the contrast medium and exposure of the roentgenograms is expressed in ml.

TOTAL VOLUME

This is the volume in ml. of contrast medium injected in the course of the investigation and comprises the volumes of the single shots and those needed for correct positioning of the catheters.

TIME ELAPSED

This is the interval in minutes from the start of the investigation, which we calculate from the injection of local anesthetic or induction of general anesthesia.

DISCUSSION AND CONCLUSION

The 3 parameters: volume per shot, total volume, and time elapsed, are the checkpoints for preventing contrast medium toxicity. Obviously, the slower the flow rate and the longer the time elapsed, the lower the toxicity. However, the diagnostician wants maximum opacification at every stage in the territory under study. The compromise maxim "least quantity injected in least time" is valid, but it has to be applied flexibly because of the wide variety of clinical problems and because of organ toxicity (Fig. 2, *A–C*).

The functional status of the kidneys, the degree of hydration and the possible administration of fluids in the course of the investigation suggest that the total time in which a given quantity of contrast medium is delivered should be taken into account. On more than one occasion, since we have been using this slide rule, we have been surprised by the high consumption of contrast material for test doses in complicated catheterizations: it is not enough to remember roughly or to equate the test doses in subsequent calculations to 10 or 20 ml. of contrast medium.

The light is dim in angiography rooms, even with an image intensifier, and the various maneuvers have to be executed in quick succession. It is, therefore, simpler and safer for one of the team to calculate the quantity as it is consumed and shift the "total volume" indicator. The recording of the time elapsed will permit retrospective analysis of the difficulties encountered in the various examinations by recalling the type of maneuver, the materials and the team involved.

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I wish to thank Mr. A. Perego for invaluable technical assistance.

WOODEN SPLINTER INDUCED "PSEUDOTUMORS" AND "OSTEOMYELITIS-LIKE LESIONS" OF BONE AND SOFT TISSUE*

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WOODEN foreign bodies embedded in the extremities can result in so-called "pseudotumors" or "osteomyelitis-like lesions" of the bone and soft tissues. This phenomenon has been described with a variety of thorns, but is less well documented with other wooden foreign bodies. Because of this and because such foreign bodies are often initially overlooked, 2 instances are presented wherein a toothpick was embedded in the foot for a prolonged period of time and resulted in such a lesion.

REPORT OF CASES

CASE 1. R.H. On August 10, 1970, this 5 year old boy allegedly sustained a puncture wound to the dorsum of the right foot, between the second and third toes. Glass was reported to be the object responsible for the injury, but none was found in the wound. Roentgenograms obtained at that time were normal. Culture of debris from the wound grew *Staphylococcus aureus* and B-hemolytic *Streptococcus*: treatment consisted of excision and drainage of the wound, and systemic antibiotics (kanamycin and ampicillin). The boy did not return for a regular follow-up visit, but about 6 months later did come back with continued infection in the right foot. *Staphylococcus aureus* was again cultured, and local incision and drainage again performed.

On August 30, 1973, the boy again sustained injury to the right foot; on this occasion the foot had been struck with a metal hub cap. The right foot was still swollen and there was now a draining sinus between the second and third toes. Evidently inflammation and infection, to one degree or another, had been present since 1970, and the lesion had never completely healed. Once again *Staphylococcus aureus* and beta-*Streptococcus* were cultured from the wound, and, while incision and drainage were performed again, no foreign body was found. Roentgenograms obtained on this admission showed soft tissue thickening between the second and third metatarsals and early periosteal reaction along the metatarsal shafts (Fig. 1). Xeroradiography failed to reveal the presence of any foreign body. However, since the roentgenographic findings suggested a



FIG. 1. Case 1. Note periosteal new bone formation along the shaft of the second metatarsal (arrows). No bone destruction is seen.

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reactive periostitis, the possibility of a chronically embedded foreign body was now entertained.

Two months later the right foot was still swollen and the sinus still draining. Repeat roentgenograms demonstrated more periosteal new bone and persistent soft tissue thickening (Fig. 2). Wide incision and drainage were performed again, but on this occasion one half of a round wooden toothpick was found in the wound. It lay along the shaft of the second metatarsal. After appropriate debridement, inflammation subsided and the wound healed completely. Histologic examination of the debrided tissue showed a sinus tract with surrounding acute and chronic inflammation. Marked eosinophilia in the area of the sinus tract was also noted.



Fig. 2. Case 1. Two months later soft tissue swelling has caused separation of the second and third metatarsals. In addition, there is periosteal new bone formation along both metatarsal shafts (arrows). On the second metatarsal it has matured, while that on the third metatarsal is new.

Comment. Neither the patient nor anyone else seemed to know how the toothpick came to be embedded in the foot. However, on repeated questioning it became apparent that both the patient and his sibling often picked and probed at the lesion, and although never admitted, it is entirely possible that the toothpick was inserted in the wound in this manner. Certainly such an occurrence is not beyond question: we have encountered a third child who did embed a toothpick in her own foot. This girl had significant emotional and mental problems and after prolonged questioning finally admitted to sticking a toothpick in her foot. We did not include her in this report as the toothpick was not present long enough to evoke a soft tissue or bone response.

Case II. M.W. This 3 year old female stepped on a round toothpick 2 months prior to admission. The toothpick evidently stuck in the dorsal aspect of the left foot, between the third and fourth toes. At that time it was allegedly completely removed by the mother. Treatment consisted of local care to the wound. Subsequently, the patient was seen in the outpatient clinic on 2 occasions and on both demonstrated a draining sinus and persistent swelling between the third and fourth toes. There was, however, no erythema, tenderness, fluctuance, pain, or fever. Debris from the wound was cultured and *Staphylococcus aureus* was grown.

Roentgenograms obtained on the second of these visits (2 months after original injury) revealed deep soft tissue swelling, marked separation of the third and fourth metatarsals, and periosteal new bone formation along the fourth metatarsal (Fig. 3). No destructive lesions of the bone were noted. However, because of the over-all appearance of the lesion it was suggested that the toothpick might still be embedded in the soft tissues. Surgical exploration was then considered, but before it could be performed, the wooden toothpick was expelled spontaneously through the draining wound (Fig. 4). After this event swelling and inflammation subsided and progressive healing occurred.

DISCUSSION

Although thorn-induced pseudotumors of bone and soft tissue have been documented



FIG. 3. Case II. The pronounced soft tissue swelling and induration has caused separation of the third and fourth toes. Also, there is rather marked periosteal new bone formation along the shaft of the fourth metatarsal (arrows). No bone destruction is noted, but the bones are somewhat demineralized.

in the past,[1,3,4,6] little has been written about a similar phenomenon resulting from other organic foreign bodies such as wooden splinters or toothpicks.[5,6] In 1947 Breinlinger[2] reported experimentally induced new bone formation around foreign bodies, but this was more in relation to certain wire sutures and cat gut as utilized in surgery, and wooden splinter induced "pseudotumors" or "osteomyelitis-like lesions" remained relatively undocumented.

In the previously reported cases of thorn-induced pseudotumors, osteolytic or osteoblastic changes in the adjacent bones were present, and in some cases associated periosteal new bone formation was seen. Toxins from the thorns were postulated to have produced these changes in some of the cases,[3] but other possible explanations include: (1) simple irritation from the foreign body; and (2) secondary infection. Indeed, it seems likely that the combination of irritation by the foreign body (be it mechanical or toxic) and secondary infection leads to the soft tissue and bone changes seen in many of these cases. The clinical course is characteristically protracted and the first diagnostic considerations are usually those of low-grade bacterial or mycotic infection.

The roentgenographic findings are often the first clue to the correct diagnosis. This is exemplified in Case I wherein the history of foreign body embedment was nebulous. In Case II, even though it was known that a toothpick was embedded in the foot, it was erroneously believed that the entire toothpick was removed immediately after initial injury. No osteolytic or osteoblastic medullary bone changes suggesting osteomyelitis or bone tumor were present in our cases and periosteal reaction was believed to be secondary to the soft tissue inflamma-



FIG. 4. Case II. The draining sinus and soft tissue induration are evident between the third and fourth toes. Also note the toothpick (T) which was spontaneously expressed from the draining sinus.

tion (*i.e.*, reactive periostitis rather than true osteomyelitis). However, it should be noted that osteolytic or osteoblastic medullary bone lesions can occur with embedded thorns, twigs, and wooden splinters,[1,3–6] and, consequently, if any of the changes just outlined are noted in a patient with an indolent, deep soft tissue infection, a foreign body should be strongly suspected.

Excisional biopsy should always be performed no matter how typical the roentgenographic appearance is for infection alone, for only in this manner will the foreign body be discovered and the inflammatory reaction resolved. Local treatment of the draining sinus is inadequate, for if the foreign body remains in the soft tissues, continued inflammation is the rule.

Histopathologically, the foreign object is usually surrounded by granulation tissue and a dense fibrous covering.[3,4,6] There may also be a small amount of encapsulated, sterile fluid, and if a draining sinus is noted, chronic cellular infiltrates are usually present.

SUMMARY

When a toothpick or other wooden splinter is embedded in the soft tissues, chronic irritation occurs and secondary infection is commonplace. It is likely that both of these factors lead to the bone and soft tissue changes which result in so-called "pseudotumors" or "osteomyelitis-like lesions." Delay in diagnosis is not uncommon, for the history of foreign body embedment may be nebulous. However, when the roentgenographic findings of: (1) deep soft tissue thickening or swelling; (2) periosteal new bone reaction; or (3) osteolytic or osteoblastic bone change are seen in the presence of prolonged soft tissue infection, with or without a draining sinus, the diagnosis of such a chronically embedded foreign body should be strongly suspected. Surgical exploration and removal of the foreign body is mandatory, for if the foreign body is not removed, inflammation and infection do not resolve. If the foreign body is removed, complete clinical and roentgenographic resolution is the rule.

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We wish to express our appreciation to Dr. Allen Alpard, William Beaumont Army Hospital, El Paso, Texas, for permission to include Case II. We are also indebted to him for obtaining the illustrations of this case.

REFERENCES

1. Borgia, C. A. Unusual bone reaction to organic foreign body in hand. *Clin. Orthop. & Related Res.*, 1963, *30*, 188–192.
2. Breinlinger, K. Fremdkörpereinheilung mit Osteoplastischer Wirkung. *Schweiz. med. Wschr.* 1947, *77*, 1179–1184.
3. Gerle, R. D. Thorn-induced pseudo-tumours of bone. *Brit. J. Radiol.*, 1971, *44*, 642–645.
4. Maylahn, D. J. Thorn-induced "tumors" of bone. *J. Bone & Joint Surg.*, 1952, *34-A*, 386–388.
5. Ritvo, M. Bone and Joint X-ray Diagnosis. Lea & Febiger, Philadelphia, 1955, pp. 732–735.
6. Weston, W. J. Thorn and twig-induced pseudotumours of bone and soft tissues. *Brit. J. Radiol.*, 1963, *36*, 323–326.

DISTAL IRREGULARITIES OF THE FEMUR SIMULATING MALIGNANCY*

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A NUMBER of years ago we became interested in the irregularities seen in the distal end of the femur of children. These changes are seen along the posteromedial aspect of the femur in the metaphyseal region. The irregularity was sufficiently striking in some children to cause concern that it might be due to a malignancy. A significant number of children have had biopsies performed in this region because of suspected malignancy. In discussing this with colleagues in both this country and Europe, we discovered that our experience was not unique. We knew that this was a normal finding, but did not know the cause or frequency.

The purpose of this paper is to discuss the anatomy of this portion of the femur and to demonstrate the etiology of the roentgenographic appearance in this region.

MATERIAL

This study is based on the review of roentgenograms of the knees of 100 children with the knee externally rotated 20 to 45 degrees. These additional roentgenograms were obtained during the examination of children in whom the request for the roentgenogram was almost without exception because of recent trauma. The children were all presumably healthy and representative of those seen in any hospital emergency room because of trauma. No child with systemic or metabolic disease or congenital anomalies was included.

The knees of 5 adults and 2 children were dissected to study the soft tissue and osseous anatomy of the distal medial and posterior aspects of the femur. The adult specimens were from individuals ranging from ages 63 to 76 years who had above-knee amputations due to diabetic gangrene of the foot and lower leg. One of the child's specimens was from a 5 year old boy, whose leg was amputated because of gangrene due to thrombopenic purpura fulminans and the other was an 11 year old boy, who had a malignancy of the proximal femur with no evidence of disease involving the distal femur.

We also reviewed roentgenograms of the knees of 75 adults obtained with the same projection of external obliquity. Forty specimens of femora, 21 right and 19 left, made available to us by the Department of Biological Structure at the University of Washington were studied. These were all from adults, but the sex and age were not available.

ANATOMY

It is pertinent to review the anatomy of the adductor magnus muscle, as well as the distal femur.

This muscle has a large triangular shape with its origin at the apex. There are 2 major divisions of the muscle, the largest consisting of the adductor portion which originates primarily from the ischio-pubic ramus. This fans out and inserts by means of a broad aponeurotic sheath on the posterior aspect of the femur along a bony ridge known as the linea aspera. The latter extends almost the entire length of the femur. The second portion of the adductor magnus muscle, and the one of major interest in our study, is an extensor in function. It arises chiefly from the ischial tuberosity and runs in a vertical fashion as a large, fleshy bundle. It ends in the lower third

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of the thigh as a relatively thick tendon which inserts on the medial aspect of the distal femur at the adductor tubercle (Fig. 1). This tendon, and its traction, over the years of development is the mechanical cause of the formation of the adductor tubercle. The latter, from the standpoint of kinetics, would be more appropriately named the "extensor" tubercle. The adductor portion of the magnus is innervated by the obturator nerve, whereas the extensor portion is by the sciatic nerve.[4,5]

In the lower portion of the femur, the linea aspera splits and extends as an inverted Y. Its lateral arm is known as the lateral supracondylar ridge (LSR), or line, and ends at the lateral epicondyle. The medial extension is known as the medial supracondylar ridge (MSR), or line, and it ends distally at the adductor tubercle (Fig. 2).

The aponeurotic attachment of the adductor magnus muscle to the linea aspera is interrupted in its length by 3 or 4 fibrous arches for perforating branches of the deep femoral artery. Beneath these arches, the ridge of the linea aspera is not present. The arches vary in size and number in various specimens. The lowermost and largest of these is known as the hiatus tendineus, and at this site the femoral vessels pass into the popliteal fossa.

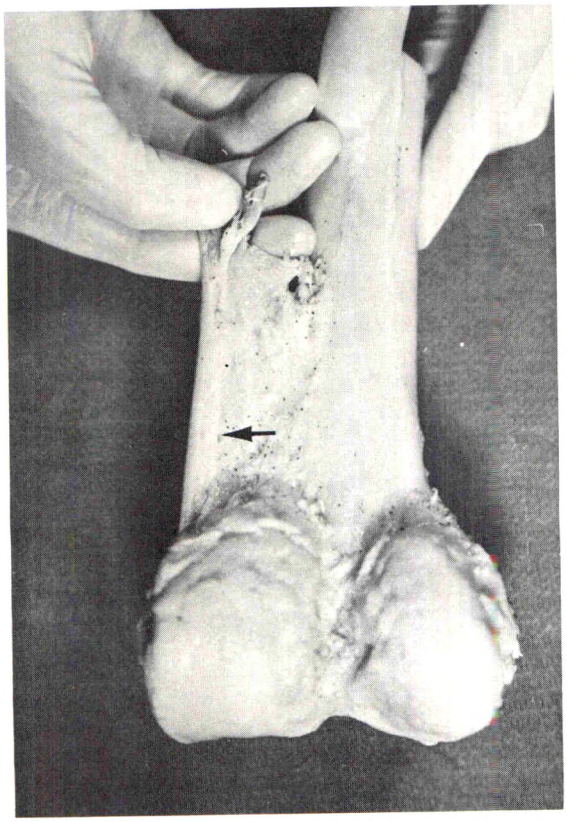


Fig. 1. Posterior aspect of distal femur showing extensor tendon of the adductor magnus (arrow).

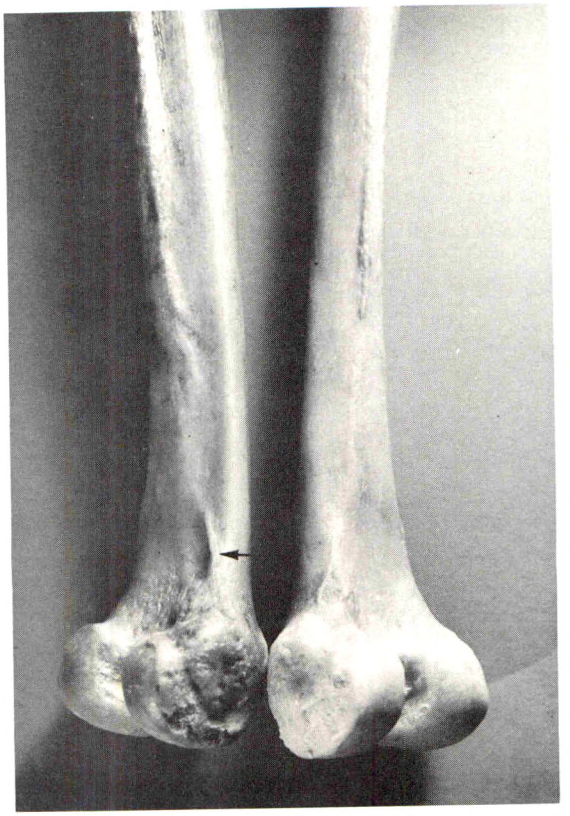


Fig. 2. Posterior aspect of femora in slight obliquity. There is a medial supracondylar ridge (arrow).

As one dissects the tendon of the extensor portion of the adductor magnus along its course to its insertion to the adductor tubercle, a relatively thin but firm, tough aponeurotic sheath is found (Fig. 3). This sheath runs in a horizontal medial direction perpendicular to the axis of the extensor tendon and is attached to (and the cause of the formation of) the previously described medial supracondylar ridge.

None of the reviewed textbooks of

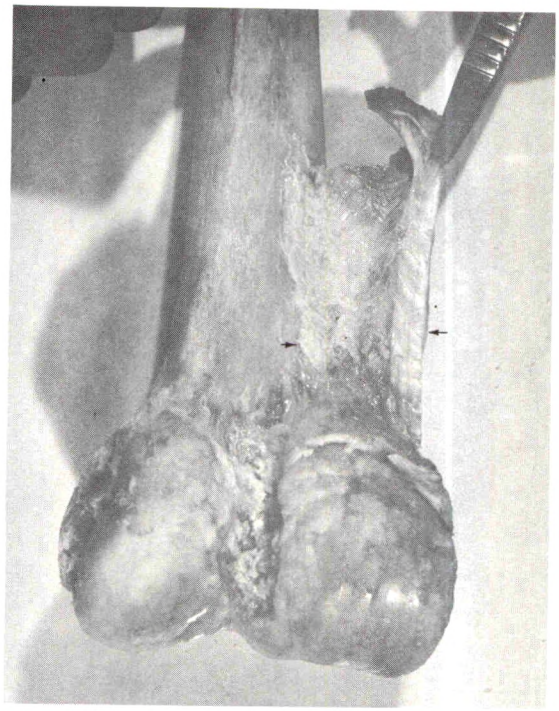


FIG. 3. Posterior aspect of femur showing extensor tendon (outer arrow), and aponeurosis from tendon to area of medial supracondylar ridge (inner arrow).

anatomy describe the aponeurotic sheath from the extensor tendon, but it was present in all dissections, and the underlying MSR conformed perfectly to its length. It apparently affords the extensor tendon a broader base upon which to carry out its function to greater mechanical advantage.

DISCUSSION

Simon[7] wrote about this finding in a retrospective study of 345 knees in children. He noted that it was slightly more than 3 times as common in boys as girls and approximately twice as common on the left as the right side. The greatest incidence was in the 10 to 15 year age group. It was his belief that the changes were most likely developmental in origin, but that they might also be due to post-traumatic alterations.

In 1953, Allen[1] cautioned that this irregularity should not be mistaken for malignancy, since it was a normal finding. Kohler and Zimmer[6] attributed the posterior changes to the origin of the gastrocnemius and plantaris muscle. Caffey and Silverman[3] pointed out that this popliteal irregularity is a normal finding.

We have observed that if the irregularity in the distal medial femur was seen, it would commonly be much more striking if the roentgenograms were obtained with the knee externally rotated from 20 to 45 degrees (Fig. 4, *A–C*). At times, the finding would not be visible in the routine anteroposterior projection, but easily seen in the oblique view. In the review of the roentgenograms of adult knees, there was a smooth irregularity in the identical location as in the children although none simulated malignancy. (We are, however, aware

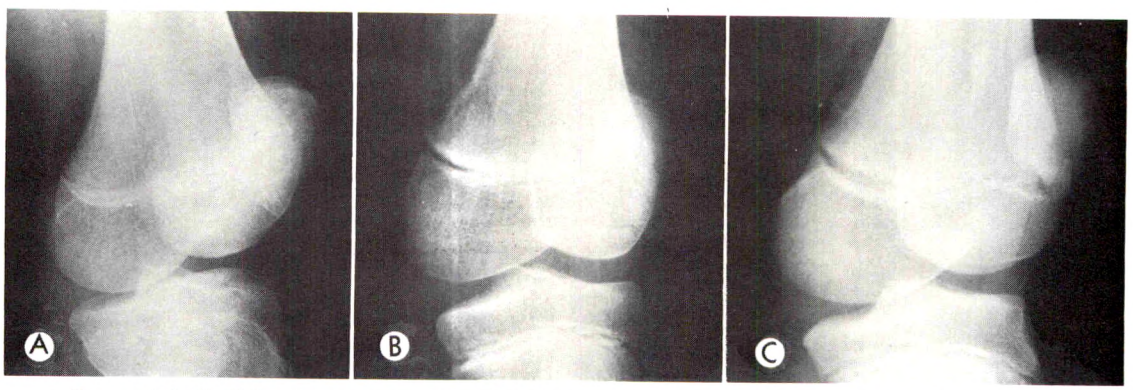


FIG. 4. (*A–C*) Oblique roentgenograms of the knee of 3 different children, showing irregularity along the medial supracondylar ridge.

of 2 young adults, who have had biopsies performed in this region because of the irregularity.)

This irregularity is rarely seen in the preschool child. The dissected knee of the 5 year old child was perfectly smooth and no ridge was present even though the aponeurotic sheath was formed and attached. It is our opinion that the development of the medial supracondylar ridge is time dependent and due to stress to the bone through the attached aponeurosis. A well formed adductor tubercle was not visualized on any roentgenograms prior to the closure of the distal femoral epiphysis. Irregularity of varying degrees from slight to striking was seen in virtually every oblique projection in the location where the adductor tubercle would eventually form.

In the anatomic specimens, the average length of the adductor tubercle at its base was 6.7 mm. on the right side and 8.5 mm. on the left. The average length of the MSR on the right was 26.0 mm. and the left 35.0 mm. (Table 1). There is considerable disparity in the measured length of the MSR on the oblique roentgenograms as compared to the measurements on the actual femoral specimens. This is explained by the varying course of the MSR on the posterior and medial aspect of the femur, as well as the variable degree of external obliquity used in making the exposure.

It is difficult to understand how a ridge is seen in 3 different projections (*i.e.*, anteroposterior, oblique and lateral), until the dissected specimen is viewed. The course of the MSR explains why one does not see the irregularity commonly on the straight anteroposterior projection and why it is best appreciated with obliquity (Fig. 5, *A–C*). The ridge is seen to run along the

TABLE I

AVERAGE LENGTH OF MEDIAL SUPRACONDYLAR RIDGE AND ADDUCTOR TUBERCLE

Roentgenograms of 100 Children External Oblique Projection			
Total Group: Number of Roentgenograms		Average Length of Medial Supracondylar Ridge (MSR)	
Right —51			15.0 mm.
Left —49			17.0 mm.
Males —62	32	Right	16.7 mm.
	30	Left	20.5 mm.
Females—38	19	Right	13.1 mm.
	19	Left	12.1 mm.
Anatomic Specimens—Femora All Adults—40 Specimens			
Average length of MSR	21	Right femur	26.0 mm.
	19	Left femur	35.0 mm.
Average length of adductor tubercle	21	Right femur	6.7 mm.
	19	Left femur	8.5 mm.

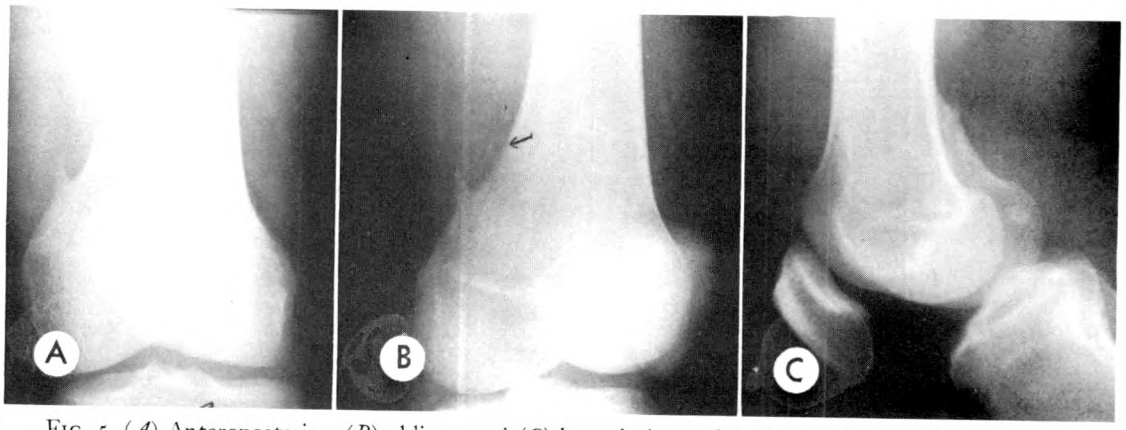


FIG. 5. (*A*) Anteroposterior, (*B*) oblique and (*C*) lateral views of the left knee of 26 year old male, illustrating medial supracondylar ridge (arrow) with small bony spur.

medial posterior aspect of the femur when viewed from the posterior aspect. It is usually more prominent close to the area of the adductor tubercle. In a few of the anatomic specimens, it was barely perceptible. In the 40 anatomic specimens, measurements of the length and height of the ridge and the adductor tubercle were recorded. They were slightly more prominent and larger on the left side, but there was no correlation between the size of the adductor tubercle and the length and prominence of the MSR. The ridge runs along the outermost medial posterior aspect of the femur close to the tubercle, but runs in an oblique fashion toward the midline in its more cephalad portion, which explains why it is not seen more commonly in the usual anteroposterior projection. It is sometimes seen extremely well in the lateral projection.

The MSR is the cause of the irregularity seen on the medial aspect of the distal femur. On the lateral view it is seen in the posterior aspect of the distal femur. Other reports have not been able to establish any muscle or tendinous attachments in this area.[2,8] It is our belief, however, that this ridge is formed due to the aponeurosis of the extensor portion of the adductor magnus muscle and the associated muscle stress. The medial head of the gastrocnemius muscle and the plantaris attachments have nothing to do with the irregularity seen in the distal femur.

SUMMARY

Roentgenograms of the knees, externally rotated, in 100 children were reviewed in evaluation of the irregularity seen on the posteromedial aspect of the distal femurs. Dissection studies of the soft tissue and osseous structures were carried out in order to determine the etiology of this irregularity. It is more commonly seen in boys and on the left side and may be mistaken for malignancy.

This apparent break in the cortex, with irregularity and occasional diminutive spicule formation, is a normal finding and due to the medial supracondylar ridge. An aponeurotic sheath from the extensor tendon of the adductor magnus muscle attaches along the medial supracondylar ridge, and with muscle stress and growth causes the development of this ridge. The development of the adductor tubercle is due to the attachment of the extensor tendon of the adductor magnus.

The medial supracondylar ridge is not usually seen in the preschool age and is most commonly identified in oblique or lateral projections of the knee in the 10 to 15 year old child. The adductor tubercle is not seen until after closure of the distal femoral epiphysis.

These findings should not be confused with malignant change and biopsy in the majority of cases is unnecessary.

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REFERENCES

1. Allen, D. H. Variation of diaphyseal development which simulates roentgen appearance of primary neoplasms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *69*, 940–943.
2. Brower, A. C., Culver, J. C., and Keats, T. E. Histological nature of medial posterior distal femoral metaphysis in children. *Radiology*, 1971, *99*, 389–392.
3. Caffey, J., and Silverman, F. N. Pediatric X-Ray Diagnosis. Fifth edition. Year Book Medical Publishers, Inc., Chicago, 1967, pp. 777–779.
4. Gardner, E., Gray, D., and O'Rahilly, R. Anatomy. Second edition. W. B. Saunders Company, Philadelphia, 1963, pp. 271–272.
5. Gray, H. Anatomy of the Human Body. Edited by C. M. Goss. Twenty-seventh edition. Lea & Febiger, Philadelphia, 1959, p. 535.
6. Kohler, A., and Zimmer, E. A. Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology. Edited by J. T. Case. Tenth edition. Grune & Stratton, Inc., New York, 1956, p. 514.
7. Simon, H. Medial distal metaphyseal femoral irregularity in children. *Radiology*, 1968, *90*, 258–260.
8. Young, D. W., Nogrady, M. B., Dunbar, J. S., and Wigglesworth, F. W. Benign cortical irregularities in distal femur of children. *J. Canad. A. Radiologists*, 1972, *23*, 107–115.

NEUROTOXICITY OF X-RAY CONTRAST MEDIA*

RELATION TO LIPID SOLUBILITY AND BLOOD-BRAIN BARRIER PERMEABILITY

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X-RAY contrast media that are employed in cerebral and spinal cord angiography occasionally produce neurologic complications that are manifestations of neurotoxicity. The experimental approach to neurotoxicity is confused, however, because multiple factors come into play and neurotoxic endpoints have not been well defined.[1] For example, hemodynamic responses to contrast media and breakdown of the blood-brain barrier have been used as endpoints,[17,37] but they are not necessarily related to the central nervous system involvement that represents neurotoxicity.[2,12,17,26,33,34,36]

Solutions of currently used x-ray contrast media (salts of triiodobenzoic acid derivatives) are as concentrated as 1.45 osmolal and can open the blood-brain barrier at equal osmotic concentrations of about 1.2 osmolal, when perfused into the internal carotid artery of rabbits.[34] They probably widen tight junctions between cerebrovascular endothelial cells by shrinking the cells osmotically. The continuous cerebrovascular endothelium constitutes the blood-brain barrier to passive entry of solutes into the brain and is permselective for lipid soluble solutes.[5,19,29,35]

Because they open the barrier at equal osmolalities, it is probable that differences in neurotoxicity among the x-ray contrast media are unrelated to osmotic barrier opening. Such differences may depend instead on specific physico-chemical properties that regulate media permeability through the barrier, such as lipid solubility and the aqueous dissociation constant.

Such a possibility has not been considered seriously before because salts of the triiodobenzoic acids are almost completely dissociated at physiologic pH.[13,15,24] Entry of organic electrolytes into the brain across the blood-brain barrier is thought to depend entirely on the concentration and lipid solubility of the undissociated lipid soluble form.[3,28]

The dissociated form of an organic electrolyte has, however, a finite lipid solubility.[10] Since the ratio between intravenous and intracisternal LD_{50} in mice is as high as 1,400:1 for some contrast media,[14] entry into the brain of even a small fraction of the arterial concentration may be sufficient to produce neurotoxicity.[20] This small entry may in part include the dissociated form. It was of interest therefore to estimate quantitatively the brain uptake of contrast media of both the dissociated and undissociated forms, and to see if neurotoxicity and systemic toxicity could be related to lipid solubility and concentration. An abstract of this paper has been published.[32]

METHOD AND RESULTS

RELATION OF NEUROTOXICITY OF X-RAY CONTRAST MEDIA TO LIPID SOLUBILITY

Recent reviews on x-ray contrast media that are derivatives of benzoic acid have concluded that they have distinct differences in their neurotoxicity.[1,12,13,36] The use of some has been discarded because of the high incidence of neurologic complications. In the United States, Conray, Hypaque and Isopaque remain in use. On the basis of these reviews, we rank ordered 8 contrast media anions with respect to neurotoxicity, as illustrated in Table 1. A rank of 3.5 for

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Table I

CORRELATION OF NEUROTOXICITY WITH OCTANOL/WATER PARTITION COEFFICIENT (P_{HA}) FOR 8 CONTRAST MEDIA*

Contrast Medium Anion	Commercial Name	Rank of Neurotoxicity	Log P_{HA}	Rank of Lipid Solubility
Iothalamate	Conray	1	1.96	2
Ioxithalamate	Vasobrix	2	1.30	1
Diatrizoate	Hypaque, Urografin	3.5	2.50	3
Metrizoate	Isopaque	3.5	2.60	4.5
Iodamide	Uromiro	5	2.60	4.5
Diprotrizoate	Miokon	6	3.50	7
Acetrizoate	Urokon	7	3.29	6
2,4,6-Triiodobenzoate	—	8	4.08	8

* Log P_{HA} values are taken from Table II. The rank correlation coefficient between neurotoxicity and lipid solubility is 0.94 ($P < 0.01$).

diatrizoate and metrizoate indicates that these 2 anions have the same neurotoxicity.

The lipid solubility of a compound can be defined as its tendency to be distributed selectively in a lipid rather than in an aqueous phase, and is conveniently quantified by its octanol/water partition coefficient.[22] The permeability of cell membranes and of the blood-brain barrier to a substance increases as the lipid solubility or partition coefficient of the substance increases.[4,5,27] As far as we know, partition coefficients have not been measured for the contrast media, and therefore we calculated octanol/water partition coefficients by using rules for their additive-constitutive properties.[6,8,10,22,23]

For a parent compound whose coefficient is P' and for a derivative compound whose coefficient is P'_x, the additive constant π_x is defined as

$$\pi_x = \log P'_x - \log P'. \qquad (1)$$

For instance, if the parent compound is benzoic acid and the derivative compound is p-iodobenzoic acid,

$$\pi_{p-I} = \log P'_{p-I\ benzoic} - \log P'_{benzoic}$$
$$1.15 = 3.02 - 1.87.$$

Experimental log P' values were obtained from tabulations by Leo *et al.*[22]

Table II illustrates how the octanol/water partition coefficients were calculated for the 8 contrast media anions in Table I. P_{HA} is the coefficient of the undissociated acid, HA. Adding iodide to benzoic acid increases P_{HA}, while adding the acetamide group decreases P_{HA}.

Comparison of the rank order of neurotoxicity with that of P_{HA} shows that the more neurotoxic agents are more lipid soluble (Table I), the rank correlation coefficient being 0.94 ($P < 0.01$). The exact rank of either neurotoxicity or lipid solubility may be unreliable because of the estimates that we make, but this significant correlation indicates their very close relation.

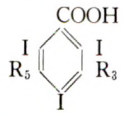
RELATION BETWEEN BRAIN UPTAKE AND LIPID SOLUBILITY

The correlation between octanol/water partition and clinical neurotoxicity suggests that a rate-limiting step regulating neurotoxicity is the passage of the contrast medium across cell membranes, presumably those of the blood-brain barrier.[5,19] The question arises as to whether brain uptake of contrast media is large enough to justify this suggestion, and is in the same quantitative order as neurotoxicity.

In the Appendix we calculate a partition parameter P which takes into account the relative concentrations of the undissociated form HA and dissociated form A^- of the contrast media acids, as well as their respective partition coefficients P_{HA} and P_A. We assume that net brain uptake of both forms of the organic acid will be a function

TABLE II

CALCULATION OF LOG P_{HA} FOR 2,4,6-TRIIODOBENZOIC ACID AND ITS DERIVATES*



Contrast Medium	Additions	Calculations	Log P_{HA}
2,4,6-Triiodobenzoate	R_3: −H R_5: −H	$\log P_{benzoic} + \pi_{p-I} + 2\pi_{o-I}$ 1.87 +1.15+2(0.53)	4.08
Acetrizoate	R_3: −NHCOCH$_3$ R_5: −H	$\log P_{tri-I} + \pi_{m-acetamido}$ 4.08 −0.79	3.29
Diatrizoate	R_3: −NHCOCH$_3$ R_5: −NHCOCH$_3$	$\log P_{tri-I} + 2\pi_{m-acetamido}$ 4.08 −1.58	2.50
Iothalamate	R_3: −CONHCH$_3$ R_5: −NHCOCH$_3$	$\log P_{tri-I} + \pi_{m-acetamido} + \pi_{m-CONH2} + \pi_{CH3\ near\ NH}$ 4.08 −0.79 −1.49 +0.16	1.96
Metrizoate	R_3: −N(CH$_3$)COCH$_3$ R_5: −NHCOCH$_3$	$\log P_{tri-I} + 2\pi_{m-acetamido} + \pi_{CH3\ on\ N}$ 4.08 −1.58 +0.1	2.60
Iodamide	R_3: CH$_2$NHCOCH$_3$ R_5: −NHCOCH$_3$	$\log P_{tri-I} + 2\pi_{m-acetamido} + \pi_{CH3\ on\ N}$ 4.08 −1.58 +0.1	2.60
Ioxithalamate	R_3: CONHCH$_2$CH$_2$OH R_5: −NHCOCH$_3$	$\log P_{tri-I} + \pi_{m-acetamido} + \pi_{m-CONH2CH3} + \pi_{m-CH2OH}$ 4.08 −0.79 −1.33 −0.66	1.30
Diprotrizoate	R_3: NHCOCH$_2$CH$_3$ R_5: NHCOCH$_2$CH$_3$	$\log P_{tri-I} + 2\pi_{m-acetamido} + 2\pi_{CH3}$ 4.08 −1.58 +1.0	3.50

* Log P_{HA} values are composed additively of individual π_x values, as discussed in text. Log P_{tri-I} = log $P_{2,4,6-triiodobenzoic\ acid}$. Source of substituent constants is Leo *et al.*[22]

of the lumped partition parameter P, since blood-brain barrier permeability increases with increasing partition coefficient for solutes in general.[5]

Blood-brain barrier permeability and brain uptake of the benzoic acid derivates have not been measured, but recently a method has been developed by which brain uptake of 7 aliphatic monocarboxylic acids was determined.[25] A mixture of C^{14} tracer and of $H_2{}^3O$ (which easily crosses the barrier) was injected into the internal carotid artery of the rat and the brain was sampled 15 seconds later. The brain uptake index (BUI) is calculated as follows:

$$\text{Brain Uptake Index (BUI)} = \frac{\text{Brain } C^{14}/\text{Brain } H^3}{\text{Injectate } C^{14}/\text{Injectate } H^3} \quad (2)$$

Figure 1 shows that the BUI's of the aliphatic monocarboxylic acids increase with increasing values of log P, as proposed above. This supports the suggestion that the rate limiting step for passive brain uptake of these acids is their transfer across the lipoid membranes of the blood-brain barrier. BUI is not a linear function of blood-brain barrier permeability, however, because very high BUI's depend also on cerebral blood flow.

Log P in Figure 1 was calculated with the use of Equations 7 and 8 from experimental values of log P_{HA},[22] letting $pK_a = 4.8$[39] and blood pH = 7.4. P_A was taken as $10^{-4.1}P_{HA}$.[7,10] The experimental BUI's in Figure 1 exclude contributions made by carrier-mediated mechanisms to the brain uptake of some of the monocarboxylic acids.[25]

The BUI for the x-ray contrast media can be predicted from the BUI of the aliphatic monocarboxylic acids, assuming that the BUI of both classes depends on log P. Contrast media that are derivatives of benzoic acids have pK_a's closer to 3.0 than to 4.8.[15] In 50 per cent methanol-water, pK_a's range from 3.0 to 3.6,[24] and calculate out to be about 3.0 in aqueous solution,[16] (using a reaction constant $\rho = 1.085$).

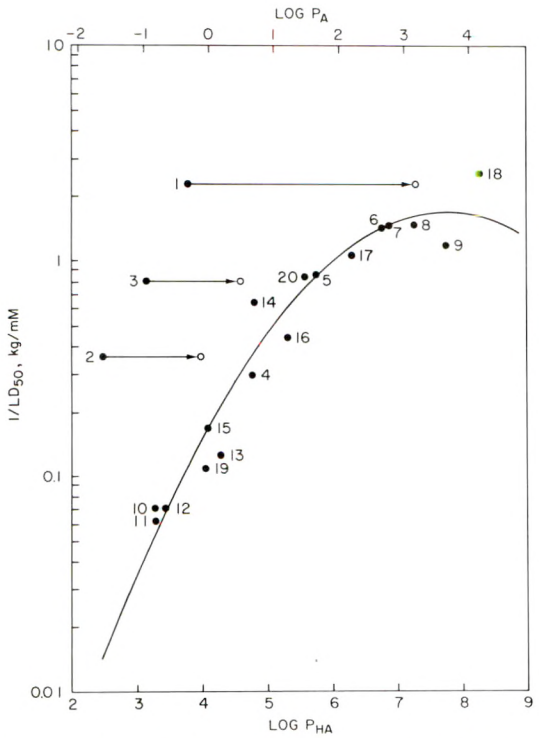


FIG. 1. *Relation of brain uptake of organic acids to octanol/water partition coefficients.* The brain uptake of an organic acid is quantified by the brain uptake index (BUI) which is the ratio of labeled acid to tritiated water in the brain relative to that injected into the internal carotid artery 15 seconds earlier[25] (see text). The experimentally determined uptakes of 7 aliphatic monocarboxylic acids (filled circles) are given on the ordinate, and the abscissa is log P as calculated from experimental log P_{HA},[22] $pK_a = 4.8$,[39] and pH = 7.4, as given by Equation 8. The experimentally determined BUI's exclude contributions made by carrier-mediated transport.[25] They are fit by the least squares curve given by the equation, log BUI = 4.459 + 0.324 log P − 1.161 (log P)^2. The open circles in the figure represent the predicted brain uptakes of the x-ray contrast media, where their log P_{HA} are given in Table 1, $pK_a = 3.0$, and log P = log P_{HA} − 3.92 (Equation 9).

By using Equation 7 in the Appendix, we were able to calculate log P for the contrast media at pH 7.4 from their log P_{HA} in Table 1, from $pK_a = 3.0$, and by letting $P_A = 10^{-4.1} P_{HA}$. The open circles in Figure 1 represent the predicted values of BUI for the x-ray contrast media acids, as based on observed BUI for the monocarboxylic acids. The lower pK_a's for the contrast media shift their effective partition parameter log P to the left of the values for the monocarboxylic acids, thereby decreasing their predicted brain uptake index.

As pointed out by Oldendorf,[25] a BUI of 2 per cent indicates that a substance is practically impermeant across the blood-brain barrier, while higher values show that it can enter the brain. On the basis of our calculations and assumptions, it appears that the contrast media will have a significant permeability across the blood-brain barrier, and that differences in their permeability may be enough to account for the correlation between partition coefficient and neurotoxicity.

RELATION OF LD_{50} OF SUBSTITUTED BENZOIC ACIDS TO OCTANOL/WATER PARTITION COEFFICIENT AND PK_a

LD_{50} is an administered quantity of drug that is lethal to 50 per cent of test animals. Its measurement alone leaves open the mode of death and the target organs affected, and therefore cannot be used without further information to indicate neurotoxicity. For a large number of drugs, LD_{50} decreases with increasing lipid solubility, indicating that the passage of agents across lipid cell membranes is an important factor in systemic toxicity.[9]

Figure 2 shows that systemic toxicity in mice, as measured by the quantity log $(1/LD_{50})$, is highly correlated with calculated log P_{HA} or log P_A for a large number of benzoic acid derivatives which are analogs of drugs used clinically as contrast media.[38] The major exceptions to this rule are 2-amino- and 4-amino-3,5-diiodobenzoic acid and 3-amino-2,4,6-triiodobenzoic acid, probably because their pK_a's are higher than those of the other acids. The pK_a's of

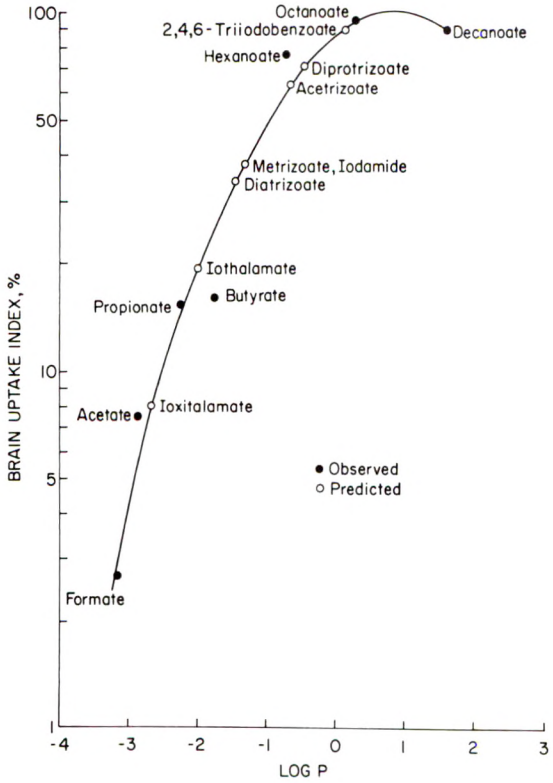


FIG. 2. *Relation of systemic toxicity to log* P_{HA} *for derivatives of benzoic acid.* Systemic toxicity is represented as log $(1/LD_{50})$ for mice.[38] Log P_{HA} is calculated as shown in text. The curve is the least squares fit to the data, excluding points (1), (2) and (3). It is given by the equation, log $(1/LD_{50}) = -0.076 (\log P_{HA})^2 + 1.17 \log P_{HA} - 4.278$. The numbers represent the following benzoic acid derivatives: (1) 2-amino-3,5-diiodobenzoic; (2) 3-amino-2,4,6-triiodobenzoic; (3) 4-amino-3,5-diiodobenzoic; (4) 2-acetylamino-3,5-diiodobenzoic; (5) 2-butyrylamino-3,5-diiodobenzoic; (6) 2-(n-caproylamino)-3,5-diiodobenzoic; (7) 2-benzoylamino-3,5-diiodobenzoic; (8) 2-(α-phenylbutyrylamino)-3,5-diiodobenzoic; (9) 2-(o-iodobenzoylamino)-3,5-diiodobenzoic; (10) 3-formylamino-2,4,6-triiodobenzoic; (11) 3-acetylamino-2,4,6-triiodobenzoic; (12) 3-propionylamino-2,4,6-triiodobenzoic; (13) 3-butyrylamino-2,4,6-triiodobenzoic; (14) 3-benzoylamino-2,4,6-triiodobenzoic; (15) 3-isobutyrylamino-2,4,6-triiodobenzoic; (16) 3-caproylamino-2,4,6-triiodobenzoic; (17) 3-caprylamino-2,4,6-triiodobenzoic; (18) 3-lauroylamino-2,4,6-triiodobenzoic; (19) 4-acetylamino-3,5-diiodobenzoic; (20) 4-benzylamino-3,5-diiodobenzoic.

Substances (1), (2) and (3) were excluded from the least squares calculation because their pK_a's differ markedly from those of the others (see text). If pK_a's are taken into account by plotting log $(1/LD_{50})$ against log P rather than log P_{HA}, the positions of (1), (2) and (3) would move to the right, relative to the positions of the other compounds, as illustrated by the open circles and arrows in the figure.

the latter are close to 3.0, the value of acetrizoic acid, while the pK_a of 2-amino-benzoic acid is 6.97, of 3-amino-benzoic acid is 4.78 and of 4-amino-benzoic acid is 4.92[15,39] (see Discussion).

A plot of $1/LD_{50}$ against log P, which is calculated with use of these pK_a's (Equation 7), reduces the discrepancies in Figure 2 for compounds (1), (2) and (3). We illustrate this in Figure 2 by showing how the positions of these 3 acids would approximate those of the others, if all $1/LD_{50}$ were plotted against log P rather than log P_{HA}. Differences of pK_a lead to differences in the additive constant of Equation 7.

DISCUSSION

Many factors have been related to the neurotoxicity of x-ray contrast media. Implicated have been agglutination of red blood cells and increased blood viscosity, vasospasm and modification of cerebral blood flow, the direct toxic effect on cells and opening of the blood-brain barrier.[1]

The data reported in this paper show that neurotoxicity of several contrast media with approximately equal pK_a's is correlated with their lipid solubility as measured by their octanol/water partition coefficient P_{HA}. If the pK_a's of the organic acids differ however, brain entry will depend also on the relative concentrations of A^- and HA. These are determined by pK_a and are accounted for in the partition parameter P (Appendix). Organic acids with higher pK_a's have higher proportions of the undissociated HA form, higher values of P and higher systemic toxicities, as illustrated in Figure 2.

Although it is generally believed that the blood-brain barrier excludes charged compounds and the dissociated form of organic electrolytes from entering the brain,[3,28]

these compounds will enter the brain to some degree if they have a finite lipid solubility. The partition coefficient of A^- amounts to $10^{-4.1}$ that of HA for benzoic acid derivatives. Since $pK_a \simeq 3.0$, $[A^-]/[HA] = 10^{4.4}$ at pH 7.4 (Equation 4). The term which determines the relative contributions of A^- and HA to P in Equation 3 is $P_A[A^-]/P_{HA}[HA]$ and equals 2.0. In other words, the dissociated acid form A^- of the contrast media should make up two-thirds of the partition parameter P. It remains to determine actual values of P for the contrast media, as well as their brain uptake index.

For the aliphatic monocarboxylic acids on the other hand, with $pK_a \simeq 4.8$, the quantity $P_A[A^-]/P_{HA}[HA] = 0.03$. Thus, the dissociated form A^- contributes only 3 per cent to P.

The permselectivity of the blood-brain barrier can be reduced by osmotically shrinking cerebrovascular endothelial cells and opening tight junctions between them. Concentrated solutions of urea and NaCl, as well as of contrast media, probably open the blood-brain barrier in this way.[29,30,31,34] It is reasonable to assume that the neurotoxicity which increases as the quantity of contrast medium administered is increased depends in part on osmotic opening of the barrier and the consequent entry of the contrast medium into the brain.

We therefore can identify 2 factors related to neurotoxicity which have to do with passage of contrast medium from blood to brain across the blood-brain barrier. The anion specific factor is the lipid solubility, or partition coefficient of the contrast medium, which determines passage *through* cerebrovascular endothelial cell membranes of the unaltered barrier. The nonspecific factor is the osmolality of the contrast media, which can open tight junctions and permit passage *between* cells.

Both of these influences would be enhanced by prolonged contact with the cerebrovascular system due to prolonged duration of perfusion or to repeated injections, and could explain the enhanced neurotoxicity under these conditions. Additional factors such as reduced blood flow may be important. Possible enhanced entry into the brain by carrier-mediated transport also must be considered.[25]

On the basis of our calculations, it would appear that the neurotoxicity of contrast media could be reduced by reducing P_{HA}. In addition, a reduction in pK_a would decrease the proportion of the more lipid soluble HA form in solution. Neurotoxicity could be reduced also by increasing protein binding,[18,21] although fractional binding is correlated with P_{HA}.[11] The use of polymeric contrast media will reduce medium osmolality[12] and the possible osmotic opening of the blood-brain barrier that also can produce neurotoxicity.

How toxicity is produced once the agents enter the brain is still an unresolved question. The basis for their action may lie in their ability to directly affect central nervous system neurons. Benzoic acid derivatives have been shown to affect the membrane permeability and electrical activity of isolated neurons with a relative potency which is highly correlated with their lipid solubility.[23] Hoppe,[14] however, found no difference in LD_{50} between acetrizoate and diatrizoate when given intracerebrally in mice, although acetrizoate was twice as toxic as metrizoate by the intravenous route. Clearly, the differential effects of contrast media on isolated cells require further study.

SUMMARY

Neurotoxicity of x-ray contrast media used in cerebral and spinal cord angiography is related to 2 factors.

The specific factor is the lipid solubility, or octanol/water partition coefficient of the contrast medium anion, which determines its passage through membranes of cerebrovascular endothelial cells of the blood-brain barrier.

The nonspecific factor is solution osmolality, which opens tight junctions between the cells through which the contrast media then can enter the brain.

Differences in neurotoxicity of 8 contrast media that are derivatives of benzoic acid are correlated with differences in their specific octanol/water partition coefficient. Brain uptake depends on the partition coefficient and on pK_a, which determines the proportion of dissociated and undissociated form of the acid in blood. Systemic LD_{50} of the benzoic acid derivates also depends on octanol/water partition and on pK_a.

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APPENDIX

The octanol/water partition coefficient of the undissociated form of an organic acid is defined as P_{HA}, and that of the dissociated form is defined as P_A. Let a combined partition coefficient P depend linearly on the fractional concentrations of A^- and HA in solution, multiplied by their respective partition coefficients,

$$P = P_{HA}\frac{[HA]}{[A^-]+[HA]} + P_A\frac{[A^-]}{[A^-]+[HA]}. \quad (3)$$

This equation means that the contributions to P of A^- and HA are in the ratio $P_A[A^-]/P_{HA}[HA]$. [HA] can be calculated from the acidic dissociation constant K_a and $[H^+]$, and can be written in terms of pK_a and pH as well,

$$\frac{[HA]}{[A^-]} = \frac{[H^+]}{K_a} = 10^{(pK_a-pH)}. \quad (4)$$

When $pH-pK_a>1$, the acid is at least 90 per cent dissociated and

$$[A^-] \simeq [HA] + [A^-]. \quad (5)$$

Substituting Equations 4 and 5 into Equation 3 gives the following:

$$P = P_{HA}(P_A/P_{HA} + [H^+]/K_a). \quad (6)$$

As discussed in the text, for the benzoic acids and aliphatic monocarboxylic acids $P_A = 10^{-4.1} P_{HA}$, and substitution into Equation 6 gives, after conversion to logarithms,

$$\log P = \log P_{HA} + \log [10^{-4.1} + 10^{(pK_a-pH)}]. \quad (7)$$

At pH 7.4 and when $pK_a=4.8$ (aliphatic monocarboxylic acids), Equation 7 gives,

$$\log P = \log P_{HA} - 2.59. \quad (8)$$

At $pH=7.4$ and $pK_a=3.0$ (benzoic acid derivatives), Equation 7 gives,

$$\log P = \log P_{HA} - 3.92. \quad (9)$$

Equations 8 and 9 differ by a constant.

We thank Dr. Giovanni Di Chiro for the many helpful discussions and for elucidating for us the clinical aspects of neurotoxicity, and Dr. Eugene Streicher for helpful criticism. The computer time for the project was supported in part through the facilities of the Computer Science Center at the University of Maryland.

REFERENCES

1. ALMÉN, T. Toxicity of radiocontrast agents. In: Radiocontrast Agents. Volume 2. Edited by P. K. Knoefel. Pergamon Press, New York, 1971, p. 443.
2. BLOOR, B. M., WRENN, F. R., and MARGOLIS, G. Experimental evaluation of certain contrast media used for cerebral angiography. *J. Neurosurg.*, 1951, *8*, 585–594.
3. BRODIE, B. B., and HOGBEN, C. A. M. Some physico-chemical factors in drug action. *J. Pharm. & Pharmacol.*, 1957, *9*, 345–380.
4. COLLANDER, R. Permeability of plant protoplasts to non-electrolytes. *Trans. Faraday Soc.*, 1937, *33*, 985–990.
5. CRONE, C. Permeability of brain capillaries to non-electrolytes. *Acta physiol. scandinav.*, 1965, *64*, 407–414.
6. FUJITA, T., IWASA, J., and HANSCH, C. New substituent constant, Π, derived from partition coefficients. *J. Am. Chem. Soc.*, 1964, *86*, 5175–5180.
7. HANSCH, C. Personal communication.
8. HANSCH, C. Quantitative structure-activity relationships in drug design. In: Drug Design. Volume 1. Edited by E J Ariens. Academic Press, New York, 1971, p. 271.
9. HANSCH, C., and DUNN, W. J. Linear relationships between lipophilic character and biological activity. *J. Pharm. Sc.*, 1972, *61*, 1–19.
10. HANSCH, C., and GLAVE, W. R. Structure-activity relationships in membrane-perturbing agents: hemolytic, narcotic and antibacterial compounds. *Mol. Pharmacol.*, 1971, *7*, 337–354.
11. HELMER, F., KIEHS, K., and HANSCH, C. Linear free-energy relationship between partition coefficients and binding and conformational

perturbation of macromolecules by small organic compounds. *Biochemistry*, 1968, *7*, 2858–2863.

12. Hilal, S. K. Trends in preparation of new angiographic contrast media with special emphasis on polymeric derivatives. *Invest. Radiol.*, 1970, *5*, 458–472.
13. Hoey, G. B., Wiegert, P. E., and Rands, E. D. Organic iodine compounds as x-ray contrast media. In: Radiocontrast Agents. Volume 1. Edited by P. K. Knoefel. Pergamon Press, New York, 1971, p. 23.
14. Hoppe, J. O., Some pharmacological aspects of radiopaque compounds. *Ann. New York Acad. Sc.*, 1959, *78*, 727–739.
15. Hoppe, J. O. Duprey, L. P., Borisenok, W. A., and Bird, J. G. Selective radiopacity in cardiovascular angiography. *Angiology*, 1967, *18*, 257–272.
16. Jaffé, H. H. Reexamination of Hammett equation. *Chem. Rev.*, 1953, *53*, 191–261.
17. Jeppsson, P. G., and Olin, T. Neurotoxicity of roentgen contrast media: study of blood-brain barrier in rabbit following selective injection of contrast media into internal carotid artery. *Acta radiol.* (Diag.), 1970, *10*, 17–34.
18. Knoefel, P. K. Binding of iodinated radiocontrast agents to plasma proteins. In: Radiocontrast Agents. Volume 1. Edited by P. K. Knoefel. Pergamon Press, New York, 1971, p. 133.
19. Krogh, A. Active and passive exchanges in inorganic ions through surfaces of living cells and through living membranes generally. *Proc. Roy. Soc. Ser. Biol.*, 1946, *133*, 140–200.
20. Lampe, K. F., James, G., Ebersfeld, M., Mende, T. J., and Viamonte, M. Cerebrovascular permeability of water-soluble contrast material, hypaque (sodium diatrizoate): experimental study in dogs. *Invest. Radiol.*, 1970, *5*, 79–85.
21. Lang J. H., and Lasser, E. C. Binding of roentgenographic contrast media to serum albumin. *Invest. Radiol.*, 1967, *2*, 396–400.
22. Leo, A., Hansch, C., and Elkins, D. Partition coefficients and their uses. *Chem. Rev.* 1971, *71*, 525–616.
23. Levitan, H., and Barker, J. L. Salicylate: structure-activity study of its effects on membrane permeability. *Science*, 1972, *176*, 1423–1425.
24. Nickel, A. R. Personal communication.
25. Oldendorf, W. H. Carrier-mediated blood-brain barrier transport of short-chain monocarboxylic organic acids. *Am. J. Physiol.*, 1973, *224*, 1450–1453.
26. Olin, T. B., and Redman, H. C. Experimental evaluation of contrast media in vertebral circulation. *Acta radiol.*, 1967, Suppl. 270, pp. 216–227.
27. Overton, E. Über die Osmotischen Eigenschaften der Lebenden Pflanzen und Tierzelle. Vierteljahresschr. Naturforsch. Ges. Zürich, 1895, *40*, 159–201.
28. Rall, D. P., Stabenau, J. R., and Zubrod, C. G. Distribution of drugs between blood and cerebrospinal fluid: general methodology and effect of pH gradients. *J. Pharmacol. & Exper. Therapy*, 1959, *125*, 185–193.
29. Rapoport, S. I. Evidence for reversible opening of blood-brain barrier by osmotic shrinkage of cerebrovascular endothelium and opening of tight junctions: relation to carotid arteriography. In: Small Vessel Angiography: Imaging, Morphology, Physiology and Clinical Applications. S. K. Hilal, Editor. C. V. Mosby Company, St. Louis, 1973, p. 137.
30. Rapoport, S. I., Hori, M., and Klatzo, I. Testing of hypothesis for osmotic opening of blood-brain barrier. *Am. J. Physiol.*, 1972, *223*, 323–331.
31. Rapoport, S. I., Brightman, M. W., and Reese, T. S. Reversible osmotic opening of blood-brain barrier (BBB) by opening of tight junctions of cerebrovascular endothelium. *Abstracts, 17th Annual Meeting Biophysical Soc.*, 1973, *13*, 230a.
32. Rapoport, S. I., and Levitan, H. Dependence of neurotoxicity of x-ray contrast media on lipid solubility. *Abstracts, 11th Annual Meeting Am. Soc. Neuroradiology*, 1973, p. 4.
33. Rapoport, S. I., and Thompson, H. K. Osmotic opening of blood-brain barrier in monkey without associated neurological deficits. *Science*, 1973, *180*, 971.
34. Rapoport, S. I., Thompson, H. K., and Bidinger, J. M. Equi-osmolal opening of blood-brain barrier in rabbit by different x-ray contrast agents. *Acta radiol.* (Diag.), 1974, *15*, 21–32.
35. Reese, T. S., and Karnovsky, M. J. Fine structural localization of blood-brain barrier to exogenous peroxidase. *J. Cell. Biol.*, 1967, *34*, 207–217.
36. Tindall, G. T., Kenan, P. D., Phillips, R. L., Margolis, G., and Grimson, K. S. Evaluation of roentgen contrast agents used in cerebral arteriography: II. Application of new method. *J. Neurosurg.*, 1958, *15*, 37–44.
37. Tornell, G. Bradycardial reactions in cerebral angiography induced by sodium and methylglucamine iothalamate (Conray). *Acta radiol.* (Diag.), 1968, *7*, 489–501.
38. Wallingford, V. H., Decker, H. G., and Kruty, M. X-ray contrast media: I. Iodinated acylaminobenzoic acids. *J. Am. Chem. Soc.*, 1952, *74*, 4365–4368.
39. Weast, R. C. Editor. Handbook of Chemistry and Physics. Fifty-first edition. Chemical Rubber Company Press, Cleveland, 1970.

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EDITORIALS

REPORT OF THE COMMITTEE ON CONTRAST MEDIA OF THE INTERNATIONAL SOCIETY OF RADIOLOGY

THE XIII International Congress of Radiology (Madrid, Spain) provided the setting for the *Committee on Contrast Media of the International Society of Radiology* to hold its first worldwide committee meeting. Attending were committee members from America, England, Europe, Australia, etc. This meeting was important since previous gatherings of the Committee, limited to the American contingency, had taken place in the United States during the annual meetings of the American Roentgen Ray Society or the Radiological Society of North America.

Dr. William H. Shehadi, Chairman of the Committee on Contrast Media (CCM), called the Madrid meeting to order on Monday, October 15, 1973, and reported on the recent and past activities of the CCM. He indicated that the CCM had received 100,000 standardized forms which represented completed pilot study questionnaires on adverse reactions to contrast media examinations. Contrast media injections were performed in participating radiological departments from several nations and contained information as to patient age and sex, type of examination (urography, cholangiography, angiography, lymphography, etc.), dose and type of contrast medium, history of allergy, results of previous examinations, types of premedication, results of pretesting, occurrence and type of adverse reactions—their treatment and the outcome of the reaction. Of the 100,000 finished questionnaire reports, computer tabulated data on 65,000 studies formed the basis of the CCM report (1) presented to the Congress members during the symposium on contrast media in the scientific program on Friday. This presentation highlighted the symposium, established the efficacy of CCM and reinforced the need for an international body capable of traversing national boundaries in the accumulation of relevant information on contrast media and on any given contrast medium available for use within any nation.

The CCM, conscious of the need to obtain larger statistics, resolved to broaden and strengthen its present program by: (1) establishing a multinational reporting on a simplified form (especially to register severe, fatal, and other major nonfatal adverse reactions); and (2) utilizing special report forms for specific subprojects within the over-all larger study. In the meantime, the present longer form will be continued until 250,000 intravascular contrast media injections have been tabulated and analyzed by the computer. The present longer form is to be retained for use when new products are evaluated in the future.

On Wednesday evening another important meeting took place. This was attended by members of the CCM and representatives of the worldwide industry (pharmaceutical companies) manufacturing contrast media to discuss interests and problems of mutual concern. Dr. Shehadi opened the session by declaring that "the members of the CCM are very willing and most anxious to provide help or information to the companies which they might find useful in the development and manufacture of the ideal (completely safe) contrast media."

Similarly, the Chairman conveyed to the industry the urgent need to redesign labels

on packages and vials as well as the package inserts since the present method places the radiologist, other physicians, and the patient at a disadvantage. Particularly, there is a need to: (1) review the *wording of indications-contraindications* in the use of contrast media; (2) *standardize the nomenclature* to provide a uniform expression of contrast media-concentrations in milligrams per ml. or percentage of product (especially as the same product appears in different countries); (3) provide *uniformity in conveying the iodine content, percentage and type of ion and cation* and the quality control of manufacturing methods; (4) to provide a *clear designation of expiration date* on all vials and packages; (5) provide for *standardization of information on new products* regarding chemical, physical, pharmaceutical, and clinical testing data; (6) *create uniform criteria for the preclinical evaluation-testing* of contrast media; and (7) to *report preclinical testing results on each organ structure* (brain, heart, liver, kidney, etc.) as well as large total body perfusions.

Following the professional meeting between industry and CCM a pleasant cocktail hour and dinner-meeting, hosted by Bracco Industria Chimica of Milan, Italy, was enjoyed by all the participants through the generosity of Bracco's Board Chairman, Dr. Fulvio Bracco. The evening arrangements were effectively handled by Dr. Bonati, Bracco's President.

Presently, the CCM continues to pursue its policies, being engaged in the prospective accumulation of data on adverse reactions to contrast media, in the establishment of appropriate subcommittees (prevention and treatment of reactions, package nomenclature, etc.) and establishing an international center for a *register on contrast media* which will receive reports, record data, tabulate adverse reactions and collect and evaluate all publications concerned with the clinical and experimental use of contrast media. This center could provide an effective working relationship with the manufacturing companies as well as the federal drug administrative bodies of all countries in the world, since the same product(s) appears in different countries under varying trade names.

Finally, while the CCM acknowledges its past financial support (International Society of Radiology, pharmaceutical industry, and others), it encourages and needs accelerated professional and economical sustenance on a continuous basis from worldwide industry, the International Society of Radiology, and other interested parties and organizations including the national radiological societies of all countries.

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REFERENCE

1. SHEHADI, W. H., and MISHKIN, M. M. Preliminary report of the Committee on Contrast Media of the International Society of Radiology; Annual Proceedings of the XIII International Congress of Radiology (Excerpta Medica) (In Press).



J. PAUL MEDELMAN, M.D.
1905–1974

DR. J. PAUL MEDELMAN, the 66th President of the American Roentgen Ray Society (1965), died July 6, 1974. Paul was born in Crary, North Dakota, February 10, 1905, graduated from Rush Medical School and had his internship and radiology residency training in the twin cities of Minneapolis and St. Paul. Except for a short period in Lubbock, Texas and 4 years (1942–1946) in the army medical corps he spent his entire professional life in St. Paul where he was Chief of the Radiology Department in St. Joseph's Hospital from 1936 to the time of his death. Paul also was senior member of a 17 man group of private practice radiologists who serve the twin city area.

Paul was a true general radiologist with superior capabilities in both radiation therapy and diagnosis. Early in his career he and his original partner, Dr. Edward Schöns, installed one of the first million volt therapy machines in the country and for the last 12 years of his life he limited his practice to therapy. Between these two periods Paul became a superb diagnostic

radiologist with special interest in chest diseases. National recognition of his diagnostic ability came in the form of election as an American Medical Association delegate to the AMA Section on Diseases of the Chest and election to the American Board of Radiology as a Trustee and regular examiner. Professional competence was also indicated by his long standing appointment as Clinical Professor of Radiology at the University of Minnesota Medical School. Paul was most enthusiastic about this assignment and devoted a large amount of time to the teaching of interns and residents.

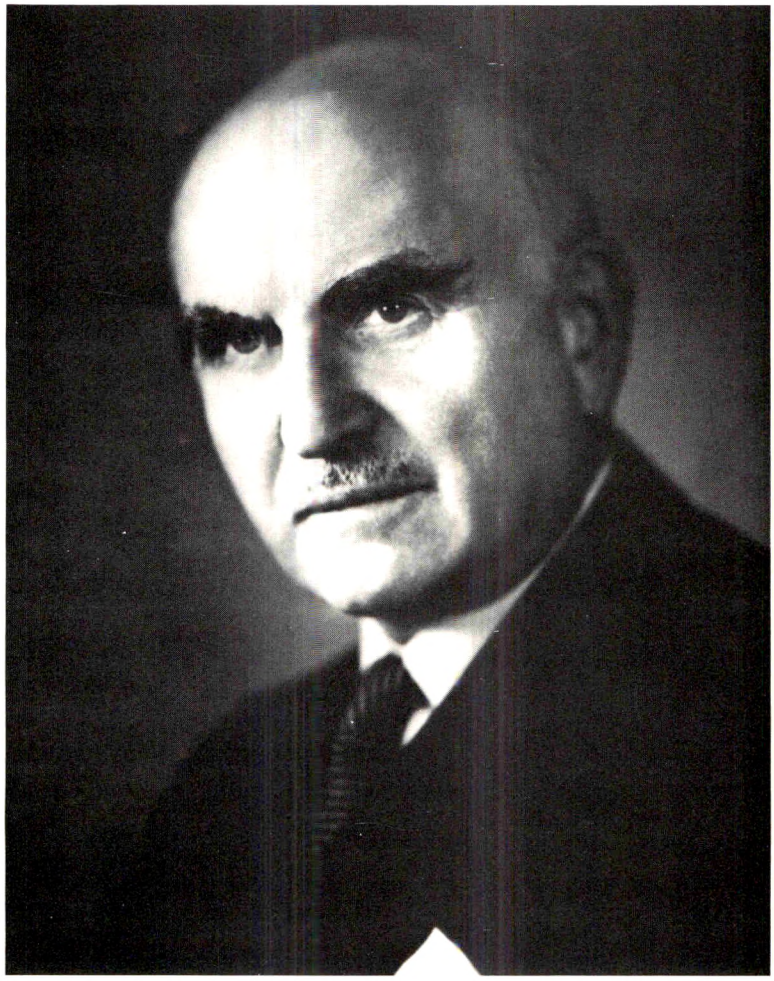
Paul was a most sincere gentleman, always considerate of others and always willing to listen. He had an uncommon ability to simplify complicated matters and apply common sense to the solution of difficult problems. These characteristics plus the ability to be an unassuming leader led to a large number of professional assignments and honors. Some of these are: President Ramsey County Medical Society, 1952; President Minnesota State Radiological Society, 1956; President Minnesota State Board of Medical Examiners, 1956; President Minnesota State Medical Association, 1964; President American Roentgen Ray Society, 1965. His executive ability was clearly demonstrated by eleven years (1960–1971) of service as Secretary of the Minnesota State Board of Medical Examiners. For notable contributions to medical science and outstanding service to its organization it was only natural that the Minnesota State Medical Association in 1968 chose him for its highest honor—The Distinguished Service Award.

Paul was not a "joiner," he never sought titles or honors, but his deep sense of obligation to the medical profession and his specialty made him willing to serve when called on. Nationally, the American Medical Association benefitted from his contributions to the Section on Diseases of the Chest and he served as Vice-Chairman and then Chairman of the AMA Section on Radiology. With enthusiasm and thoroughness he carried out several committee assignments from the American College of Radiology.

This rare combination of professional excellence, leadership, unassuming greatness and willingness to serve brought Paul a great many friends. Those so privileged will always treasure the memories and all can look upon him as a man whose life was truly well spent.

Paul is survived by his wife Helen, two sons, John P., St. Paul, and James K., Denver, a sister, Katherine Medelman, Jordan, Minn., and 5 grandchildren.

CLYDE A. STEVENSON, M.D.
Spokane, Washington



MAURICE LENZ, M.D.
1890–1974

DR. MAURICE LENZ, Emeritus Professor of Clinical Radiology at the College of Physicians and Surgeons of Columbia University, died on January 4, 1974. In 1890, he was born in Kovno, Russia, and in 1907 emigrated to the United States. The College of Physicians and Surgeons conferred the degree of Doctor of Medicine upon him in 1913. Following a general rotating internship at the Lebanon Hospital in the Bronx, New York, Dr. Lenz was in general practice until 1917, at which time he accompanied the American Expeditionary Force to France as a Lieutenant in the Medical Corps.

At the conclusion of World War I, he became assistant dermatologist at Mt. Sinai Hospital, New York, and at the Vanderbilt Clinic performed radiation therapy for dermatologic conditions.

In 1921, he took his mother to Germany to be irradiated for cancer of the ovary. Her death from ovarian cancer helped persuade Dr. Lenz to change his specialty to radiation therapy. Consequently, he pursued a Fellowship in Pathology with Professor Aschoff at Freiburg University, in Germany, and later became a Fellow in

Radiation Therapy at the Institute Curie, in Paris, working with Monod, Regaud, and Coutard.

Completing his program at the Institute Curie, he returned in 1924 as Assistant Radiotherapist at Mt. Sinai. He continued in radiation therapy practice as Chief of Radiation Therapy at Montefiore Hospital, New York, until 1930. Dr. Lenz then assumed the position of Chief of Radiation Therapy at Presbyterian Hospital and Professor of Clinical Radiology at the College of Physicians and Surgeons of Columbia—posts which he held until 1955. During this period he also was Director of the Division of Cancer of the Department of Hospitals for the City of New York and Chief of Radiation Therapy at the Frances Delafield Hospital.

In 1955 he was made Professor Emeritus of Radiotherapy at Columbia University and, in 1968, received the Silver Service Medal of the University.

Dr. Lenz was President of the American Radium Society and of the New York Roentgen Society. In addition, he was a Life Member of the American Medical Association, the New York Academy of Medicine, the International Radiotherapy Club, and an Emeritus Member of the American College of Radiology.

Dr. Lenz received frequent recognition, including Honorary Fellowships in the Texas Radiological Society, the Fort Worth Radiological Society, the Royal Society of Medicine, the Nord. Foren for Med. Radiology in Sweden as well as honors from many Central and South American Radiological and Cancer Societies.

In 1959, the World Health Organization selected Dr. Lenz as a Visiting Professor to universities and hospitals in India, Ceylon, the Philippines, Hong Kong, Japan, and Russia. Writing of his trip to the U.S.S.R., he told with particular care and understanding the problems related to cancer patients in that country.

He was honored by the New York Cancer Society in 1966, and in 1972 the American Society of Therapeutic Radiologists paid tribute to him at the 14th annual meeting.

Dr. Lenz's contributions to the literature of cancer were broad and significant. In more than 100 publications he dealt with the treatment of cancer and, in particular, with malignancies of the head and neck and of the breast.

His specific contributions related to descriptions of detailed, meticulous techniques for the treatment of head and neck cancer with special reference to careful analyses of the histology of the disease, to its natural history and spread, and to the end results of treatment. His publications on the natural history of breast cancer and the mechanisms for dissemination are hallmarks in understanding the disease.

A contributor to his specialty until his death, Dr. Lenz's acquaintance spanned workers in radiation therapy from the early days of the Curie Foundation to present investigators. His pioneering efforts influenced a generation of radiation therapists who were stimulated as much by his warm humanity as by his knowledge and dedication. Even in retirement, he continued to be concerned for the development of younger physicians by devoting hours to teaching and demonstrating appropriate radiation therapy techniques and to emphasizing the need for careful follow-up procedures.

Dr. Lenz exemplifies the best of our profession—a radiation therapist who bequeathes a legacy of intellectual honesty, knowledge, and commitment to a vast array of younger physicians at cancer centers throughout the world. To him, whose influence on many medical careers has been immeasurable, we are deeply indebted.

In his passing, we have lost a great friend, but all of us are richer for his having passed our way.

LUTHER W. BRADY, M.D.

NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

THE election of 6 new members and the re-election of 2 members to the National Council on Radiation Protection and Measurements (NCRP) was announced today by Dr. Lauriston S. Taylor, President of the NCRP.

The Council, successor to the National Committee on Radiation Protection and Measurements, was chartered by Congress in 1964 as a non-profit corporation to collect, analyze, and disseminate scientific information and recommendations about radiation measurement and protection against radiation.

Those newly elected at the Council's annual meeting were: William J. Bair, Ph.D., 50, Manager, Biology Department, Pacific Northwest Laboratories, Battelle Memorial Institute, Richland, Washington; Asher J. Finkel, M.D., 59, Director, Department of Environmental, Public and Occupational Health, American Medical Association, Chicago; Marvin Goldman, Ph.D., 46, Director, Radiobiology Laboratory, University of California, Davis; A. W. Guy, Ph.D., 45, Assistant Professor of Rehabilitation Medicine, Department of Rehabilitation, School of Medicine, University of Washington, Seattle; Seymour Jablon, M.A., 56, Statistician, National Academy of Sciences, Washington; and Chauncey Starr, Ph.D., 62, President, Electric Power Research Institute, Palo Alto, California.

The 2 members who were re-elected are Cyril L. Comar, Ph.D., 60, Head, Department of Physical Biology, Cornell University, Ithaca, New York; and Harry F. Schulte, M.S., 60, Group Leader, Industrial Hygiene, Health Division, Los Alamos Scientific Laboratory, University of California, Los Alamos, New Mexico.

For additional information, please contact: W. R. Ney, Executive Director, National Council on Radiation Protection and Measurements, 7910 Woodmont Avenue, Suite 1016, Washington, D. C. 20014.

NEWS ITEMS

DEPARTMENT OF HEALTH, EDUCATION AND WELFARE PUBLIC HEALTH SERVICE: FOOD AND DRUG ADMINISTRATION

The Food and Drug Administration announced a Federal Standard for diagnostic x-ray equipment effective August 1, 1974, at a press briefing held at the Bureau of Radiological Health, 10:00 A.M., July 30, Conference Room 416, 12720 Twinbrook Parkway, Rockville, Maryland 20852.

A statement about the standard was presented by Bureau of Radiological Health Director, John Villforth. Other representatives of the Agency were present to answer questions. A press release, copies of the statement, and other information about the standard were available at that time.

For further information, please contact: Michael E. W. Shaffer, Public Information Officer, Bureau of Radiological Health, Food and Drug Administration, 5600 Fishers Lane (HFX-25), Rockville, Maryland 20852.

CLINICAL APPLICATIONS OF THE GAMMA SCINTILLATION CAMERA INDIANA UNIVERSITY SCHOOL OF MEDICINE

A 3 day Workshop will be held September 5–7, 1974, in Indianapolis, Indiana.

The enrollment is limited to 30.

Further information may be obtained by writing to: Division of Postgraduate Medical Education, Indiana University School of Medicine, 1100 West Michigan, Indianapolis, Indiana 46202.

WORKSHOP AND DEMONSTRATION ON MOULD ROOM TECHNOLOGY

The Section on Radiation Therapy of the New York Roentgen Society announces a "Workshop and Demonstration on Mould Room Technology" by Mr. C. W. Dickens, Chief Mould Room Technician, The Royal Marsden Hospital, London and Surrey, England.

The same Workshop will be offered on the following dates: Workshop A—September 9–13, 1974 and Workshop B—September 23–27, 1974, at the Memorial Sloan-Kettering Cancer Center, Radiation Therapy Department (Room 205H).

For registration please write to: Mr. Louis Maddalone, Course Coordinator, Department of Radiation Therapy, Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York, New York 10021.

RADIOLOGY OF THE PEDIATRIC CHEST

This Symposium, sponsored by the Commission on Diagnostic Radiology, Division on Postgraduate Education Committee on ACR Symposia, American College of Radiology, will be held at Tampa, Florida, September 6, 7, 8, 1974.

Each participant will share a viewbox with a partner and have the essential roentgenograms at hand. The course will cover: Obstructing Lesions of the Airway; Inflammatory Disease; Systemic Diseases as Manifested in the Chest; Thoracic and Mediastinal Masses; and Conditions Peculiar to the Newborn Infant.

The faculty is headed by John A. Kirkpatrick, Jr., M.D., and Donald H. Altman, M.D., Co-Chairmen. They will be joined by Edward Singleton, M.D., and John Gwinn, M.D., for this first symposium.

Enrollment is limited to 200 registrations. All registrations will be accepted on a first come-first served basis.

Applications should be sent to: American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

RADIOLOGY OF THE GASTROINTESTINAL SYSTEM

Approved for Category 1 (15 Hours)

The Symposium, sponsored by the Commission on Diagnostic Radiology, Division on Postgraduate Education Committee on ACR Symposia, ACR American College of Radiology, will be held at Chicago, Illinois, October 18, 19, 20, 1974.

This gastrointestinal symposium empha-

sizes observation, analysis, and evaluation of basic radiologic features of barium studies of the GI tract—esophagus, stomach, duodenum, small bowel, and colon. Problems which are likely to be encountered by radiologists in their everyday practices will be featured and discussed in detail. Each diagnostic possibility for each case will be put into proper perspective and an estimate of the confidence level of each possibility will be made.

The faculty is headed by Bernard S. Wolf, M.D. Joining Dr. Wolf on the faculty are the following recognized experts on radiology of the gastrointestinal system: Drs. Thomas C. Beneventano, Arthur R. Clemett, Alexander R. Margulis, Richard H. Marshak, Morton A. Meyers, William B. Seaman, and Walter M. Whitehouse.

Registrations will be accepted on a first come-first served basis.

All registrations and inquiries regarding this Symposium should be sent to: The American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

A COURSE IN COMPUTERIZED TOMOGRAPHY OF THE HEAD

The Departments of Diagnostic Radiology and Biomedical Engineering of the Rush Medical College are offering a "Course in Computerized Tomography of the Head" to be held at the Medical School in Chicago on October 24th and 25th, 1974.

The course will consist of a presentation of the clinical uses of computerized tomography in the diagnosis of brain tumors, hemorrhages, hydrocephalus, orbital tumors, cerebrovascular occlusive disease, dementia and trauma.

Part of one day will be devoted to computer interaction with the scanner and there will also be lectures and panel discussions concerning recent technical advances in the field as well as the role of computerized tomography in future departmental planning.

The Faculty of the course consists of members of the Rush Medical College faculty as well as individuals from several institutions which have had at least a year or more of experience with this technique.

The Guest Faculty will include: Saul Aronow, Ph.D., Hillier Baker, M.D., David O. Davis, M.D., Romeo Ethier, M.D., David King, B.Sc., Paul New, M.D., Juan Taveras, M.D., Christopher Thompson, M.Sc.

Further information may be obtained by writing to: Michael S. Huckman, M.D., Director, Section of Neuroradiology, Rush-Presbyterian-St. Luke's Medical Center, 1753 West Congress Parkway, Chicago, Illinois, 60612.

RADIOLOGY OF THE SKELETAL SYSTEM

This Symposium sponsored by the Commission on Diagnostic Radiology, Division on Postgraduate Education, Committee on ACR Symposia, American College of Radiology will be held at Montreal, Quebec, Canada, November 8–10, 1974.

The Symposium will begin at 7:30 P.M. on Friday, November 8, and will end at about 2:00 P.M. on Sunday, November 10. A block of rooms has been set aside for participants; therefore, a hotel reservation card will accompany the notice of acceptance for this Symposium.

The Symposium has received AMA credit in Category 1 for the Physician's Recognition Award for 15 hours of credit. This credit may also be used for state and local organizations' requirements.

All registrations are on a first come-first served basis.

Applications should be sent to: American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

FORTIETH ANNUAL SCIENTIFIC ASSEMBLY OF THE AMERICAN COLLEGE OF PHYSICIANS

"Meeting Tomorrow's Clinical Needs Today" is the theme of the 40th Annual Scientific Assembly of the American College of Chest Physicians to be held at the Marriott Hotel—Rivergate Exhibition Center, New Orleans, Louisiana, November 3–7, 1974.

Scientific Program Chairman is John T.

Sharp, M.D., Professor of Medicine, University of Illinois School of Medicine, Chicago.

The meeting will feature nationally known physicians discussing such topics as "Long-Term Membrane Oxygenation," "Newer Methods of Cardiopulmonary Support" and "Lung Cancer—Current Overview." The Assembly will consist of 5 days of workshops, symposia, fireside conferences and the presentation of original investigations. Also highlighting the meeting are medical motion pictures, luncheon panels, evening seminars and tutorial sessions.

For further information, please write: Bradford W. Claxton, M.Ed., Director, Continuing Education, American College of Chest Physicians, 911 Busse Highway, Park Ridge, Illinois 60068.

HYPOXIA: CRITICAL CARE ASPECTS

Hypoxia, the common denominator of all critical illnesses, will be the topic of the national symposium for nurses and allied health professionals to be held Monday, November 4, 1974. This program is sponsored by the American College of Chest Physicians in association with their 40th Annual Scientific Assembly to be held in New Orleans, Louisiana, November 3–7, 1974. Course Chairman is Norma Shepard, R.N.

The subject of hypoxia will be introduced by a case presentation which will serve as a fulcrum for the program content. The program is designed to emphasize the role of each team member in their clinical approach to the care of the critically ill patient and the importance of the collaborative role of all members of this team.

Objectives of the program will be met throughout the day by a panel of experts that comprise the health care team. The assessments, interventions and projected role of each team member will be discussed following each formal presentation. Audience participation is encouraged during informal discussions.

Registrants are encouraged to attend the 40th Annual Scientific Assembly of the ACCP at the Marriott Hotel—Rivergate Exhibition Center in New Orleans, Louisiana.

For further information, please write: Bradford W. Claxton, M.Ed., Director, Continuing Education, American College of Chest Physicians, 911 Busse Highway, Park Ridge, Illinois 60068.

SPECIAL PROCEDURES IN DIAGNOSTIC RADIOLOGY: BASIC AND NEW INFORMATION

This Seminar, sponsored by the Department of Radiology at Mount Sinai Medical Center in collaboration with the Department of Radiology at Jackson Memorial Hospital, University of Miami School of Medicine, Miami, Florida, will be held January 12–16, 1975 at the Miami Beach Hyatt House (formerly the Playboy Plaza) in Miami Beach, Florida.

For further information, please contact Manuel Viamonte, Jr., M.D., Department of Radiology, Mount Sinai Medical Center, Miami Beach, Florida 33140.

MIDWINTER RADIOLOGICAL CONFERENCE OF THE LOS ANGELES RADIOLOGICAL SOCIETY

The Twenty-seventh Annual Los Angeles Midwinter Radiological Conference for 1975 will again be held at the Century Plaza Hotel, Los Angeles, California 90067, on January 31st, February 1st and 2nd of 1975.

Outstanding speakers will again be featured in Diagnostic and Therapeutic Radiology, as well as Nuclear Medicine and Ultrasound.

The small workshops which were very popular will be expanded for the coming meeting. The country's second largest Radiologic technical exhibit will be open for viewing on all 3 days.

An added feature this coming year will be a post-conference trip to Hawaii that will feature outstanding speakers as well as ample time for relaxation.

For further information, please write: Larry Kussin, M.D., Publicity Chairman.

BOOK REVIEWS

Human Radiation Biology. By Kedar N. Prasad, Ph.D., Associate Professor of Radiology; Chief, Section of Radiation Biology, University of Colorado Medical Center, Denver, Colo. Cloth. Pp. 524, with 162 illustrations. Price, $25.00. Harper & Row, Publishers, Inc., 2350 Virginia Avenue, Hagerstown, Md. 21740, 1974.

First, we would begin by stating that this is a reasonable attempt for a readable and useful text about Radiation Biology. It is not entirely about *Human Radiation Biology* (and rightly so) as the title would have you believe. In the Foreword it is stated that the importance and necessity of medical radiobiology stem from questions of genetic dangers to radiation exposure and a desire for more fundamental knowledge to use in treatment. Regarding the need for greater basic knowledge we would suggest that the fundamental premise would be to develop better means of *prevention* and *detection* of cancer.

The preface informs the reader that the text is for radiology and radiation therapy residents and medical students, but that it may be used as a reference book by others including physicians in general and technologists. In our way of thinking this is attempting to achieve too much.

The author is the chief of the section in radiation biology at a large medical center and, as such, has access to material one could classify as Human Radiation Biology. A review of the Table of Contents reveals that there are 23 chapters, of which the first 7 or about one-third are on topics which deal with cellular and subcellular phenomena. The other 16 chapters are either based entirely on non-human information or liberally sprinkled with non-human derived information. Radiation and Genetic Damage, Chapter 18, contains information gleaned from work with *Vicia faba* (beam roots), *Drosophila melanogaster* (fruit fly), and mice but no human work. It could not be otherwise. Radiosensitivity of human tumors, Chapter 20, is correctly documented with considerable nonhuman and *in vitro* material.

The first chapter details the basic principles that are presented in rapid-fire fashion. Later the reader becomes aware that each subject will be considered in meaningful detail. However, we feel that this should be emphasized and repeated at the outset.

Chapter 3 is a useful review, but is aimed somewhat low for the primary audience, the resident. In reality, residents most commonly enter the specialty with the depth of general knowledge that will allow them to begin at a level of sophistication greater than that characteristic of this chapter. Also, the bibliography at the end of the chapter is incomplete at best.

Chapter 4 again only gives a glimpse of the subject under discussion, but the reader would benefit from a review of the articles in the *New England Journal of Medicine* by Little.

The references for Chapter 6 appear complete and should be utilized by the reader. At the end of this basic section we would recommend a discussion of the subjects covered in Chapters 21–23 before moving to the more clinical aspects. Those subjects covered in Chapters 9, 10 and 11 might well be considered in a single complete chapter.

Regarding the remaining sections, too much emphasis is placed on such aspects as cataracts, nervous system response, etc. Perhaps the weakest chapter in terms of our bias is that written on the subject of Genetic Damage, which is really not up to recent interpretations and data. The chapter on Hazards to the Human Fetus does not properly utilize the work by the Oxford Group and other epidemiological studies. The answers that evolve from good epidemiological studies are of great moment to the group involved in radiation research as well as the general population.

There are a number of excellent texts on Radiation Biology, however, they all tend to have certain outstanding primary virtues, but are limited in scope. Hall's text has many of the virtues of this text and might be considered as an alternative. *Human Radiation Biology* is more of a listing of facts and data than an attempt to organize ideas and propose general concepts, but the presentation is generally clear and simply stated. We feel that because of the general scope of this text that it would be a useful addition to a personal or departmental library.

Timothy Merz, Ph.D.
A. Everette James, Jr., Sc.M., M.D.

MODERN THIN-SECTION TOMOGRAPHY. Edited by Arnold Berrett, M.D., Associate Clinical Professor of Radiology, New York Medical College, New York, N. Y.; Sam Brünner, Radiologist-in-Chief, Gentofte Hospital, Copenhagen, Denmark; and Galdino E. Valvassori, Professor of Radiology, University of Illinois College of Medicine, Chicago, Ill.; with a foreword by Sölve Welin, M.D., Professor of Diagnostic Radiology, University Hospital of Lund, Malmö, Sweden. Cloth. Pp. 337, with some figures. Price, $26.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1973.

The current status of tomography in clinical radiology is reviewed in this latest treatise on the subject. It is a multi-authored work by experts in the field, both in this country and abroad. The subject material covers historical aspects, physical principles, equipment and its application to the study of the skull, larynx, spine, lungs and mediastinum, and abdomen. The largest section is devoted to the temporal bone. The material covered is succinct and valuable, but its usefulness is impaired by the poor quality of the reproduction of the roentgenograms, so that the reader often cannot identify on the roentgenograms the structures which are illustrated in accompanying line drawings. One serious omission is the absence of any discussion on the usefulness of tomography in pneumoencephalography. A less serious omission is any reference to the use of the submental vertical view for study of the optic canal.

The bibliography is sufficiently broad to permit the reader to expand his fund of knowledge by appropriate further reading.

I believe that this book will find its greatest usefulness to the trainee and as a bibliographic source and, for this reason, that it would be a valuable addition to the library of training institutions.

THEODORE E. KEATS, M.D.

THE YEAR BOOK OF RADIOLOGY, 1974. Radiologic Diagnosis; edited by Walter M. Whitehouse, M.D.; associate editors, Joseph J. Bookstein, M.D., Trygve O. Gabrielsen, M.D., John F. Holt, M.D., William Martel, M.D., and John R. Thornbury, M.D. Radiation Therapy: edited by Howard B. Latourette, M.D.; associate editors, A. Curtis Hass, M.D., and Robert T. Guthrie, M.D. Cloth. Pp. 435, with some illustrations. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill. 60601, 1974.

With today's emphasis on continuing education and upgrading professional standards, the *1974 Yearbook of Radiology* becomes almost a necessity for the overburdened radiologist. It represents countless hours of review of world medical journals and the compilation and abstracting of the most important information pertinent to radiology during the past year.

Drs. Whitehouse, Latourette and their associate editors have continued the tradition of excellence acquired for the Yearbook over the past several decades. Their sage editorial comments are freely sprinkled throughout the text. There are frequent additional references for those who desire to pursue a topic further. The illustrations and photographs selected are of highest quality.

As in recent past editions, the selected abstracts are divided about equally between diagnosis and therapy. A few pages are devoted to isotopes in radiation therapy, and there are even some articles on diagnostic isotope procedures, such as lung scanning. Ultrasound, xeroradiography, electron radiography, and survey radiologic procedures are but a few of the topics reviewed under the heading of technical developments. It would be impossible to single out any specific article in the diagnostic or therapeutic sections for special mention, but all are concisely presented for rapid and interesting reading. This text is a valuable addition to the radiologist's personal reference library.

ARCH H. HALL, M.D.

BOOKS RECEIVED

PEDIATRIC NUCLEAR MEDICINE. Edited by A. Everette James, Jr., Sc.M., M.D., Associate Professor of Radiology; Director of Radiological Research, The Johns Hopkins University School of Medicine; Henry N. Wagner, Jr., M.D., Professor of Radiology; Director of Nuclear Medicine; Professor of Medicine, The Johns Hopkins University School of Medicine; and Robert E. Cooke, M.D., Vice Chancellor for Health Sciences; Professor of Pediatrics, University of Wisconsin; Formerly, Given Foundation Professor of Pediatrics; Director of the Department of Pediatrics, The Johns Hopkins University School of Medicine. Cloth. Pp. 544, with 378 illustrations. Price, $35.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

ANGIOGRAPHY IN INFANTS AND CHILDREN. Edited by Michael T. Gyepes, M.D. Cloth. Pp. 378, with

many illustrations. Price, $24.75. Grune & Stratton, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1974.

Basic Science Principles of Nuclear Medicine. Edited by Charles M. Boyd, M.D., Associate Professor of Radiology (Nuclear Medicine), and Medicine; Head, Division of Nuclear Medicine, University of Arkansas Medical Center; Chief, Nuclear Medicine Section, VA Hospital, Little Rock, Ark.; and Glenn V. Dalrymple, M.D., Professor and Chairman, Department of Radiology; Professor of Biometry, Physiology, and Biophysics, University of Arkansas Medical Center; Chief, Radiology Service, VA Hospital, Little Rock, Ark.; Professor of Medical Physics, University of Arkansas at Little Rock. Cloth. Pp. 272, with 253 illustrations. Price, $21.00. C. V. Mosby Company, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1974.

The Doctor's Lawyer: A Legal Handbook for Doctors. By Marc J. Lane, J.D., Member, Medico-Legal Relations Committee, Chicago Bar Association. Cloth. Pp. 99. Price, $12.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

Instrumentation in Nuclear Medicine. Volume 2. Edited by Gerald J. Hine, Veterans Administration, Washington, D. C.; and James A. Sorenson, Department of Radiology, University of Wisconsin Medical Center, Madison, Wisc. Cloth. Pp. 582, with some figures. Price, $39.50. Academic Press, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1974.

Practical Nuclear Medicine. Edited by Fuad S. Ashkar, M.D., Assistant Professor of Radiology and Medicine, University of Miami School of Medicine; Director of Nuclear Metabolic Section, Division of Nuclear Medicine, Jackson Memorial Hospital, Miami, and Mount Sinai Medical Center, Miami Beach, Fla. Cloth. Pp. 218, with many illustrations. Price, $25.00. Medcom, Inc., 2 Hammarskjöld Plaza, New York, N. Y. 10017, 1974.

Handbook for Radiologic Technologists and Special Procedures Nurses in Radiology. By Nieta Whitman Powell, R.N., Formerly, Clinical Instructor, Department of Medicine, Oklahoma University Health Sciences Center; Formerly, Supervisor, Institute for Cardiovascular Diseases, Good Samaritan Hospital, Phoenix, Ariz. Cloth. Pp. 105, with some figures. Price, $8.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

AMERICAN RADIUM SOCIETY
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Atlantic City, N. J., June 15–19, 1975.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following city during this year: Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1975.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1975 is September 30, 1974.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

The third examination is scheduled for Saturday, Sept. 21, 1974. Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017. The deadline for receipt of completed applications was June 1, 1974.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D.C. 20010. Annual Meeting.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 30–Nov. 4, 1974.

AMERICAN NUCLEAR SOCIETY
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545. Winter Meeting: Washington, D.C., Oct. 27–Nov. 1, 1974.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil in 1977.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Univ. of South Florida, Tampa, Fla.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Secretary General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland, Meeting: June 22–27, 1975.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters 2161 Taft Ave., Manila, Philippines.
Meeting: Manila, Philippines, Nov. 10–14, 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Mark M. Mishkin. The Graduate Hosp., Univ. of Pennsylvania, Philadelphia, Pa. 23rd Annual Meeting: San Diego, Calif., May 4–6, 1975.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Philip R. Shalen, Maj. MC, Departm. of the Army, Departm. of Radiology, US Army General Hosp., Landstuhl, APO New York 09180.

Blockley Radiological Society
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, Grant Hosp., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Alvin Segel, 3395 Scranton Rd., Cleveland, Ohio 44109. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Rodger W. Lambie, 701 E. 63rd St., Ste. 108, Kansas City, Mo. 64110.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Robert F. Conti, Holy Cross Hosp., Fort Lauderdale, Fla. 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, 716 Carrolton Blvd., West Lafayette, Ind. 47906.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce Saxe, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Calvin Williams, Valley Presbyterian Hosp., 15107 Vanowen St., Van Nuys, Calif. 91405. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 16, 1975.

NEW ENGLAND SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

NORTH AMERICAN SOCIETY OF CARDIAC RADIOLOGY
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Pablo Ramos, Box 1618, Bismarck, N. D. 58501. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 5301 F Street, Sacramento, Calif. 95819. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 175 S. Merkle Rd., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 36th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1975.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Stanley G. Seat, 476 Prospect St., LaJolla, Calif. 92037. Meets the first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broad-

way, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P. Q., Canada.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. James O. Richardson, Jr., 280 Mamaronek Ave., White Plains, N. Y. 10605. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociacón de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St.

Catherine St. West, Montreal 107, Que., Canada. Annual Meeting: Toronto, Ont., May 11–16, 1975.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincoln's Inn Fields, London WC2A 3PN, England.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting: Madrid, Apr. 24–26, 1975.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14–1151, Teheran, Iran. The Society meets on the second Saturday of each month. 5th Iranian Congress of Radiology, Apr. 12–17, 1975. Deadline for acceptance of papers, Jan. 20, 1975.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel. Nineteenth National Radiology Congress: Tel-Aviv, Israel, Nov. 6–8, 1974.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, Editor

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ROENTGEN DIAGNOSIS

Neck and Chest

Ogakwu, M., and Nwokolo, C. Radiological findings in pulmonary paragonimiasis as seen in Nigeria: a review based on one hundred cases. *Brit. J. Radiol.*, Sept., 1973, *46*, 699–705. (From: Department of Radiology, Specialist Hospital, and Department of Medicine, University of Nigeria, Enugu Campus, Enugu, Nigeria.)

Paragonimiasis in humans usually involves the lungs, and is the result of eating improperly cooked, infested, crustaceans. Pulmonary abscess cavities or foreign body granulomata result from the presence of the ova, and the ova occasionally evoke a nonspecific inflammatory reaction. The adult worms, about 5×5 and 12 mm. in size, occasionally encyst in the nervous system, muscles, and abdominal viscera. The ova are passed in the sputum or feces, develop in certain snails, then pass in another form to crabs and crayfish which carry the disease to man, as well as to dogs, cats, and in Asia, tigers and mongooses.

Clinically the infested patient may look healthy, is usually young, and complains of hemoptysis. Epilepsy or hemiplegia may announce nervous system involvement. Pulmonary complications include empyema; abscesses in the abdominal viscera or in muscle may occur. Specific chemotherapy is usually successful. Paragonimiasis exists in Asia, South America, and Africa, and is endemic in Cameroun and Nigeria.

One hundred patients, whose sputum contained the ova of *Paragonimus* and did not contain tubercle bacilli, were subjected to chest examination. Of these, 21 presented no abnormality in the film studies. Among the rest, the pulmonary densities were nonspecific, occurred in any portion of the lung, and were usually quite inconspicuous. One-third exhibited poorly defined "cottonwool shadows" and occasionally these were accompanied by linear densities, as they cleared. Some of the conglomerate densities excavated, producing thin walled cystic structures, resembling bubbles. One to 4 cysts developed in a given conglomerate mass, and each of the cysts was usually between 4 to 20 mm. in diameter. Various types of densities may develop at the same time in 1 patient. Although none of these lesions is entirely characteristic, the presence of inconspicuous, streaky or homogeneous densities, with development of bubble-like cavities, in the presence of rusty or chocolate sputum in a patient in an endemic region permits the diagnosis to be made with confidence.

Differentiation from tuberculosis can be suggested from the roentgenogram. Paragonimiasis usually involves the midregion of the lung, as opposed to tuberculosis in the apices. Paragonimiasis much more frequently is confined to 1 lung. The cavities in paragonimiasis are thin walled, multiple, and contain no fluid. Finally, the lesions change much more rapidly in paragonimiasis. Complete clearing may occur in 2 or 3 months, after institution of specific treatment.—*E. Frederick Lang, M.D.*

de Coster, A., Sergysels, R., Degaute, J. P., Jaspar, N., and Englert, M. Le concept du poumon de choc. (Concept of shock lung.) *Lille méd.*, April, 1974, *19*, 424–430. (Address: Dr. A. de Coster, Département de Pneumologie, Hôpital Universitaire Saint-Pierre, 322, rue Haute, 1000 Bruxelles, Belgium.)

As advances in the treatment of shock in Shock Centers have improved the immediate outlook in affected patients, longer term consequences have become more frequent. In particular, the entity of "shock lung" (SL) has emerged.

Eleven patients, mostly without prior lung disease, developed the SL pattern clinically. Shock was hypovolemic, toxi-infectious, or both. In 9 patients disseminated intravascular coagulopathy was associated. Interestingly, only the youngest patient, 22 years old, and the 2 patients over 65 did not survive SL—a good result for the ominous condition.

There are 3 phases to SL: (1) shock, responding to standard measures, especially giving fluids of high osmotic gradient; (2) transient improvement, up to 48 hours, followed by hypoxia and hyperventilation; and (3) deterioration, with polypnea, cyanosis, and severe hypoxia refractory to increasing oxygen artificial ventilation. During this third phase the roentgenogram rapidly develops an increasingly confluent bronchopneumonia pattern, up to total lung opacification except for air bronchogram. If the patient survives, this roentgenographic pattern clears.

Two of the 8 survivors in this report were treated during SL with positive end expiratory pressure (PEEP) ventilation, with remarkable increase (improvement) in the arterial oxygen partial pressure. Follow-up on 5 survivors revealed moderate dyspnea on exertion, and a diffusion capacity no more than 70 per cent of normal.

In terms of pathophysiology, SL results from an alteration of capillary endothelium permeability, yielding a protein-rich interstitial edema, including fibrinogen degradation products. As a consequence, there is a change in alveolar cells, especially Type II (surfactant producing pneumocytes). Surfactant loss causes alveolar atelectasis, which may explain the efficiency of PEEP in countering SL. Otherwise, the use of intensive oxygen causes extensive interstitial fibrosis.

This same pathophysiologic pattern develops regardless of the reason for the permeability change, whether SL, "pump lung," "wet lung," or multiple

fat embolism. However, the syndrome is clearly not the same as usual cardiogenic pulmonary edema, in which there is transudation of liquid, and in which conventional intermittent positive pressure is effective.—*Alan E Oestreich, M.D.*

Turiaf, J., Battesti, J. P., and Lehner, J. P. La sarcoïdose de l'enfant. (Sarcoidosis in childhood.) *Presse méd.*, May, 1974, *21*, 1351–1354. (Address: Pr. J. Turiaf, Chaire de Pathologie respiratoire, Hôpital Bichat, 170 bd Ney, F 75877 Paris Cedex 18, France.)

Since many of the cases of sarcoidosis that are known in childhood are discovered only incidentally on routine chest roentgenography, the reported incidence may well be an underestimate. Only 3 per cent of the cases of sarcoid encountered in 18 years by the authors were children; 11 of these 14 patients were found on routine screening. The ages were 9–15 years Two patients presented with parotid swelling and 1 with hemoptysis.

On the chest roentgenogram, all 14 had large bilateral mediastinal lymph nodes, and 10 had reticulonodular pulmonary infiltrates. Clinically, 10 had peripheral lymphadenopathy; 2, cutaneous lesions; and 2, splenomegaly. Skin test for tuberculosis was negative in all 14, despite previous BCG or tuberculosis in several. Four of 9 had hyperglobulinemia; 1 of 10 had hypercalcuria and none had hypercalcemia. Kveim test was positive in 8 of 9 cases.

Steroids were considered necessary in 7 children. Of these, 3 relapsed, including 1 child progressing rapidly to pulmonary fibrosis. These 3 children were among the 6 affected children of biracial Caribbean origin.

The subject of sarcoidosis should possibly be a consideration of routine roentgenography in schoolchildren.—*Alan E. Oestreich, M.D.*

Caron, J., L'Hoste, Ph., Dauver, A., and Caron-Poitreau, C. Les anéurysmes artério-veineux pulmonaires. (Pulmonary arteriovenous aneurysms). *J. de radiol., d'électrol. et de méd. nucléaire*, April, 1974, *55*, 309–316. (Address: Pr. J. Caron, Service d'Electroradiologie B, C.H.U., F 49000 Angers, France.)

Although arteriovenous aneurysms (AVA) of the lesser circulation have been rare, more cases are being recognized as roentgen investigation has become more sophisticated. Most examples appear to be congenital. Perhaps half of the patients affected have Osler-Rendu-Weber disease. Symptomatic patients show exertional dyspnea and cyanosis, often during childhood. Polycythemia and reduced arterial oxygen pressure may be present. When AVA is identified or suspected in the lungs, pulmonary angiography should be done.

There are four case presentations.

1. In a 35 year old asymptomatic woman 2 left lower lung AVAs were noted on routine chest roentgenogram. Tomograms and angiograms confirmed AVA. Hematocrit was 57 per cent. Successful surgical removal without sequelae.
2. A 59 year old man, with left ventricular failure, probably secondary to systemic hypertension had 2 AVAs on the chest roentgenogram and angiograms. Hematocrit was 65 per cent. Surgery not considered feasible.
3. In a 46 year old man, with marked right lung saccular bronchiectasis, a right hilar density was shown *via* pulmonary angiography to be an AVA fed by a relatively small right pulmonary artery, with no supply from the large right bronchiolar artery. Surgery advised, but refused.
4. A 13 year old boy with cyanosis, dyspnea on exertion, clubbing, and repeated pulmonary infections, had a systolic murmur. Hematocrit was 60 per cent. Arterial oxygen pressure was 40 mm. Hg. On chest roentgenography the cardiovascular silhouette was normal; catheterization showed no intracardiac shunt. A bilateral reticulonodular infiltrate on a plain roentgenogram was found to represent disseminated AVAs throughout both lungs, much too numerous to be resected.

None of these 4 patients showed non-pulmonary manifestations to suggest Osler-Rendu-Weber disease.

Whenever feasible, elective surgery to remove AVAs is recommended.—*Alan E. Oestreich, M.D.*

Westcott, Jack L., and Cole, Solon R. Interstitial pulmonary emphysema in children and adults: roentgenographic features. *Radiology*, May, 1974, *111*, 367–378. (From: The Departments of Radiology and Pathology, Hartford Hospital, Hartford, Conn.)

Interstitial pulmonary emphysema (IPE) as a cause of pneumomediastinum and pneumothorax has been widely recognized ever since the classic experimental and clinical observations of Macklin in 1939. Although IPE is common beyond infancy, there have been very few reports describing the roentgenographic features in adults and older children. The authors report the roentgenographic and pathologic features in 12 patients with autopsy-proven IPE.

IPE may occur when the pressure or relative distention of the alveoli exceeds that in the adjacent vascular bed and perivascular connective tissue. The rupture occurs in the walls of the alveoli situated immediately adjacent to the pulmonary vessels and interlobular septa. The air dissects along the perivascular sheaths and septa centrally to the lung hilus and peripherally toward the pleura. Continued dissection may produce subpleural air cyst formation,

pneumomediastinum, pneumothorax, and spread of air into the neck, chest wall, and connective tissue planes of the abdomen. A wide variety of clinical and physiologic events can precipitate IPE. A partial listing includes atelectasis, pneumonia, coughing, straining, chest trauma, forceful inhalation during anesthesia or resuscitation, positive pressure breathing, hyaline membrane disease, decompression from depths, and various air block syndromes, such as bronchiolitis, asthma, and bronchial plug or foreign body. Pathologically IPE is characterized by the presence of clefts, bubbles, and cystic air spaces in the interstitial connective tissue.

In many, and probably most cases, IPE is not extensive, is quickly reabsorbed, and is probably of no pathologic significance. However, when it becomes extensive, IPE and pneumomediastinum may compress and displace the heart, great veins, and pulmonary vessels. Cardiac output may be diminished, and dyspnea, cyanosis, and pulmonary edema may occur.

IPE is often difficult or impossible to see roentgenographically against a background of normal aerated lung, but may become visible in adults in those conditions in which the lungs become radiopaque. The direct signs of IPE are subpleural air cysts and direct visualization of the interstitial air collections, including visualization of perivascular air.

IPE characteristically changes rapidly. In some patients, it diminished or disappeared completely within 1 to 2 days. In others, it became more extensive and often progressed to subpleural air cyst formation, pneumomediastinum, or pneumothorax. If interstitial air collections and subpleural air cysts become secondarily infected, they will persist with very little change for days or even weeks.

Once IPE is recognized, the patient should be watched even more closely for the development of untoward sequelae such as cardiovascular embarrassment, pneumothorax, and secondary infection. Recognition of IPE helps to identify one of the confusing and changing patterns seen in a complicated group of patients with "shock lung" syndrome, and diffuse pneumonias, edema, or hemorrhage. It often explains the apparent paradox of roentgenographic "improvement" coinciding with clinical deterioration which occurs in some patients. Extensive IPE may make the lung appear better aerated, but interstitial air causes further impairment of the lung.

The authors conclude that IPE is not rare. Localized areas of IPE are common and are roentgenographically visible at some time in many patients receiving positive pressure breathing and in those who develop pneumomediastinum from other causes. It is especially common in patients in whom "adult respiratory distress" or "shock lung" syndrome develops. —*E. F. Schoolar, M.D.*

Fennessy, John J., Lu, Chien-tai, Variakojis, Daina, Straus, Francis H., and Bibbo, Marluce. Transcatheter biopsy in the diagnosis of diseases of the respiratory tract: an evaluation of seven years' experience with 693 patients. *Radiology*, March, 1974, *110*, 555–561. (Address: John J. Fennessy, M.B., Department of Radiology, The University of Chicago Hospitals, 950 East 59th Street, Chicago, Ill. 60637.)

Seven years' experience with 693 patients is presented by the authors. The technique and indications for endobronchial brush biopsies are reviewed. In brief, the indications are:

1. Pulmonary lesion of suspected malignancy beyond the reach of the bronchoscopist
2. Slowly or nonresolving pneumonia
3. Nonspecific infiltrate in a patient in an immune-deficiency state.

Contraindications for endobronchial transcatheter biopsy according to the authors are:

1. Absolute contraindications
   A. Recent severe hemoptysis
   B. Suspected vascular lesion
2. Relative contraindications
   A. Thrombocytopenia or other bleeding diathesis
   B. Severe dyspnea at rest.

The results of this series can be summed up by the authors' statement of "the combined accuracy of cytology and histopathology in diagnosing primary pulmonary neoplasm was 76 per cent." The authors indicate that the use of the steel bristle brush and the small forceps enables them to obtain sufficient tissue for histopathologic examination. The technique of brushing has been of little use in bacteriologic diagnosis and establishment of organisms in routine pneumonias. The results have been rewarding, however, in the diagnosis of opportunistic agents in immune-deficiency situations It is of note that of the neoplastic states evaluated, metastatic lesions were diagnosed least often, as a positive brushing was obtained in only 53 per cent or in 16 out of 30 such cases.

An extensive bibliography is available for those interested in considering the technique or in refreshing one's information.—*J. P. Eberts, M.D.*

Schoenbaum, Stephen W., Koerner, Spencer K., Ramakrishna, Byranna, and Goldman, Martin L. Transbronchial biopsy of peripheral lesions with fluoroscopic guidance: use of the fiberoptic bronchoscope. *J. Canad. A. Radiologists*, March, 1974, *25*, 39–43. (Address: Stephen W. Schoenbaum, M.D., Department of Radiology, Albert Einstein College of Medicine, Montefiore Hospital and Medical Center, Bronx, N. Y.)

Histologic diagnosis of peripheral pulmonary lesions has been attempted by means of obtaining tis-

sue through a needle, by bronchial brushing, and by examination of sputum cytology. None of these methods is entirely satisfactory. A small biopsy forceps has been devised which will pass through a fiberoptic bronchoscope. The location of the jaws of the forceps can be monitored by fluoroscopy if the lesion is not visible through the bronchoscope, and thus material for examination can be obtained directly.

This procedure was carried out in 18 patients in whom bronchial brushing and bronchial washing were also used. The transbronchial biopsy material was more satisfactory for diagnostic studies, and gave a more consistently accurate picture of the process in question, than either of the other 2 methods. Of the 18 patients, 10 had primary bronchial carcinomas, and 8 were accurately classified. In the other 2 patients, the lesion was close to the pleural surface, where use of the forceps is less safe, and material near the lesion was obtained. Two patients had metastatic carcinoma in the lung, and both were accurately classified. Four patients had inflammatory disease, and this was properly diagnosed by biopsy in all 4. Two others were classified as having inflammatory lesions, and while this is consistent with the clinical picture, the true state has not been determined, and neoplasm has not been conclusively excluded, as yet.

There was no complication to the procedure. An addendum indicates that this study has been carried out 350 times in 160 patients, again with no indication of a complication, such as pneumothorax or significant bleeding. Some of the patients experienced hemoptysis, less than 10 ml., but this was less in amount and less frequently encountered than after bronchial brushing.

One disadvantage of the method is the radiation exposure to the patient and operator. Usually the fluoroscopic time is less than 5 minutes, but in the presence of a lesion near the pleura, it may reach 10 minutes.

The examination is relatively painless, safe, and can be performed on an out patient basis.—*E. Frederick Lang, M.D.*

Keiler, A., Staffen, A., and Syré, G. Zur Problematik der isolierten tumorförmigen Amyloidose der Lunge. (Problems in isolated tumor-like amyloidosis of the lung.) *Thoraxchirurgie, Vaskuläre Chir.*, April, 1974, *22*, 86–93. (Address: Dr. A. Keiler, II Chirurgische Universitätsklinik, Spitalgasse 23, A-1090 Wien, Austria.)

Amyloid was first recognized by Rokitansky in 1842, and was characterized in 1854 by Virchow, whose method for gross identification is still in use. Amyloidosis occurs in 4 forms:

1. Primary, idiopathic, atypical amyloidosis occurs uncommonly, without demonstrable underlying disease, and is seen in both diffuse and localized forms.

2. Secondary, typical amyloidosis is relatively frequent in chronic inflammatory processes, such as bronchiectasis and osteomyelitis, as well as in chronic infections such as tuberculosis and leprosy.

3. Amyloidosis occurs as a result of paraproteinemias, such as Waldenström's macroglobulinemia, and plasmacytoma, and this type accounts for as much as 10 per cent of all amyloidosis.

4. Isolated tumor-like amyloidosis is most rare, and the literature contains reports of only 33 cases of lung involvement of this type, of which only 11 were diagnosed during life.

A review of a total of 12 patients discloses that in almost half the disease was discovered by accident. Of the rest, some presented with hemoptysis, others with only cough. The roentgen appearance varied. In some patients there were several adjacent nodules, in some a solitary nodule, and in 1, an excavating lesion. The solitary nodule may be central or peripheral, sharply defined, or diffuse and irregular, lobulated or smooth. In a typical patient, in whom laboratory studies and pulmonary function studies were all normal, except for insignificant elevation of the $alpha_1$ and $alpha_2$ globulin fractions of the serum, there was a $4 \times 3.5 \times 2.4$ cm. lesion in the periphery of the lingula. This was successfully enucleated, without sacrifice of other lung tissue. Microscopically the waxy, friable mass consisted of amyloid deposited in and around the walls of the alveoli, bronchi, and blood vessels, with obliteration of most of the contained normal lung architecture. Granulation tissue surrounded the masses of amyloid, and the lesion contained islands of cartilage and bone. A fruitless search for diffuse amyloidosis included sternal puncture, rectal biopsy, examination of sputum and feces, and both electrophoresis and immunoelectrophoresis.

It is usually not possible to classify these slowly growing masses preoperatively, but enucleation, rather than lobectomy, in the presence of a peripheral lesion, is curative.—*E. Frederick Lang, M.D.*

Kinoshita, Fumio, Ushio, Teruo, Maekawa, Akira, Ariwa, Rokuro, and Kubo, Atsushi. Scintiscanning of pulmonary diseases with ^{67}Ga-citrate. *J. Nuclear Med.*, April, 1974, *15*, 227–233. (Address: F. Kinoshita, M.D., Department of Radiology, Ohkubo Municipal Hospital, 1-461, Nishiohkubo Shinjyuku, Tokyo, Japan.)

The authors summarize their experience in the use of 2 millicuries of Ga^{67}-citrate for lung scanning in 162 patients with a variety of pulmonary diseases. In 72 patients with primary carcinoma of the lung, all but 1 had positive scans with 62 of them being strongly positive. Eighteen patients with metastatic

pulmonary carcinoma had positive scans which included 1 with a tumor less than 2 cm. in diameter.

Strongly positive scans were found slightly more often for squamous cell carcinoma and undifferentiated carcinoma then for adenocarcinoma

The gallium scan also contributed useful information by indicating the presence of lymphatic spread into the mediastinal area, which was oftentimes found to be difficult to diagnose on a chest roentgenogram.

Inflammatory lesions including pneumonia, abscess, tuberculosis and sarcoidosis had more clearly positive scans during the acute stage of the disease than did carcinoma.

The article includes 13 figures of chest roentgenograms, scans and 2 tables.—*Merle K. Loken, M.D., Ph.D.*

ABDOMEN

MASTER, S. P., TRUSCOTT, D. E., TEMPLETON, A. C., and MIDDLEMISS, J. H. Neonatal necrotizing enterocolitis. *Brit. J. Radiol.*, Dec., 1973, *46*, 1063–1069. (From: Departments of Surgery, Radiology and Pathology, Mulago Hospital and Makerere Medical School, Kampala, Uganda.)

Idiopathic neonatal necrotizing enterocolitis is a serious disease which afflicts infants in the first 3 weeks of life, usually developing in the first 5 days, may have a fulminating, fatal course, and occurs usually in small, premature infants. The cause of the disease is unknown, but infection, intestinal ischemia, intestinal stasis and hypersensitivity to bacterial endotoxins have been suggested. Early diagnosis and treatment are of crucial importance for a favorable outcome. The incidence in Europe and United States is about 1–2 per cent, while in Uganda it was less, possibly because of lack of recognition following home deliveries.

In a group of 26 patients subjected to roentgen examination, all presented with lethargy, and 69 per cent were premature. Abdominal distention, vomiting, edema of the anterior abdominal wall, and either constipation or frankly bloody feces were common. In 21 there were perinatal complications leading to hypoxia or shock.

Examination of the abdomen by roentgenograms made with the patient recumbent and upright was carried out every 6 hours until the patient recovered or succumbed. The earliest sign is "veiling" of the abdomen with loss of the flank stripe. Soon there develops a granular or foamy appearance due to gas in the wall of the involved segments, and with this there are shown fluid levels in the upright roentgenograms. The pneumatosis is shown as a fine line paralleling the lumen of the intestine, and when the gut is shown axially, there are concentric rings. Frequently the next stage is perforation of the intestine with development of a collection of air along the peritoneal border in the flank or under the diaphragm. The appearance of gas in the portal venous system is, of course, ominous. In those patients who recovered, reduction of the distention occurred usually within 12 hours, but the pneumatosis occasionally persisted for as long as 4 days. The roentgen appearance returned to normal in successfully treated patients within 1 week.

Fifteen of the patients were subjected to autopsy. The most frequently involved site was the terminal ileum, but in 1 the entire gastrointestinal tract from the esophagus to the anus was necrotic. In all, the hemorrhagic surfaces of the ulcerated areas were covered by a yellowish, necrotic slough. The gangrene was sometimes superficial, sometimes full thickness; surprisingly, tissue near the regions of gangrene appeared normal, with few inflammatory cells. The mesenteric and intestinal vasculature seemed normal.

In the diagnosis and management of this disease, roentgen studies play a most significant part. Perforation may be shown before it is even suspected clinically. Early institution of conservative, supportive treatment, and frequent, serial roentgenograms are important in the management of these patients. —*E Frederick Lang, M.D.*

GRAY, ROBIN. Disappearing gallstones: report of 2 cases. *Brit. J. Surg.*, Feb., 1974, *61*, 101–103. (From: Plymouth General Hospital, Plymouth, Devon, England.)

The author describes 2 patients, 1 with radiolucent and 1 with radiopaque gallstones.

The patients adhered strictly to a fat free diet for from 6 months to 2 years following the diagnosis of cholelithiasis, and subsequent surgery discerned that the stones had disappeared. Disappearance was by dissolution or by passage into the intestinal tract without biliary colic.

The literature was reviewed and other instances of stone disappearance were found.

It is suggested that patients with roentgenographic evidence of cholelithiasis wait 3 months or longer and that repeat cholecystography be performed prior to surgery.—*Robert P. Andrews, M.D.*

GENITOURINARY SYSTEM

KOROBKIN, MELVYN, PICK, RUTH A., MERTEN, DAVID F., PERLOFF, DOROTHEE L., and PALUBINSKAS, ALPHONSE J. Etiologic radiographic findings in children and adolescents with nonuremic hypertension. *Radiology*, March, 1974, *110*, 615–625. (Address: Melvyn Korobkin, M.D., Department of Radiology, University of California School of Medicine, San Francisco, Calif. 94143.)

The definition, etiology and prognosis of sustained nonuremic hypertension are not as well established in children as they are in adults. In an attempt to

clarify these factors, and to document the spectrum of potential curable underlying disorders, the authors present a cooperative study of juvenile, nonuremic hypertension based on a review of the medical records at 3 major teaching hospitals. Only those patients with sustained hypertension were evaluated, and only those patients whose hypertension was first diagnosed at or prior to the age of 21 years were included. Transient hypertension, sustained hypertension with uremia, and coarctation of the thoracic aorta were excluded. A total of 61 patients are reported in whom a radiologically demonstrable abnormality was thought to be the cause of their hypertension.

There is no widely accepted definition of juvenile hypertension because average diastolic pressures increase with age during childhood. However, diastolic pressures greater than 90 mm. Hg are accepted as abnormal in both adults and children, and that value was used in this study, since all patients in this series presented diastolic pressures of more than 90 mm. Hg off therapy. The underlying disorders, with hypertension, fell into 3 broad categories: renal parenchyma abnormalities; renovascular disorders; and adrenal disease.

Thirty-nine of the children and adolescents with hypertension had renovascular abnormalities. Isolated renal artery stenosis was demonstrated radiographically in 30 of these patients; it was unilateral in 26, and bilateral in 4. In 22 of the 26 the stenosis affected the main renal arteries; in the other 4 it was confined to a segment of renal artery. Narrowing of the abdominal aorta was identified in the other 9 patients, 8 of whom had associated abnormalities of the renal artery. Additional information on all patients is presented in a tabular form. Despite the extensive research regarding renal artery stenoses and hypertension, the mechanism involved in this relationship remains obscure. Not all patients with significant renal artery stenoses are hypertensive, nor do all patients with renal artery stenosis and hypertension benefit from a technically adequate operation. How to define these patients with hypertension and renal artery stenosis who will benefit still remains a problem. Various differential function tests have been used, but these are technically difficult in small children. Presently, the ratio between the renin concentration in the 2 renal veins has been shown to have a highly predictive value in evaluating those patients for possible surgery. Unfortunately, most of these patients were evaluated prior to the availability of accurate and sensitive renin assays. Nevertheless, the surgical results in patients with isolated stenosis are encouraging since, of the 21 patients receiving surgery, the results were excellent in 18 and good in 2 patients, with 1 lost to follow-up. Surgical therapy is far less successful in those patients with associated abdominal aortic stenosis. Of these patients, 7 received surgery with 1 excellent and 1 satisfactory result.

Eighteen patients presented radiologic evidence of renal parenchyma disease. Chronic atrophic pyelonephritis was present in 5 patients, unilateral hydronephrosis in 5, post-traumatic subcapsular scar in 1, a renin secreting Wilms' tumor in 1, and there was a group of 6 hypertensive patients with a small artery feeding a small kidney, but without evidence of focal scarring. The etiology of the contracted kidney seen on the excretory urogram can be exceedingly difficult to determine since ischemic atrophy, postobstructive atrophy, chronic atrophic pyelonephritis and congenital hypoplasia can all appear similar radiographically. The authors' criteria for a diagnosis of chronic pyelonephritis was irregular focal atrophy associated with caliectasis. The relationship between hypertension and chronic pyelonephritis is not clear and that of unilateral hydronephrosis to hypertension is controversial. Perhaps renal vein renin levels will be informative and predictive of operative success. Of the 5 patients with unilateral hydronephrosis, 4 received excellent results following relief of ureteral obstruction. Similar excellent results, with postoperative normotension, followed nephrectomy for the subcapsular scar and the renin secreting Wilms' tumor.

Four patients had hypertension associated with an adrenal tumor. Pheochromocytoma was present in 3 patients; the tumor was intra-adrenal in 2 patients and extra-adrenal in 1 patient. The fourth patient was found to have a partially calcified aldosteronoma. Surgical excision produced a return to normotension in all patients.

The present study demonstrates the value of vigorous radiologic evaluation, including arteriography, of children and adolescents with unexplained systemic hypertension. The discovery of underlying disease of the renovascular system, renal parenchyma and adrenal gland permitted surgical therapy which resulted in cure or improvement of hypertension in a large number of patients.—*Edward B. Best, M.D.*

Maling, T. M. J., and Rolleston, G. L. Intra-renal reflux in children demonstrated by micturating cystography. *Clin. Radiol.*, Jan., 1974, *25*, 81–85. (From: The Department of Radiology, Christchurch Hospital and Clinical School, Christchurch, New Zealand.)

Intrarenal reflux or pyelotubular backflow associated with voiding cystourethrography was demonstrated in 16 children under the age of 5 years.

These children came from a series of 386 patients of all ages and both sexes, all of whom had vesicoureteral reflux without evidence of mechanical obstruction.

The technique used in voiding cystourethrography was with the patient sitting or supine, depending on whether they were female or male, respectively. Injection of the contrast material with a small bore catheter was carried out. The reservoir height was adjusted so as not to exceed the expected normal

range of bladder pressures and filling was discontinued if spontaneous dorsiflexion of the toes occurred or spontaneous micturition took place. Filling was also discontinued if there was no further ingress of fluid with the reservoir held at a height of less than 50 cm.

Anesthesia did not appear to influence the production of intrarenal reflux and it is felt by the authors that the finding correlates with subsequent development of scarring associated with chronic pyelonephritis at the site at which it occurs.

It is stressed that the quality of the roentgenograms obtained during the procedure was of significant importance in demonstrating the finding.—*Robert P. Andrews, M.D.*

COSGROVE, MALCOLM D., and MOWAT, PAUL. Evaluation of the xenon 133 renal blood flow method. *Brit. J. Urol.*, April, 1974, *46*, 137–147. (From: Department of Urology, Los Angeles County University of Southern California Medical Center, Los Angeles, Calif.)

The authors report the results in the study of renal blood flow in 22 patients. All patients were undergoing renal arteriography and at the completion of that study, xenon in saline was administered *via* catheter directly into the renal artery(s). The clearance of the xenon from the renal parenchyma was determined from a strip chart record which permitted fast and slow components of flow to be determined by "curve stripping." Patients with a variety of forms of renal pathology were included.

The authors state that the xenogram has not by itself saved any lives, but has not caused any increased morbidity and may be accomplished in approximately 10 minutes with essentially no additional radiation exposure. The usual injected dose was 500 microcuries of xenon 133.

In most categories of renal pathology, the authors feel that the xenogram provides useful information for managing individual cases.

The xenogram does have certain deficiencies which include its inability to demonstrate areas of total occlusion and its inability to distinguish renal tumors from benign cysts. It does permit quantitation of renal ischemia, in the diagnosis of renal vein thrombosis, in the prediction of recovery of renal function following relief of obstruction, and in the evaluation of possible renal transplant rejection.—*Merle K. Loken, M.D., Ph.D.*

BLOOD AND LYMPH SYSTEM

POCHACZEVSKY, RUBEM, MUFTI, MAHTAB A., LAGUERRE, JOSEPH N., BRYK, DAVID, KASSNER, E. GEORGE, RICHTER, ROBERT M., and LEVOWITZ, BERNARD. Arteriography of penetrating wounds of the extremities—help or hindrance? *J. Canad. A. Radiologists*, Dec., 1973, *24*, 354–361. (Address: R. Pochaczevsky, M.D., Department of Radiology, City Hospital Center at Elmhurst, 79–01 Broadway, Elmhurst, N. Y. 11373.)

Experience with 75 patients with penetrating injuries of the extremities showed that a significant hematoma formation, even without other clinical findings, was a fairly reliable sign of arterial injury.

A potential pitfall of arteriography was emphasized by experimental animal studies. Even though arteriography was shown to be reliable in clinical material, as well as in experimental transverse wounds, it was not accurate in defining longitudinal wounds with a fairly large number of false negatives occurring in the latter category. Significant hematoma formation, on the other hand, proved to be a consistently reliable indicator of the presence of both transverse and longitudinal wounds. Significant hematoma formation was also shown to be absent in experimentally produced venous and muscle injuries.

Arteriovenous fistulas may not be present initially, but may develop as a complication of untreated injuries.

Arteriography in penetrating wounds of the extremities would be most helpful and informative in the following circumstances: (1) when there is a history of preexisting arterial disease; (2) when associated fracture or massive muscle injury (*i.e.*, shotgun wounds) may contribute to significant hematoma formation; (3) when initial physical examination is delayed and a history of previous swelling (*i.e.*, "hematoma") is unreliable; (4) when, in the operating room or postoperatively, a diminished or nonpalpable distal pulse may be due to thrombotic occlusion or technical error; and (5) to demonstrate the extent of a thrombosed arterial segment if this information is indispensable for correct surgical management.

It is concluded that delay in the surgical repair of penetrating injuries of the extremities, when associated with significant hematoma formation, is not warranted in most cases.—*David Morse, M.D.*

RADIATION THERAPY

DIMOPOULOS, J., WICKENHAUSER, J., CANIGIANI, G., and WICKE, L. Das Ewing-Sarkom: Kritische Analyse von 41 Fällen. (Ewing's sarcoma: critical analysis of 41 cases.) *Radiol. clin. et biol.*, 1974, *43*, 56–70. (Address: Dr. J. Dimopoulos, Strahlentherapeutische Klinik und Institut für klinische Strahlenbiologie der Universität Wien, Alserstrasse 4, A-1090 Wien, Austria.)

Ewing's sarcoma of the bone represents a highly malignant tumor and occurs predominantly in childhood and adolescence. In spite of the malignant

nature, it also shows a great radiosensitivity. An attempt at a radical destruction of the tumor is indicated. Nevertheless, the prognosis remains very poor.

The diagnosis is based on roentgenographic, clinical, and pathologic findings. The clinical findings, comprising localized pain and swelling and subfebrile temperatures, are not specific. A histologic proof is always required. Needle biopsy is considered inadequate as the results cannot often be correlated with roentgen findings. Therefore, a surgical biopsy becomes necessary. Because of the threat of tumor dissemination, a preoperative prophylactic irradiation must be performed. The authors recommend a dose of 800 to 1,000 r immediately prior to the surgical procedure. The field of irradiation can be kept small in this instance.

Radiotherapy remains the method of choice as the survival rate of surgically treated patients has significantly decreased from 24.4 to 19.9 months. The affected bone has to be irradiated in its entirety with doses of 4,000 to 5,000 r given within a period of 4 weeks. Megavolt therapy has proved most effective. Simultaneous cytostatic medication is considered to be of little value. It should be reserved to cases with widespread dissemination. Solitary metastatic foci in bones and in the lung can still be treated with high energy electrons or radiocobalt.

The therapeutic results in 41 patients, observed during the past 17 years, are evaluated. Five cases have been deleted because of inaccurate diagnostic conclusions. Of the remaining 36 patients, 31 have died during the first 2 to 3 years, the shortest interval being 8 months. Two patients have survived 5 years. An early diagnosis has aided in improving the survival rate.

It is now believed that the disease affects not only a bone in its entirety, but that a multicentric involvement exists from the beginning. Therefore, 6 patients have more recently been treated with high energy electrons (30–42 mev.). There have been three survivals so far at intervals of 21, 31, and 39 months, respectively.—*Ernest Kraft, M.D.*

Martini, Nael, Huvos, Andrew G., Smith, Julius, and Beattie, Edward J., Jr. Primary malignant tumors of the sternum. *Surg., Gynec. & Obst.*, March, 1974, *138*, 391–395. (From: The Departments of Surgery, Pathology, and Radiology, Memorial Sloan-Kettering Cancer Center, New York, N. Y.)

Fourteen patients with primary malignant tumors of the sternum are reviewed. There were 8 chondrosarcomas, 1 fibrosarcoma, 1 osteogenic sarcoma, 1 angiosarcoma, 2 plasma cell myelomas, and 1 reticulum cell sarcoma.

All patients presented with a palpable mass and 7 patients had dull pain in the sternal area. The 8 patients with chondrosarcoma had coarse mottled calcification within the tumor. Tomography of the sternum and selective internal mammary angiography were performed in some patients. Only the angiosarcoma and 1 patient with multiple myeloma had an abnormal vascular pattern in the area of the tumor. The remaining tumors were avascular.

Wide surgical excision was performed in all the solid tumors of the sternum. The patients with myeloma and reticulum cell sarcoma were successfully treated with external radiation therapy.—*Ronald J. Ross, M.D.*

Archambeau, John O., Bennett, Gerald W., Levine, Gerald S., Cowen, Richard, and Akanuma, Atsuo. Proton radiation therapy. *Radiology*, Feb., 1974, *110*, 445–457. (Address: John O. Archambeau, M.D., Department of Radiation Therapy, Nassau County Medical Center, 2201 Hempstead Turnpike, East Meadow, N. Y. 11554.)

The well-defined range of protons in tissue and their characteristic energy distribution along this range (Bragg ionization curve) permit the therapist to confine the major fraction of the absorbed dose to a designated volume. As a consequence, protons should have a definite clinical usefulness. Either the dose to normal tissues can be reduced while giving the required dose to the designated tumor-containing volume, or the dose to the tumor-containing volume can be increased without exceeding the tolerance of the normal tissues.

At a recent conference on the application of protons for radiation therapy it was concluded that, although further preclinical investigation is required, a clinical trial is warranted. In view of the ability to increase the dose to a designated volume, it will be necessary to determine whether a dose-volume relationship exists. A clinical trial should then be set up to document whether the ability to increase the dose to a designated volume could result in increased control of a cancer (*i.e.*, cancer of the pancreas).

The primary purpose of this communication is to demonstrate the improved dose distribution obtainable with proton irradiation.

The characteristics of protons compared to those of cobalt gamma photons, fast neutrons, pions and heavy ions are neatly tabulated. The specific physical, technologic, biologic and clinical advantages of protons are presented in detail. A comparison of proton and x-ray treatment plans demonstrates the superiority of the dose distribution of the former by stressing the differential low dose to normal tissues as the key factor. This is done using treatment plans consisting of single field irradiation and parallel opposed (overlapped) field irradiation. Many isodose curves, schemata and tables illustrate these characteristics of proton irradiation. For example, conditions for which a lowered integral dose or decreased dose to normal tissue would offer particular advantages are listed as follows: children's cancers; Hodg-

kin's disease; paraaortic irradiation; obesity; severe illness; and re-irradiation.

The status of proton irradiation today is also discussed. Except for pituitary ablation and limited clinical application in Sweden, and Russia, full exploitation has not been realized. Obstacles to clinical use of protons include: limited availability of proton accelerators; difficulty in designating the tumor volume; difficulty in localizing the Bragg peak within tissues; preoccupation with other radiation sources; and clinical lethargy.

It is concluded that no advantage can be claimed for protons *per se*. Yet, the ability to confine the proton dose to a designated volume provides greater flexibility and resourcefulness for proton irradiation therapy than does x-ray therapy for dose delivery to the cancer.—*Paul M. Kroening, M.D.*

SCHMITZ, ROBERT L., CHAO, JEN-HUNG, and BARTOLOME, JUANITO S., JR. Intestinal injuries incidental to irradiation of carcinoma of the cervix of the uterus. *Surg., Gynec. & Obst.*, Jan., 1974, *138*, 29–32. (From: The Departments of Surgery and Radiology, Mercy Hospital and Medical Center, Chicago, Ill.)

Significant injury to the intestinal tract occurs in about 2 per cent of cases with carcinoma of the cervix or endometrium treated with irradiation.

If the exposure is in small dosages at long intervals and in a total amount less than 4,000 roentgens, the effects are not severe. There will be inflammation and edema of the intestinal wall with superficial ulceration, excess mucus production, hyperperistalsis, nausea, and diarrhea. Soon after treatment is stopped, these effects disappear, usually with little or no residue.

If the exposure is in larger dosages at shorter intervals, and the total amount exceeds 4,500 roentgens, the effects are proportionately more severe. The intestinal wall may be edematous, the mucosal ulcerations will be deeper and may bleed significantly. Peristalsis will be more active. The bloody diarrhea combined with moderate nausea and vomiting may be severe enough to require fluid and blood replacement. Necrosis of the intestine may occur with hemorrhage, sloughing, and/or perforation. An endarteritis often develops after the acute reaction to large doses of irradiation. This may lead to atrophy, scarring and, rarely neoplasia.

Factors increasing intestinal injury are: (1) type of bowel (small bowel being more susceptible than large bowel); (2) patient variability (light-complexioned patients are more susceptible); (3) immobile bowel (*e.g.* rectum or small bowel bound down with adhesions); (4) faulty factors in treatment plans leading to overexposed areas.

Thirty-seven patients with severe radiation injuries were treated. Injury occurred in the ileum 14 times, in the sigmoid 3 times, and in the rectum 20 times. The likelihood of injury is twice as great for patients with carcinoma of the cervix, as it is for patients with carcinoma of the endometrium. This is not only due to the close, fixed relationship between the cervix and the rectum, as 11 of these patients with carcinoma of the cervix had ileal injuries and another 3 had injuries to the rectosigmoid. The injuries were concomitant with the symptoms of radiation sickness in 10 patients. The others had a latent period lasting as long as 20 years.

The types of injury encountered included severe enterocolitis, local ulceration, stenosis, rectovaginal fistula, atrophy of the terminal ileum with spruelike picture, and latent neoplasia.

Treatment of the enterocolitis was with fluid and blood replacement. Occasionally, vigorous medical management of local ulcers was successful. In most instances surgical diversions or resections were the treatment of choice.—*Frederick P. Stitick, M.D.*

FREEMAN, J. E., JOHNSTON, P. G. B., and VOKE, J. M. Somnolence after prophylactic cranial irradiation in children with acute lymphoblastic leukaemia. *Brit. M. J.*, Dec., 1973, *4*, 523–525. (From: St. Bartholomew's Hospital, and Hospital for Sick Children, Great Ormond Street, London, England.)

Twenty-eight children with acute lymphoblastic leukemia in remission received cranial irradiation as part of central nervous system prophylaxis. Twenty-two of these children had a transient cerebral disturbance characterized by a varying degree of somnolence. All the patients had 2,400 rads delivered to the midplane of the cranium in 20 fractions given over 28 days. A similar amount of radiation was given to the spine in 7 patients. In 21 patients lower doses of radiation and intrathecal methotrexate were employed. The therapy of the spinal canal did not alter the frequency of postirradiation somnolence.

Somnolence was the major symptom. This varied from mild drowsiness to prolonged periods of sleep. Severe somnolence lasted for 3 to 7 days. Other symptoms lasted longer. Somnolence was often preceded and followed by several days of anorexia and irritability.

There was no evidence of raised intracranial pressure or focal neurologic abnormality in any affected child. No convulsions occurred. In every child the symptoms subsided spontaneously and completely. Normal levels of alertness and physical activity were fully re-established.

Although 22 children (79 per cent) developed the syndrome to some degree, in 11 (39 per cent) the symptoms were mild. In the other half they were severe. The younger children tended to have the more severe symptoms. Symptoms appeared after a latent period of 24 to 56 days following completion of cranial irradiation. Eighteen out of the 22 children

first developed symptoms during the fifth or sixth week after cessation of therapy. Symptoms lasted from 10 to 38 days. There was a suggestion that the patients, who developed symptoms earlier, tended to have a more prolonged course.

Five of the children had a lumbar puncture. In 1, the cerebrospinal fluid was normal, the others had varying degrees of mild elevation of protein and leukocytosis (monocytes). In 5 children an electroencephalography (E.E.G.) was performed. The electroencephalographic abnormalities were consistent with a diffuse cerebral disturbance, without focal abnormality. Repeat E.E.G.s showed improvement, correlating with the clinical recovery.

Although there is no clinicopathologic proof, the authors postulate that this syndrome is an encephalitis caused by suppressed myelin synthesis due to the irradiation. This syndrome should be regarded as a minor and relatively unimportant complication of cranial irradiation in children. It is a relatively small price to pay for reducing the incidence of meningeal leukemia.—*Frederick P. Stitik, M.D.*

RADIOISOTOPES

Grames, George M., Jansen, Carl, Gander, Martin P., Wieland, Howard C., and Judkins, Melvin P. Safety of the direct coronary injection of radiolabeled particles. *J. Nuclear Med.*, Jan., 1974, *15*, 2–6. (Address: George M. Grames, Division of Nuclear Medicine, Department of Radiology, Loma Linda University School of Medicine, Loma Linda, Calif. 92354.)

At the time of coronary arteriography, labeled albumin microspheres and macroaggregated albumin were administered to 800 patients to determine the perfusion to the peripheral vascular bed.

The particle preparations were carefully prepared, and scrupulously quality controlled, so that only 10–30,000 particles were administered to each coronary bed.

Sixty per cent of patients undergoing examination had no adverse reaction resulting from tracer administration. Twenty per cent had some ECG changes without rhythm disturbances, while the remaining 20 per cent had rhythm disturbances ranging from ectopic beats in 13 patients to ventricular tachycardia in one.

Since the myocardium has an estimated 3,300 capillaries per millimeter, a 20,000 particle dose would deliver less than 100 particles/gram. This large safety factor permits the administration of particles to patients with severe coronary artery disease.

From this study, the intracoronary administration of microspheres appears to be a safe procedure, which can be employed in patients with severe coronary artery disease.—*William H. Strauss, M.D.*

McNeil, Barbara J., Holman, B. Leonard, and Adelstein, S. James. The scintigraphic definition of pulmonary embolism. *J.A.M.A.*, Feb., 1974, *227*, 753–756. (Address: Barbara J. McNeil, M.D., 25 Shattuck Street, Boston, Mass. 02115.)

A retrospective analysis of pulmonary perfusion and ventilation scanning in 57 patients suspected of having pulmonary emboli is presented. The presence of pulmonary emboli was confirmed or excluded by pulmonary angiography.

Parameters of significance as stated by the authors include: patterns of regional perfusion; ventilation perfusion; mismatches; and changes in perfusion defects in patients with serial scans.

Eighty per cent of lobar defects were associated with pulmonary emboli, while segmental or subsegmental defects had only a 30 per cent association with pulmonary emboli. Twenty patients had the combined ventilation/perfusion scan. Of these, 95 per cent of patients with a V/Q mismatch had pulmonary emboli. No patient with normal ventilation/perfusion had pulmonary emboli.

Of 15 patients with repeat scans, 10 patients had change in perfusion defects and all were associated with pulmonary emboli. In the 5 patients without emboli, a changing pattern was not present.

A combination of parameters included a large perfusion defect, and ventilation/perfusion mismatch correlated strongly with pulmonary emboli.—*Donald P. Harrington, M.D.*

MISCELLANEOUS

Marcove, Ralph C., Lyden, John P., Huvos, Andrew G., and Bullough, Peter B. Giant-cell tumors treated by cryosurgery: a report of twenty-five cases. *J. Bone & Joint Surg.*, Dec., 1973, *55-A*, 1633–1644. (Address: Ralph C. Marcove, M.D., 444 East 68th Street, New York, N. Y. 10021.)

Twenty-five consecutive patients seen at the Memorial Hospital for Cancer and Allied Diseases and The Hospital for Special Surgery, New York, with proved benign giant cell tumors were treated with cryosurgery. All but 1 tumor was in a long bone with the majority localized about the knee; the other lesion was in the innominate bone. Encouraging results in the treatment of metastatic tumors and locally aggressive and recurrent primary bone tumor, such as aneurysmal bone cysts, resulted in the present study.

Radiation therapy has proven to be of value principally only in the vertebrae and sacrum, because the areas are difficult to approach surgically. The lesions are, however, radioresistant, pathologic fractures are common from radionecrosis and secondary malignant transformation occurs 4 to 5 times more often

as in non-irradiated tumors. Cryosurgery is believed advantageous because it enhances the curettage and allows less sacrifice of tissues needed for reconstruction or joint function.

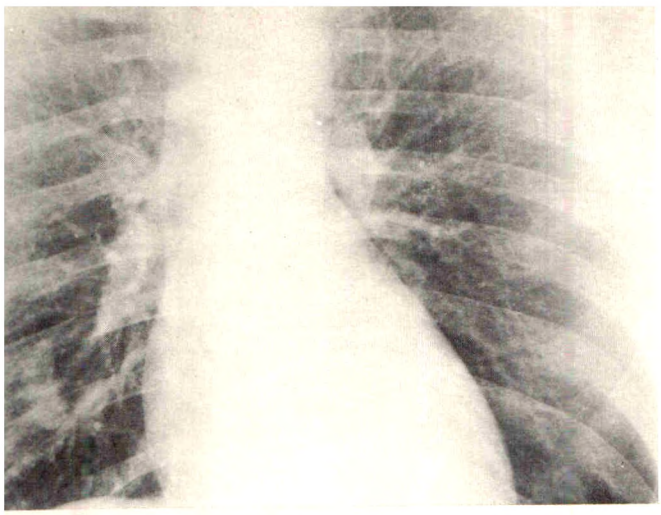
Each patient was treated by thorough curettage followed by instillation of liquid nitrogen *via* either a double lumen probe with circulating liquid nitrogen poured directly into the curetted cavities through a funnel. This destroys residual tumor beyond the limits of the curettage. In 20 patients a repeat open biopsy was performed 3 to 6 months after the original curettage and cryosurgery. If a frozen section of the biopsy tissue proved to contain residual tumor, the cryosurgery was repeated. Initially homogeneous or autogenous bone chips were used to fill the cavity following cryotherapy, but later strut grafts of fibula were utilized in order to prevent collapse of the remaining bone near the site of the tumor and still allow a thorough follow-up biopsy to be performed. Twelve patients utilized an ischial weight bearing brace for up to 30 months following treatment.

The bone immediately adjacent to previously treated cavities revealed histolcgic evidence of bone necrosis with total absence of osteocytes. Months after cryosurgery, where peripheral sclerosis was seen on roentgenograms, histologic examination revealed new bone formation in granulation tissue and on the surface of the necrotic bone trabeculae.

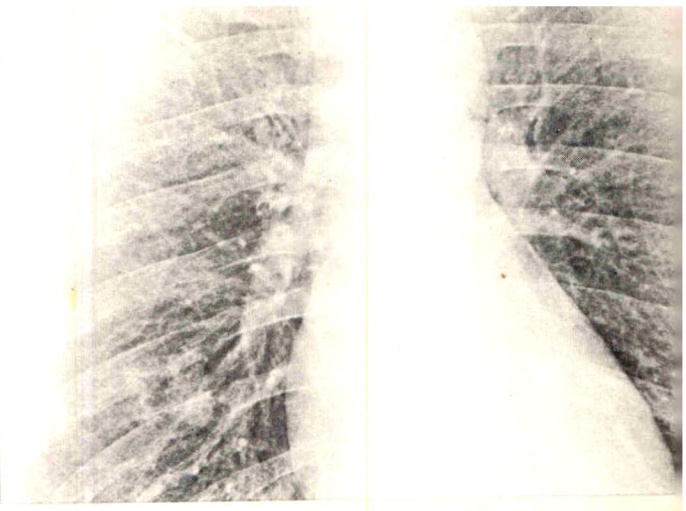
Fourteen of the 25 patients had previously been treated elsewhere by curettage and had recurrent tumor which was often highly destructive. There were 2 obvious clinical recurrences and 6 instances of residual fossae of tumor demonstrable only by the follow-up biopsy and in these cases it is not clear whether clinical recurrence would ever have become evident. Five of these 8 tumors were controlled with 2 repeat applications of cryotherapy; 1 had 2 applications and 2 had *en bloc* resections to eradicate the tumor.

There were 7 cases of postoperative infection. One necessitated amputation because of persistent osteomyelitis. Sepsis apparently has been a problem in any type of therapy for giant cell tumors. Two infections resulted from skin freezing and necrosis in another from faulty skin closure. It is hoped that the skin problems can be avoided by wider skin retraction.

Seven patients suffered fracture after cryosurgery and 3 healed uneventfully, 1 required a bone graft, and 3 ultimately were treated by insertion of a femoral condylar prosthesis. More frequent use of supporting fibular bone struts, use of ischial weight bearing braces, and increased protection from weight bearing, it is hoped, will reduce the rate of fracture occurrence. Five patients required fusion of the knee because of fracture or infection. Neuropraxis occurred in 3 patients and in each ultimately resolved, but this required up to 2 years for full recovery of nerve function.—*John T. Underberg, M.D.*



Conventional Silver Halide Image



Electron Radiographic Image

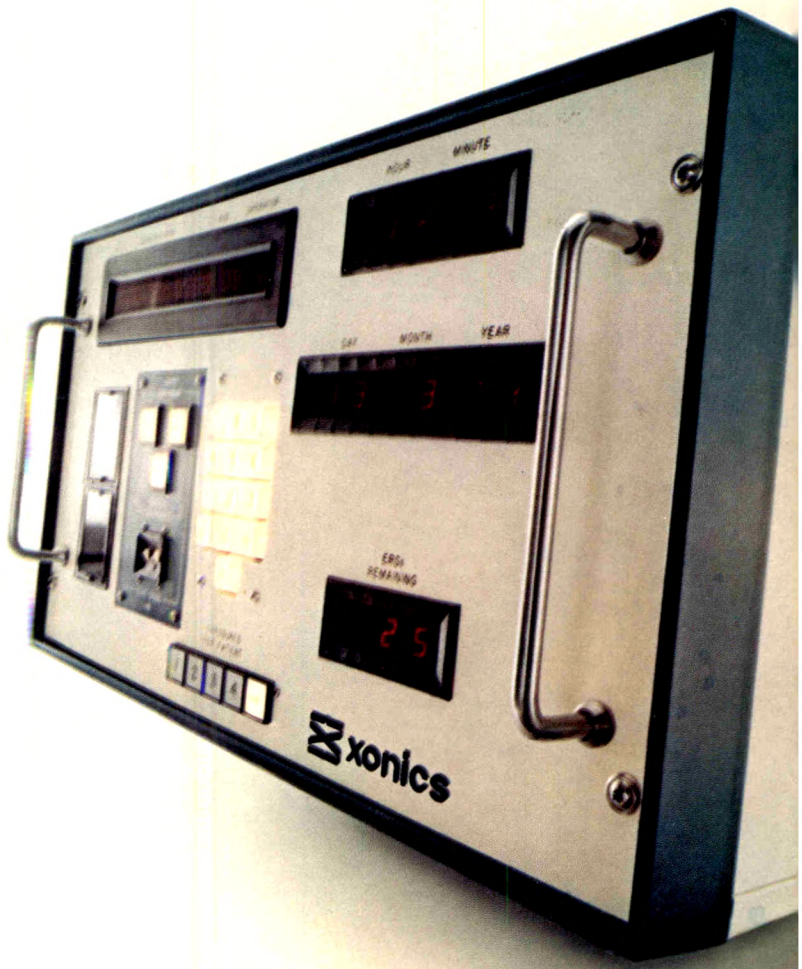
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You get films with considerably better detail and far greater latitude. A push-button system prints out patient identification, date, time and technician number.

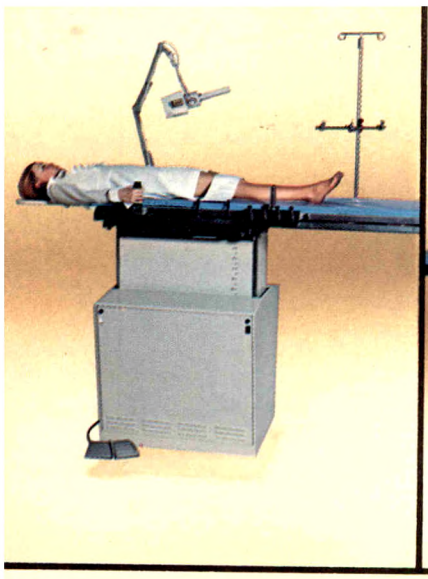
Exposures will be developed inside the unit in 60 seconds. And cost far less than silver halide film. Installation is simple—no plumbing required—compatible with x-ray generating equipment of any manufacture.

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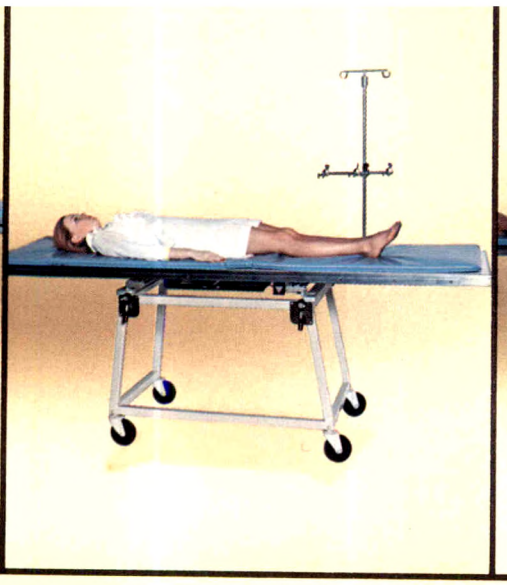
XONICS, INC.
2080 Stonington Ave.
Hoffman Estates, IL 60172
Telephone 312 884-9300



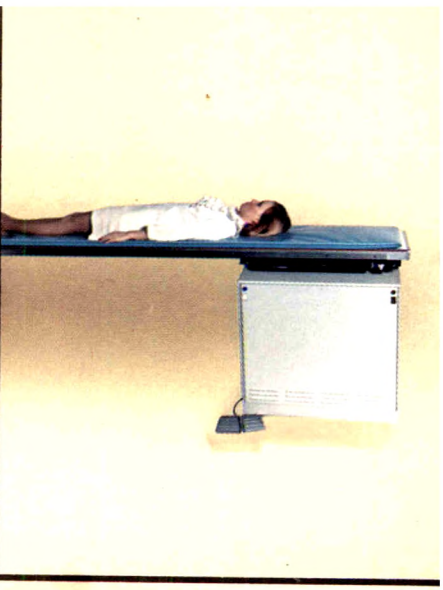
Xonics. Wait and see. Really see.



Neurovascular Configuration



Top Transfer Cart



Peripheral/Long Bone Study Configuration

Special procedures used to dictate the purchase of separate tables.

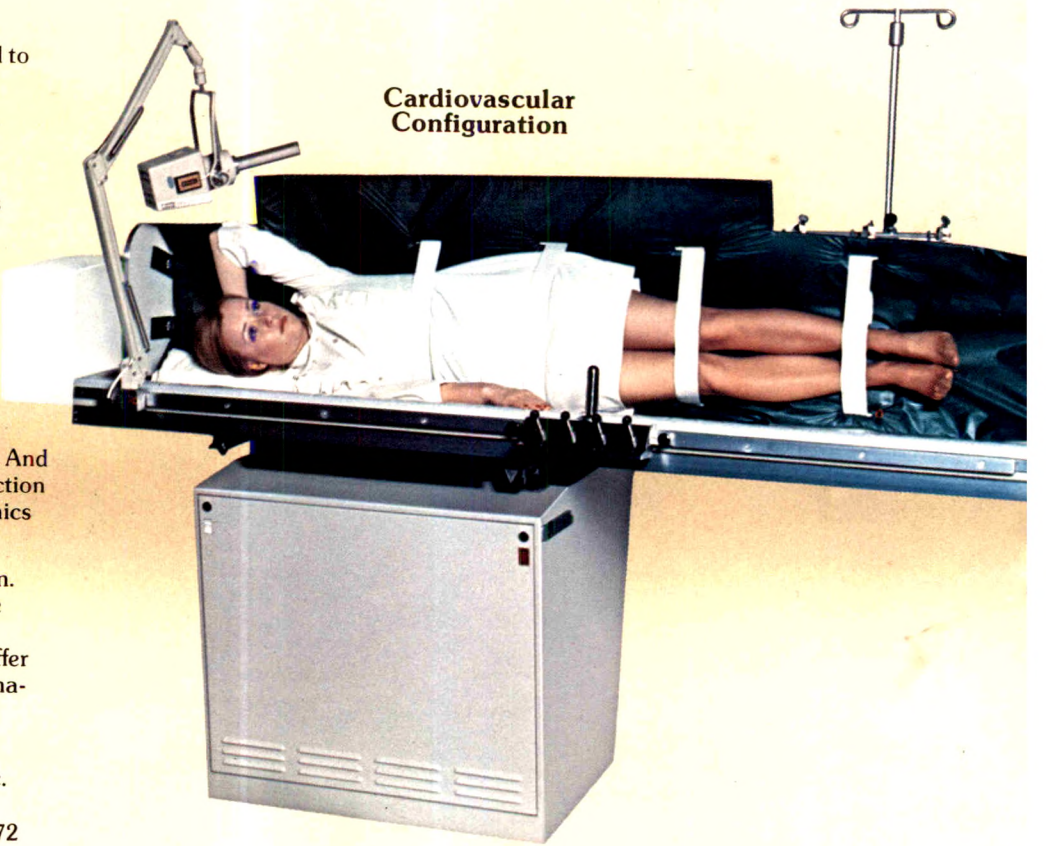
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Cardiovascular Configuration



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GE 400
film processor

Second-effort performance, plus big capacity and high speed separate this automatic film processor from the others. Learn all the reasons why the GE 400 processor is like getting twice the unit for your money.

Turn page for details



GENERAL ELECTRIC


Not just high speed
processing, but
second-effort
performance
when other units
might fail.

Big capacity, high speed

These are two important reasons for choosing the General Electric 400 automatic film processor. The unit will process 400 sheets of 90-second films dry-to-dry per hour—any mix of 5 x 7 inch to 14 x 36 inch. Also, 17-inch wide 90-second roll film at 70 inches per minute.

But there's still another reason why this processor is the smart choice for centralized and dispersed processing. And that's its second-effort ability to keep working when ordinary processors might quit.

Dual developer heaters, dual thermostats, dual dryer fans and heaters provide reliable processing performance. If one of these tandem units should malfunction, the other is more than adequate to take over and keep film processing on schedule. Not true of other processors. And this means less downtime. More volume you can count on.



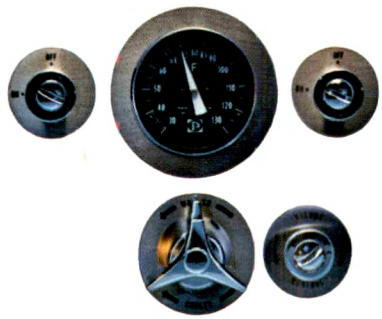
How the GE 400 unit processes, is equally interesting. Simply insert the film to start the process. Instantly, automatically, film width and length are sensed by foolproof, mechanical roll switches, and only the proper amount of developer replenisher is metered-in. No more than necessary . . . no less. Real savings in chemicals. Consistent film quality film after film.

Two heaters; two thermostats

Two 1000-watt heaters provide prompt warm-up of the developer solution; with one heater then maintaining a thermostatically controlled temperature within a critical ±0.5°F. This second heater alone is powerful enough to deliver effective heating for processing. Similarly, a standby thermostat is also built in.

External valve—no added cost

An external thermostatic mixing valve, normally a costly option with other processors—is included at no extra cost. This valve tempers the incoming wash



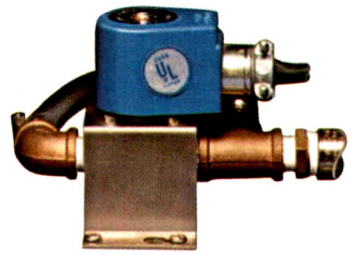
water, whose temperature is then maintained by conduction from the heated developer solution.

Thorough, 2-stage drying

Processed film is fed into the drying chamber where it is pre-dried by two recirculating warm air fans.

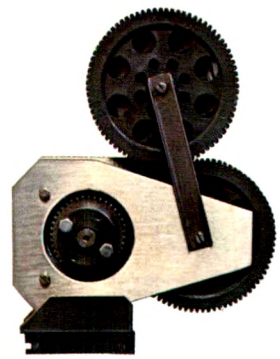
Then both sides of the film are subjected directly to heated air from two dryer heaters. In each drying stage, one of the fans and heaters will provide sufficient drying capacity.





Water savings, too

Water flow is also metered and is never more than 1½ gallons per minute. This flow is sharply reduced and automatically maintained at one quart per minute when no film is being processed—a water savings of 75 gallons per hour in the standby mode! Again, standard equipment for which you pay no more.



Smooth gear drive

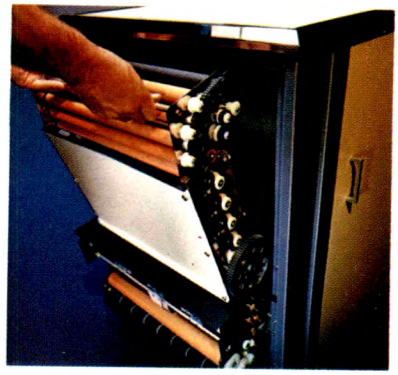
Heavy-duty gear drive, unlike chain drives in some processors, delivers a smooth steady roller movement to maintain continuously-uniform film-image quality. Gear drive system allows for quick adjustment from standard 90-second processing time to 3½ or 7 minutes if desired.

GENERAL ELECTRIC

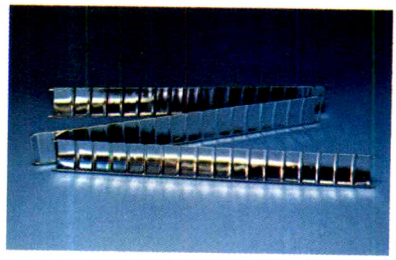
Maintenance in minutes, with full access servicing.

Routine maintenance and cleaning are easy to perform because top and side panels lift completely away for easy access.

Complete dryer rack can be removed for access to dryer fans and heaters, which are removable themselves as a unit (two bolts) in minutes.



Outside rollers of transport system lift out for cleaning when necessary, without dismantling the racks. New, semi-soft developer rollers give optimum transport with any film. Require only a gentle spraying to clean—no hard scrubbing or special cleansers.



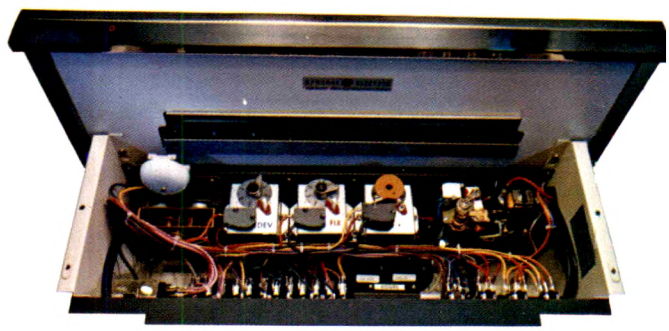
Cleaning cut to a minimum

Turnaround film deflectors—rather than roller crossovers—allow spotless clean-ups with just a damp cloth.

Corrosion-proof construction

Corrosion-proof features include stainless steel racks and tanks, torsion-spring roller compressors, and developer filter dolly.





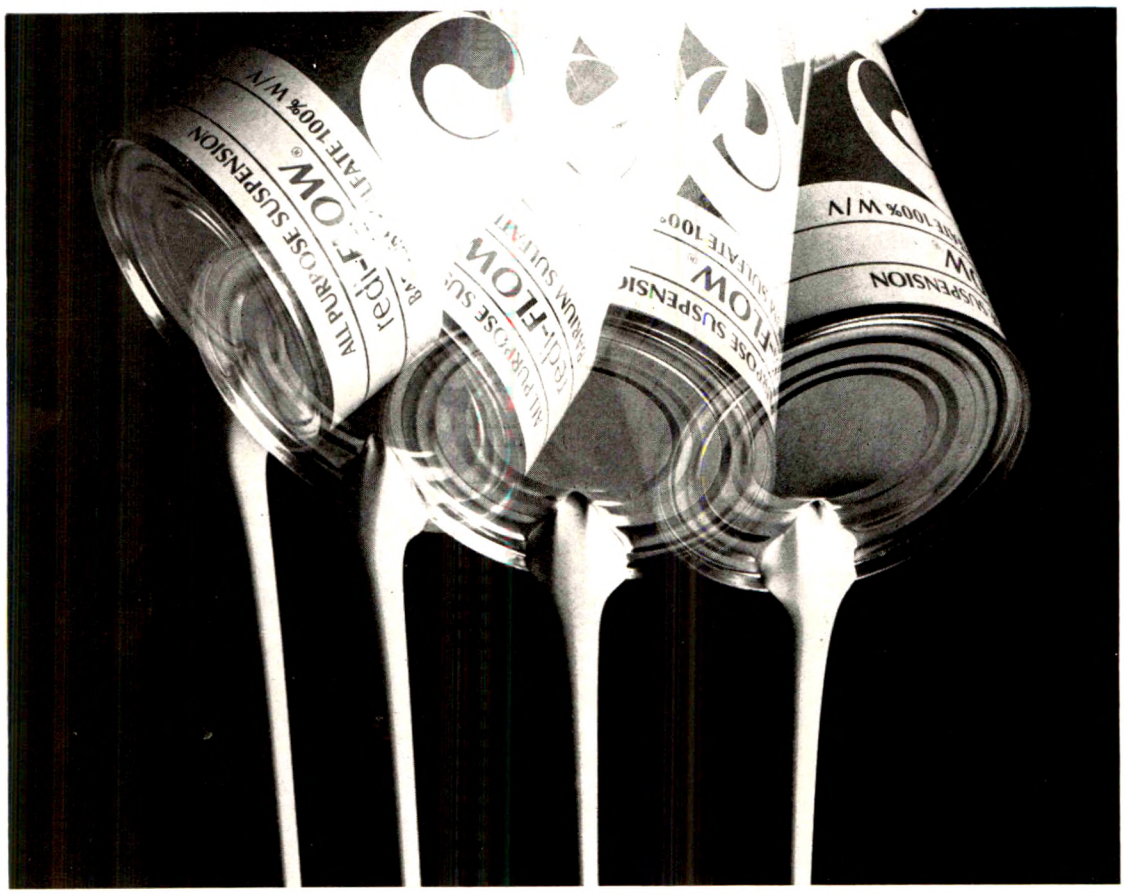
Protected electrical controls

Electrical controls are contained in a closed compartment below the feed tray—away from the hostile environment of the processing and drying sections. Each component is clearly labeled, easily removed (pull-out type), and serviceable from a stand-up position.

Let your GE representative provide details on the 400 film processor, and learn why it's like getting twice the processor for your money.

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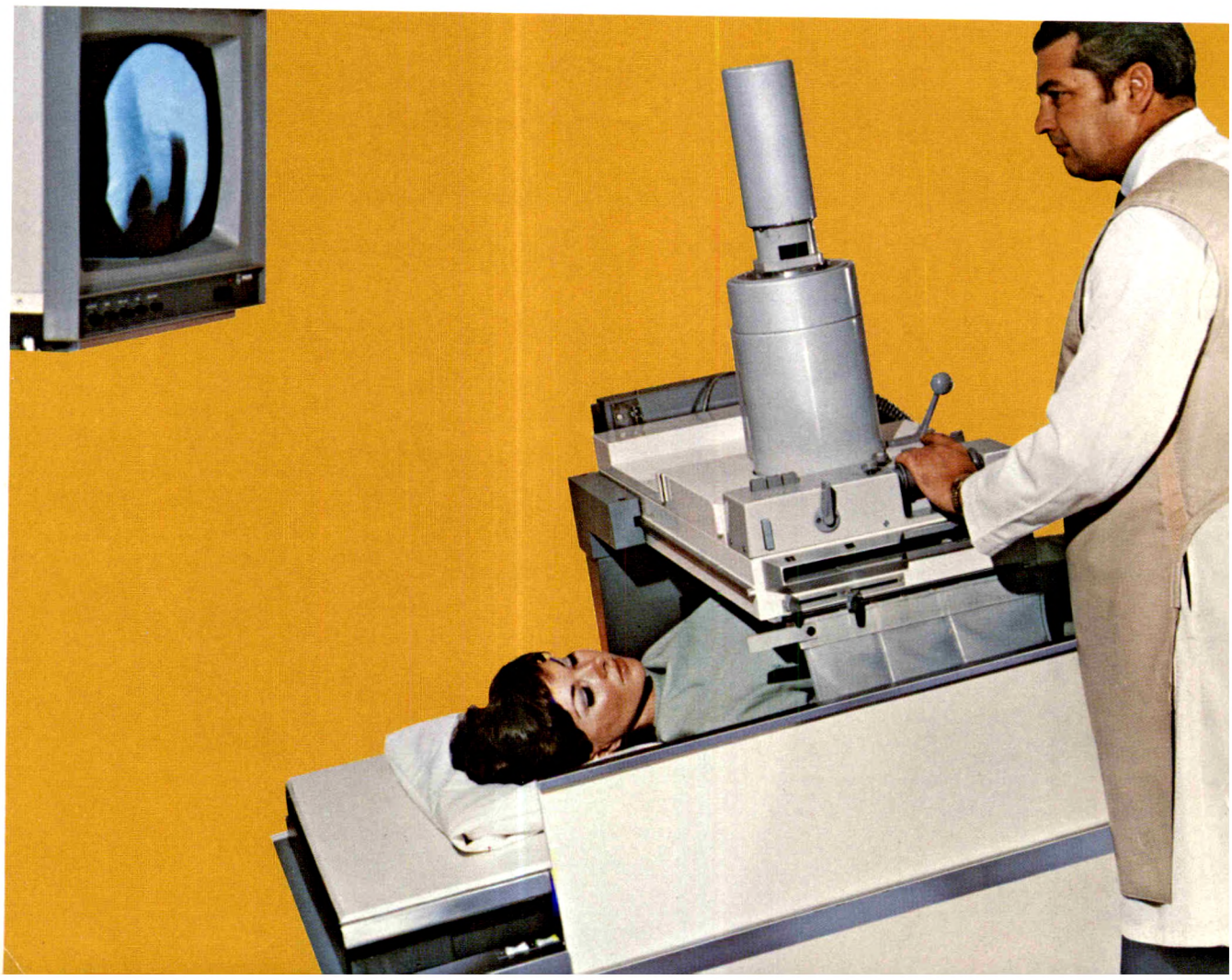
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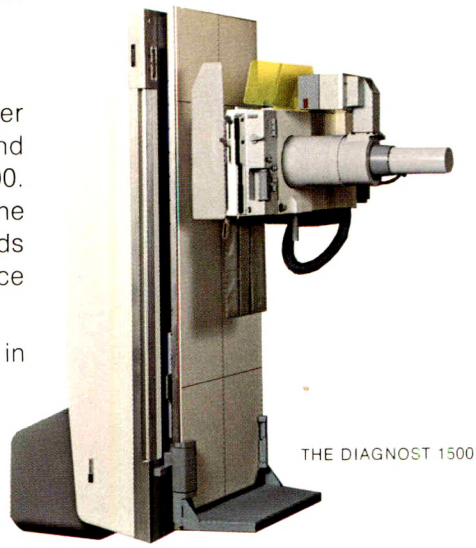
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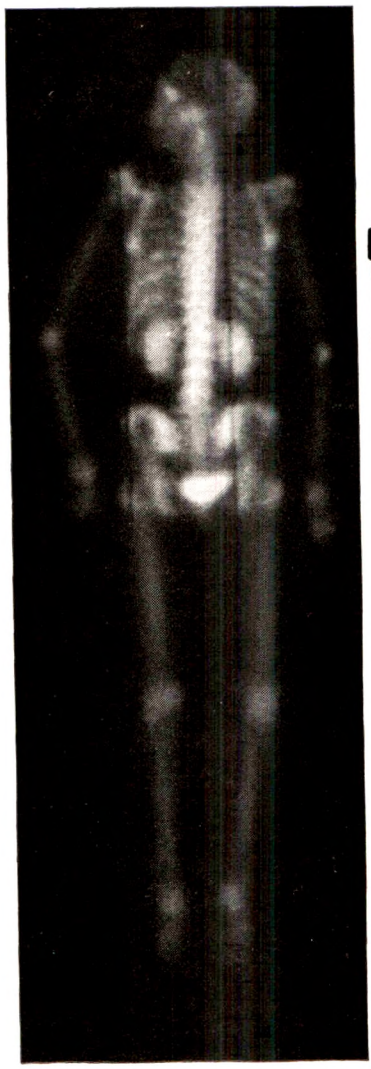
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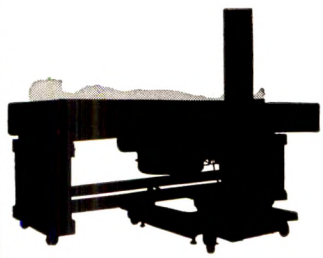
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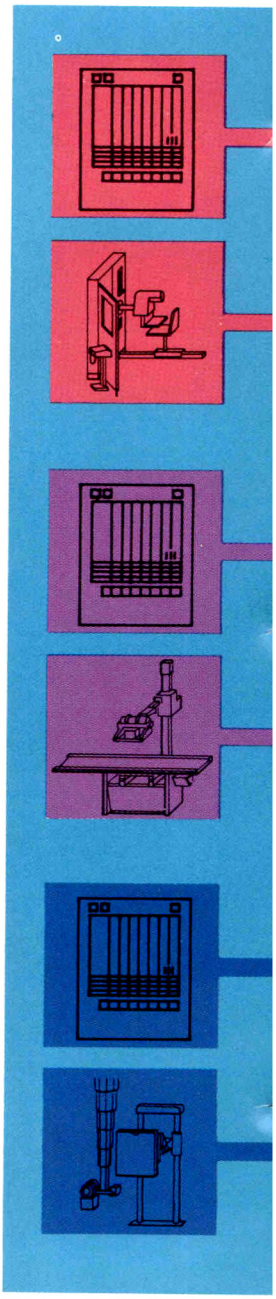
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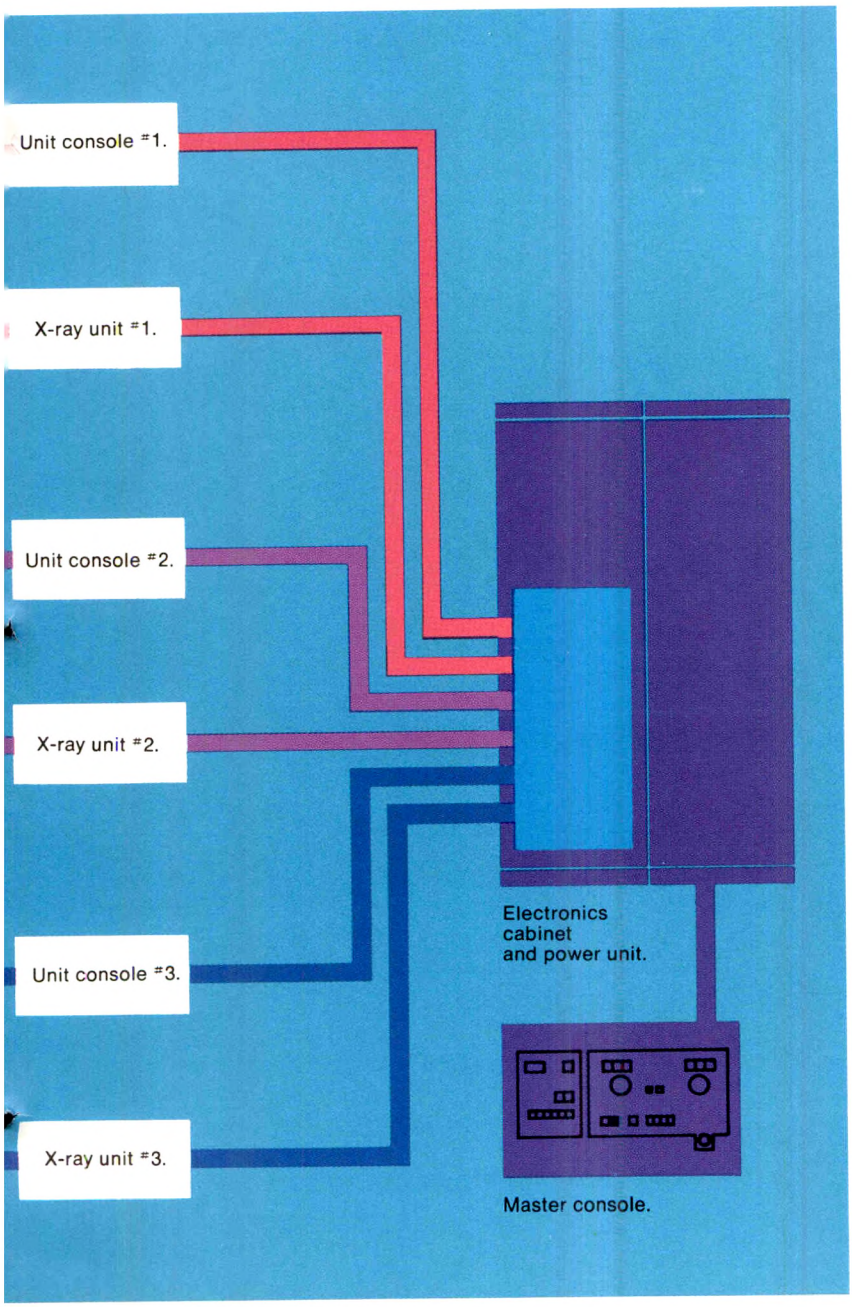
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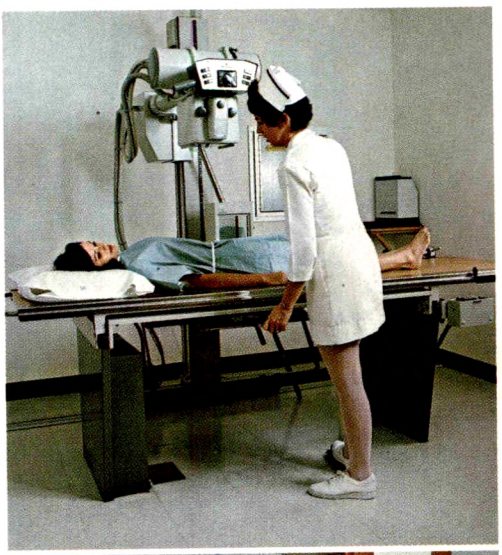
You can handle a vastly increased patient load, because automated procedures let three or more X-ray rooms operate at virtually the same time. While a patient is positioned in one room, a technician in the next room could be selecting preset radiographic data, while an X-ray is being taken in the third room. No running back and forth to make adjustments. Organ-related buttons automatically select kV, focal spot, screen combination and film blackening by means of an ionization chamber (Iontomat); cut to a minimum the number of decisions and settings required to get a diagnostic radiograph every time.

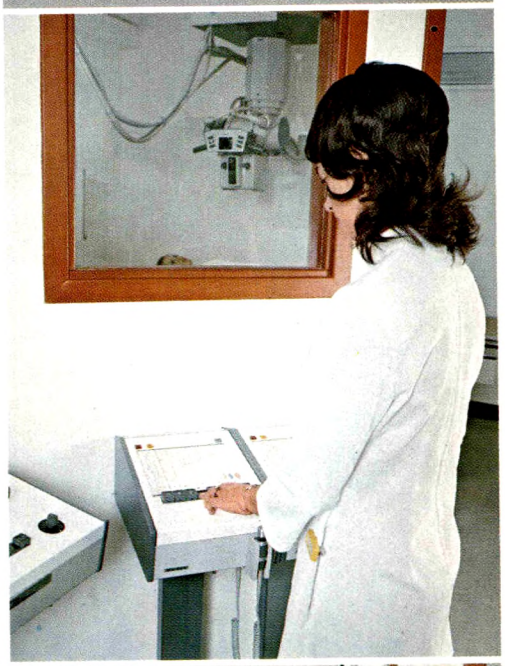
Three Siemens Optimatic generators offer outputs ranging from 50KW to 150KW. And all Optimatic systems feature electronic falling load, insuring maximum tube loading for shortest possible exposure times. Tubes are matched to the output of the generator.

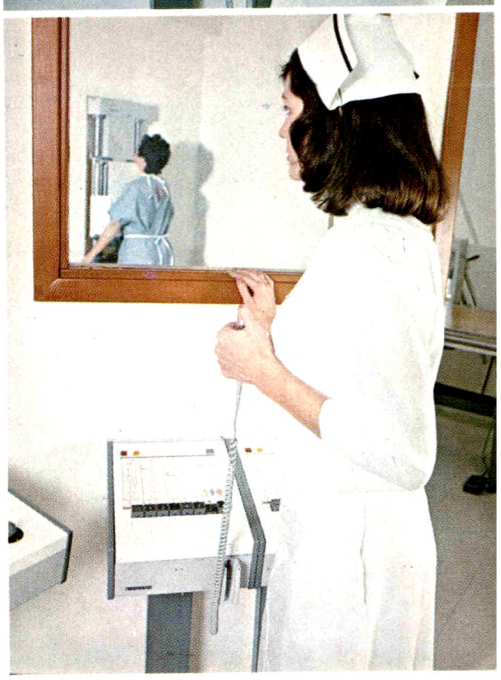
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X-ray unit #2.
Unit console #3.
X-ray unit #3.
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Master console.



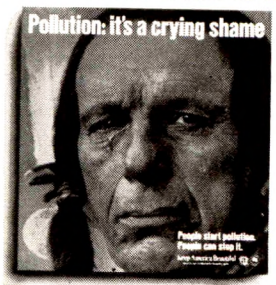




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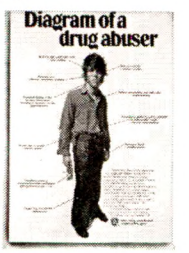


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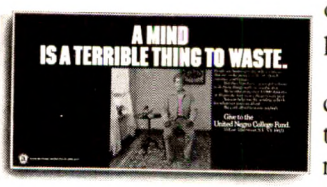




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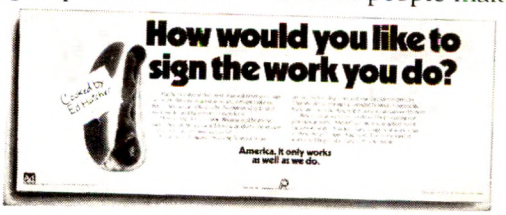
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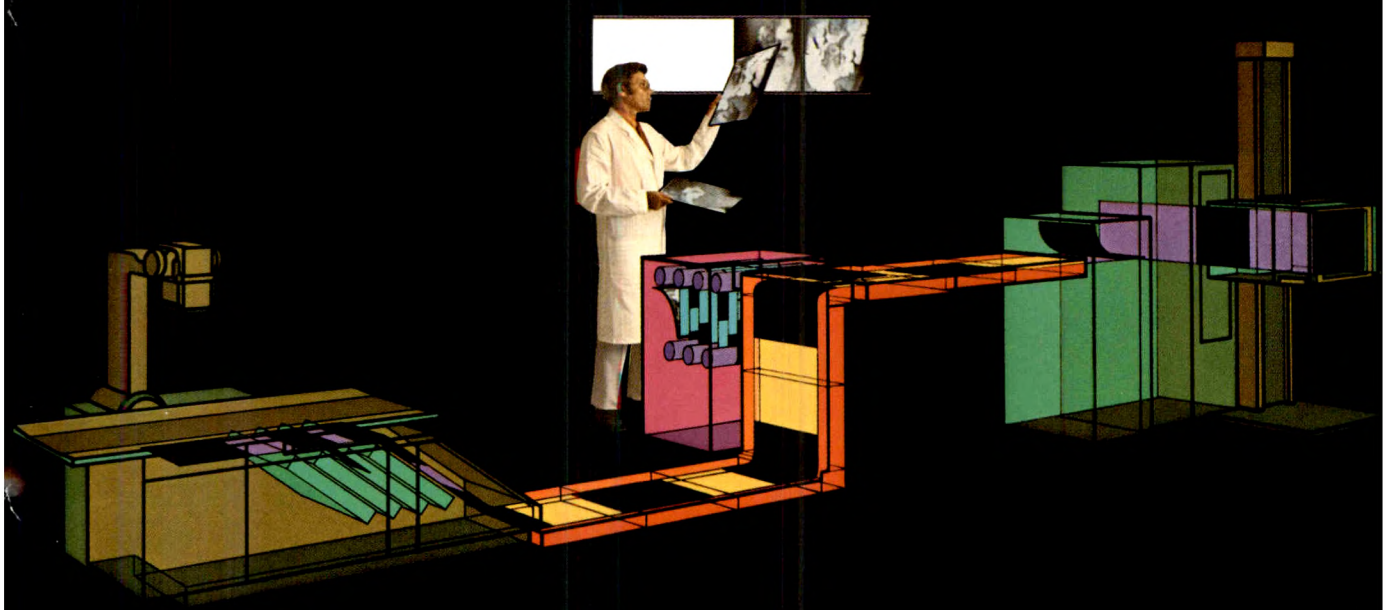
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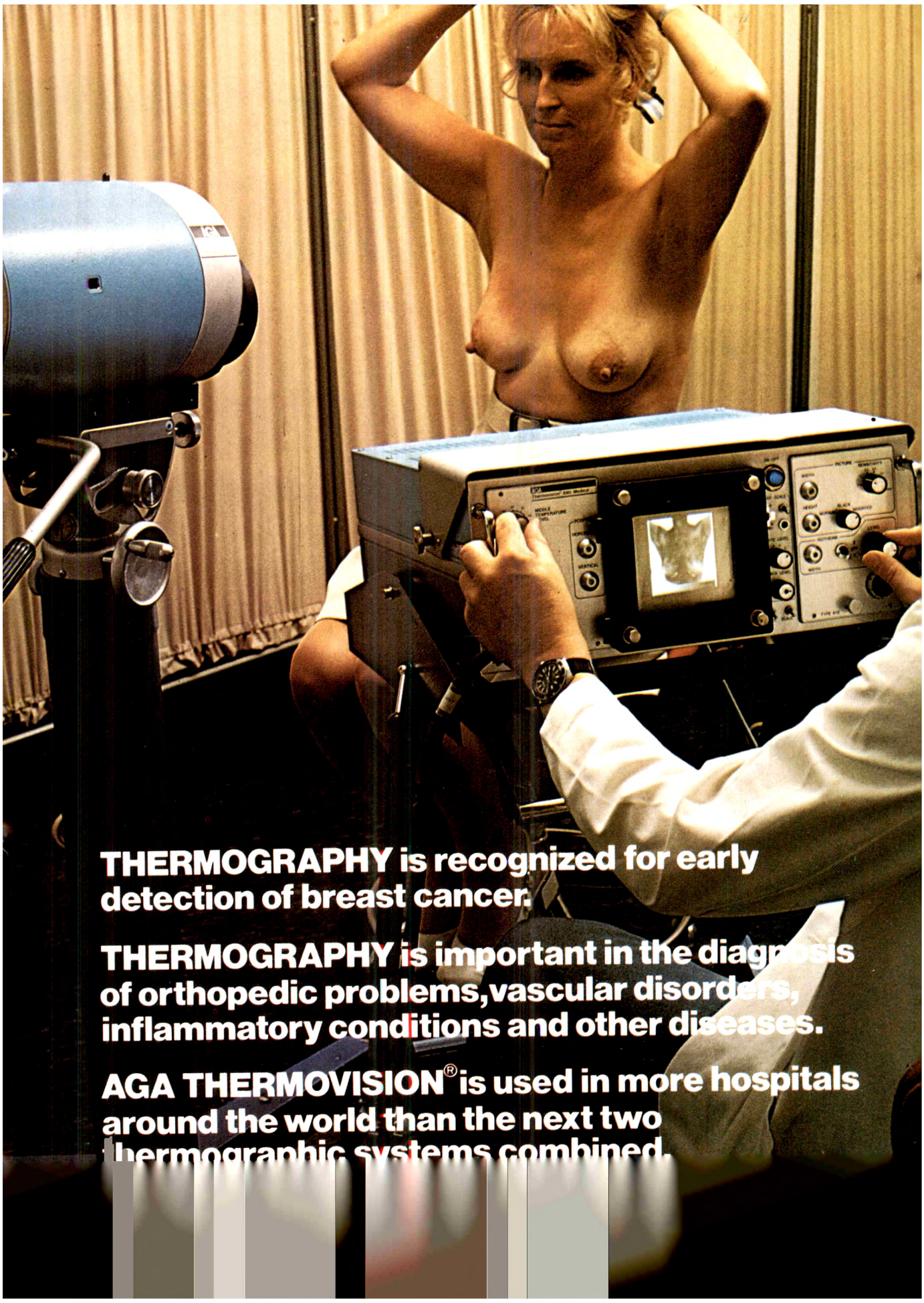
ROENTGEN EXAMINATIONS IN ACUTE ABDOMINAL DISEASES (3rd Ed.) *by* **J. Frimann-Dahl,** *Ulleval Hospital, Oslo, Norway.* This new third edition is completely revised with 133 new black and white illustrations and two new color illustrations. Written for radiologists and students in diagnostic radiology, this book also serves as a guide and support for surgeons involved in this important and difficult emergency field. The book is primarily concerned with roentgen anatomy of the abdomen and unusual or normal variations. The diagnosis of the most important acute abdominal diseases is also covered including the following cases: the situation in which the actual lesion can be diagnosed on plain films only, the lesions which may be observed by repeat examinations, and emergency cases in which diagnosis must be made immediately. Acute abdominal diseases are among the most frequently referred cases. It is shown that roentgen examination is often decisive in the diagnosis and correct treatment which may save the life of the patient. Because there is often no opportunity for prolonged discussion of the cases, it is very important to have a book in which information can be sought concerning the actual case. The book begins with a discussion of procedure and technique including apparatus, quality of the films and use of contrast media. Normal findings are discussed for the peritoneal cavity, the development of peritoneum and alimentary canal and the roentgen anatomy of the abdomen. Normal variations of the roentgen anatomy of the intestinal tract and of the flank are reviewed along with the accumulation of gas and occurrence of fluid levels. Some abdominal diseases discussed are strangulating and obturating obstruction, mechanical obturation of the large bowel, congenital malformations causing intestinal obstruction, hernias, intussusception, volvulus and intestinal knot syndrome. Roentgen examinations are discussed for diagnosis in mesenteric thrombosis and strangulation, abdominal angina, acute regional enteritis, acute appendicitis, acute colitis, acute cholecystitis and pancreatitis, ulcer perforation of the stomach and duodenum, gynecological lesions and lesions in the urinary tract. Also discussed are rupture of the diaphragm, liver, spleen, kidney, stomach, duodenum, small intestine, colon and aneurysms. *'74, 632 pp., 614 il. (4 in full color), $29.50*

THE BIOLOGICAL AND CLINICAL BASIS OF RADIOSENSITIVITY. *Edited by* **Milton Friedman,** *Instituto Regina Elena, Rome, Italy. (35 Contributors)* Which are the most important factors influencing radiosensitivity? At which biological level do major effects occur? What are the differences between radiosensitivity and chemosensitivity? What clinically useful information can be derived from cell survival curves? Can the variation in sensitivity during each phase of the cell cycle be used to improve the ultimate radiation effect? Has hyperbaric oxygenation resulted in increased cure rates? Should its possible usefulness be explored further, or should it be abondoned? At the preclinical level, the lethal dose for each tumor in man is depicted. Combined radiation and chemotherapy is evaluated not only for potential benefits, but also for possible disruption of efficient irradiation; the neglected field of dynamic radiation pathology is examined; and, finally, the validity of a number of radiobiological laws is analyzed during the treatment of squamous cell carcinoma. *'74, about 589 pp. (7 x 10), 373 il., 67 tables*

THE FUNDAMENTALS OF RADIOLOGICAL SCIENCE *by* **John Hale,** *Hospital of the Univ. of Pennsylvania, Philadelphia.* This book is invaluable for the study of the fundamentals of radiological science, the primary aim of which is to bring the student to an understanding of a relatively few basic principles. From this, the student should be able to continue to assimilate new ideas and methods through his professional life. A body of basic scientific knowledge is presented that is common to the four fields of applied radiological science, that is radiotherapy, clinical nuclear medicine, diagnostic radiology and radiation safety physics. The subject matter is divided into a review of basic mathematics, basic physical concepts, atomic and nuclear physics, machines for producing radiation and the radiation field, interaction of radiation of matter, dose kerma and exposure. While the book is directed toward first-year physicians, it should also be useful as a beginning text for physicists and engineers who are interested in radiological physics or health physics. *'74, 356 pp., 172 il., 3 tables, $14.75*

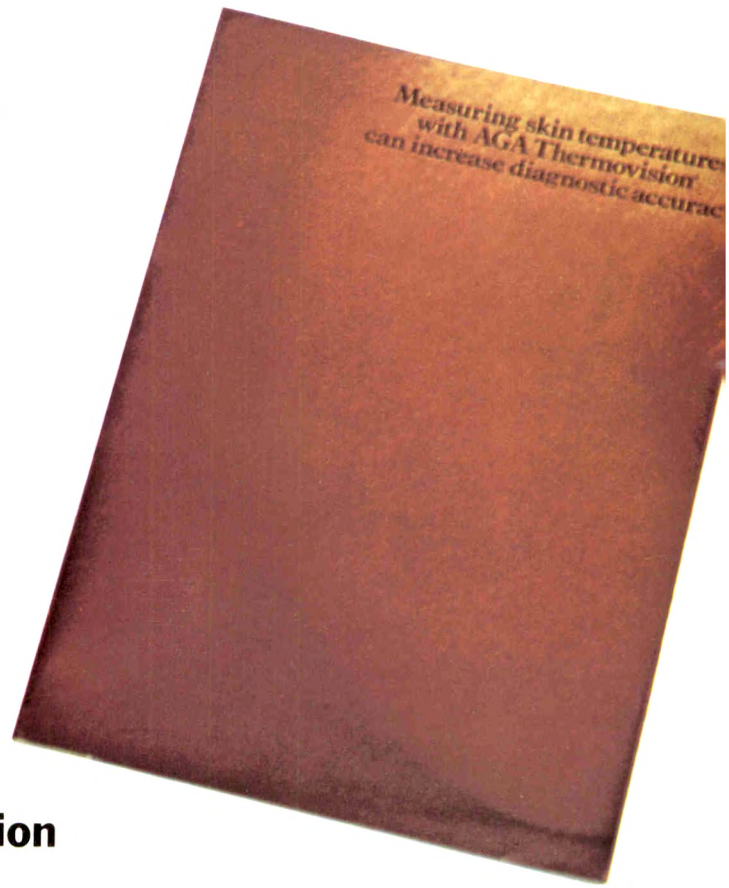
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THE SPLEEN *by* **A. I. S. Macpherson, John Richmond** *and* **A. E. Stuart,** *all of the Univ. of Edinburgh, Edinburgh, Scotland.* The histological anatomy of the spleen and splenic circulation is examined with particular reference to electron-microscopic findings. The cell population of the spleen and its place in immune responses have been reviewed. The technique and risks of splenectomy and other aspects of splenic disease and injury are considered along with results of splenectomy in the treatment of diseases which involve the spleen. The place of laboratory investigations, and especially of tests using radioisotopes, is fully examined. *'73, 290 pp., 97 il. (6 in full color), 7 tables, $15.75*

NONTOXIC GOITER: Concept and Controversy *by* **Joel L. Hamburger,** *Northland Thyroid Laboratory, Southfield, Michigan.* This volume provides an in-depth discussion of the methodology and interpretation of thyroid scanning. The section on simple goiter reduces the problem to a stepwise progression, outlined on the flowsheet, which permits an efficient determination of the general pathophysiologic category and proper management. Clinical evaluation of thyroid nodules for malignant potential is examined in detail, including instructions on the most effective way to examine the thyroid gland. Thyroid cancer is examined from the standpoint of pathology, surgical management and radiation therapy. *'73, 232 pp., 75 il., 13 tables, $9.75*

INDICATIONS AND ALTERNATIVES IN X-RAY DIAGNOSIS: A Guide to the Effective Employment of Roentgenologic Studies in the Solution of Diagnostic Problems (2nd Ed.) *by* **Melvyn H. Schreiber,** *Univ. of Texas, Galveston.* This illustrated volume describes the various types of roentgenologic examinations available for unravelling diagnostic problems. Provided is a thorough background in the uses to which ordinary and special x-ray studies may be put, with alternatives emphasized for situations where the indicated examination cannot be performed or when more than one kind of examination is available for the solution of a specific problem. *'74, about 185 pp., 98 il.*

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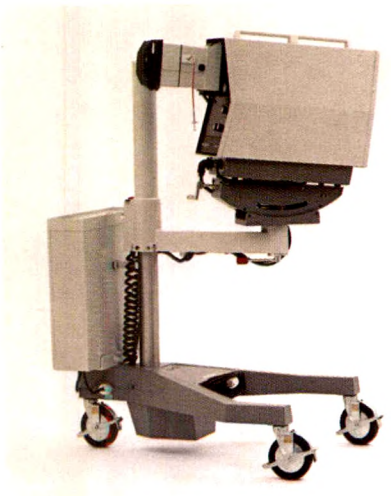
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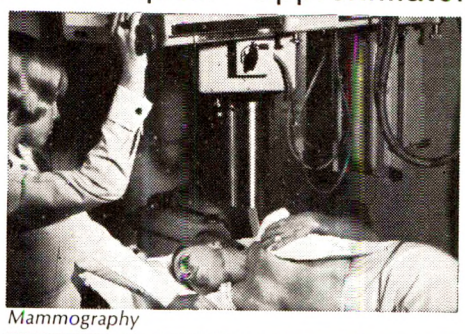
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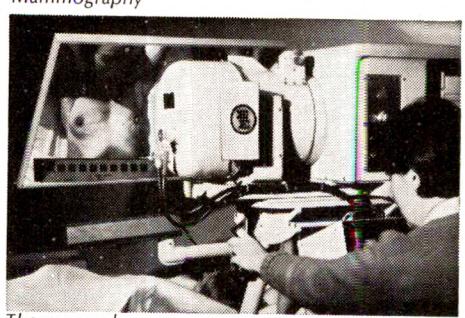
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Futility and frustration beset the physician confronted with breast cancer. For the last 35 years, the survival rate has not significantly changed despite intensive educational programs aimed at earlier detection, and improvement in treatment techniques.

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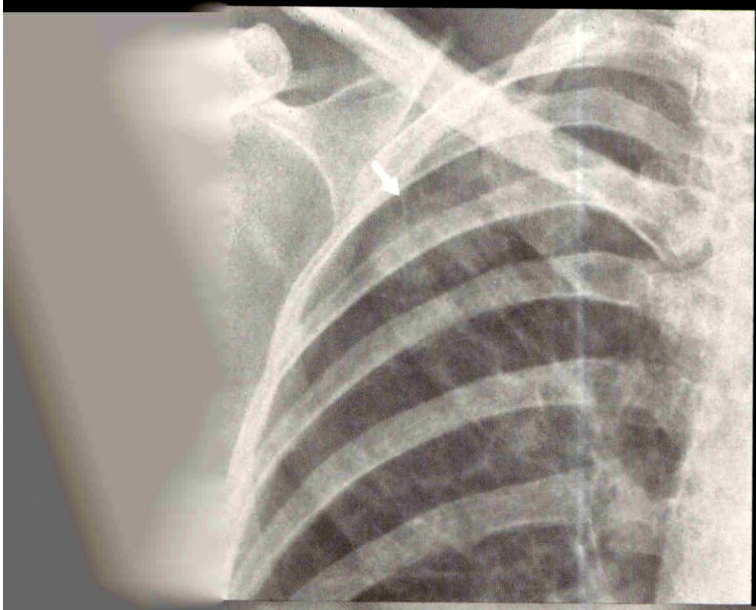
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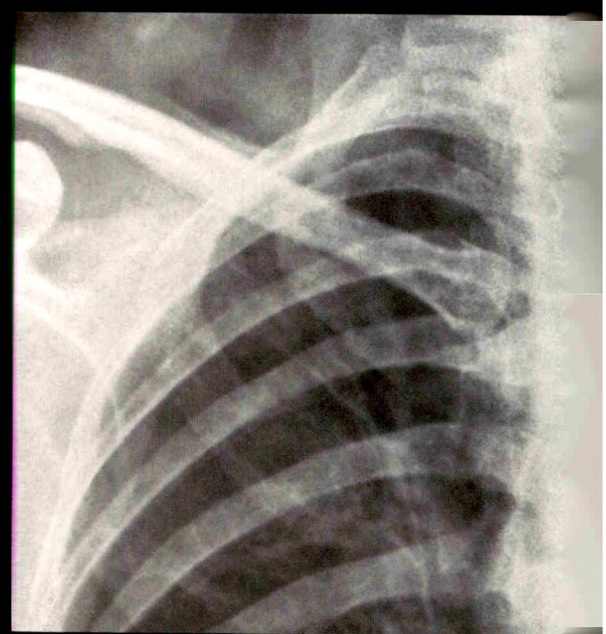
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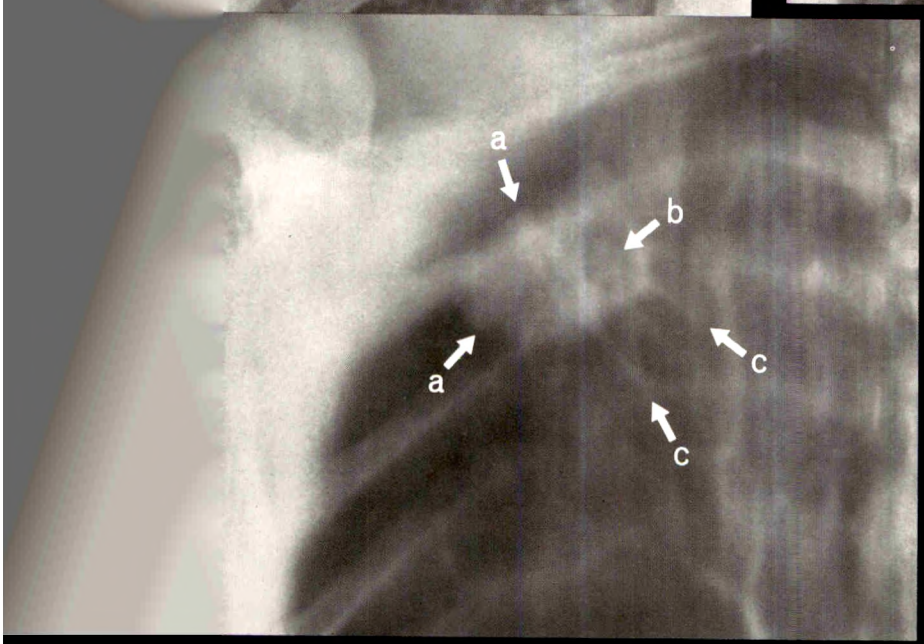
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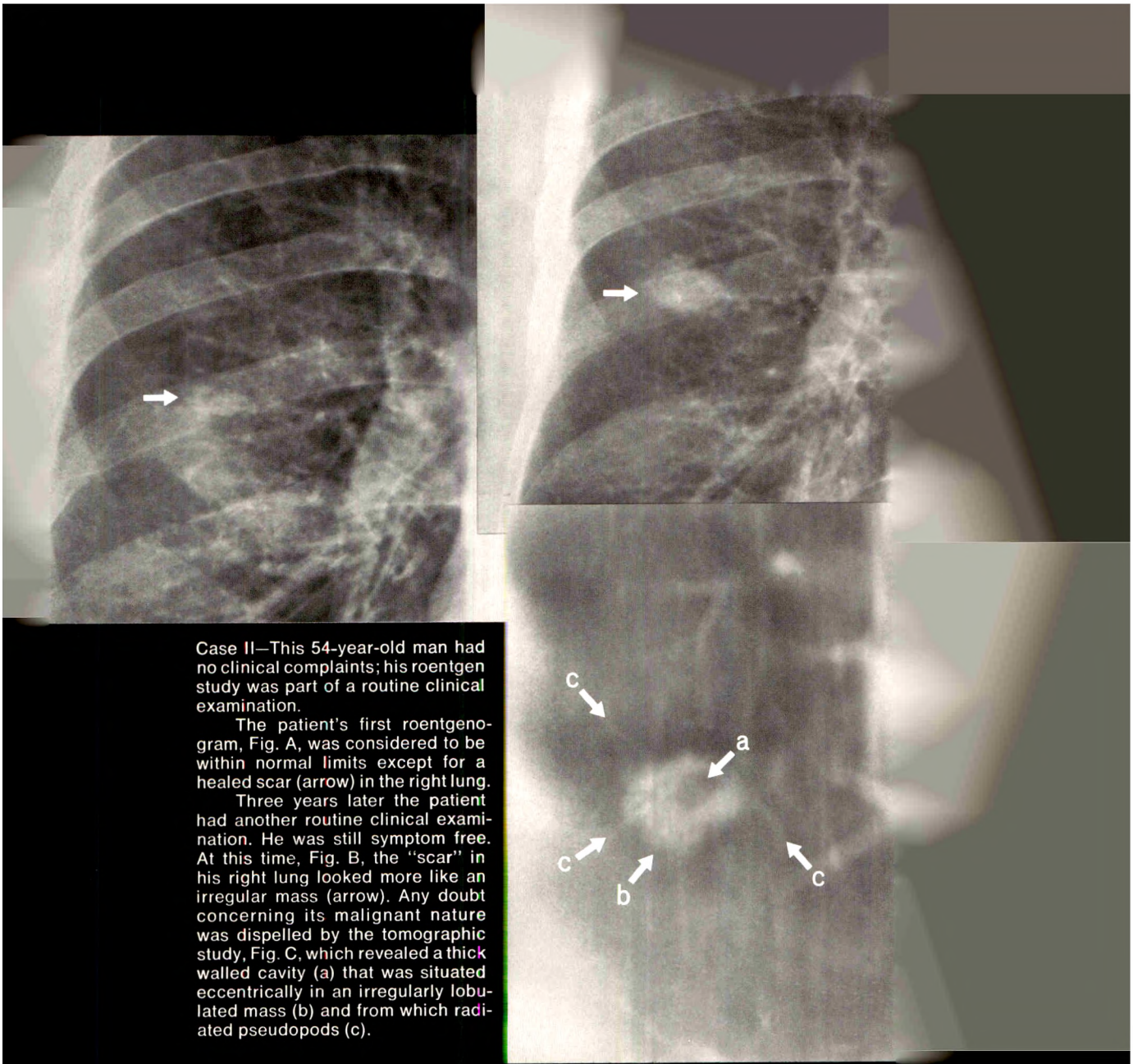
Case I—This 45-year-old lady had a mild cough for several weeks; there were no other subjective or objective clinical findings.

The first roentgen examination of her chest, Fig. A, revealed an ill-defined mass in the right upper lobe (arrow) which the radiologist thought compatible with early bronchogenic carcinoma. The patient's physician was not convinced.

Three months later, Fig. B, the lesion seemed less well defined.

At the insistence of the radiologist tomographic studies were made, Fig. C, which revealed an irregular mass (a), an eccentric cavity with a relatively thick wall (b), and fine pseudopods (c), all hallmarks of primary lung malignancy.

Radiographs and caption material from the American College of Radiology—*An Atlas of Tumor Radiology*—Yearbook Medical Publishers, Inc. Reprints of this and subsequent reports available upon request. Please write Eastman Kodak Company, Dept. 740, Radiography Markets Division, Rochester, New York 14650



Case II—This 54-year-old man had no clinical complaints; his roentgen study was part of a routine clinical examination.

The patient's first roentgenogram, Fig. A, was considered to be within normal limits except for a healed scar (arrow) in the right lung.

Three years later the patient had another routine clinical examination. He was still symptom free. At this time, Fig. B, the "scar" in his right lung looked more like an irregular mass (arrow). Any doubt concerning its malignant nature was dispelled by the tomographic study, Fig. C, which revealed a thick walled cavity (a) that was situated eccentrically in an irregularly lobulated mass (b) and from which radiated pseudopods (c).

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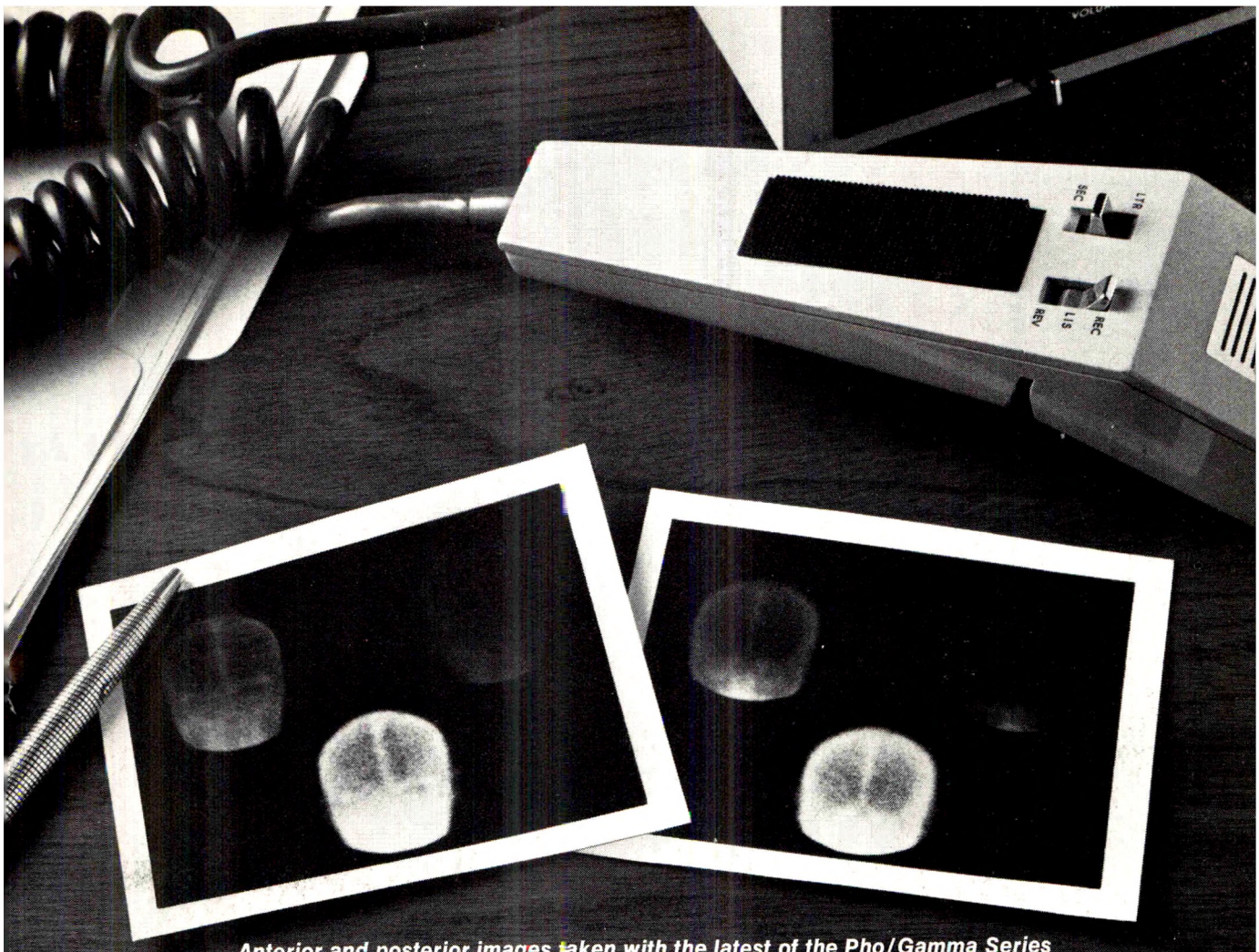
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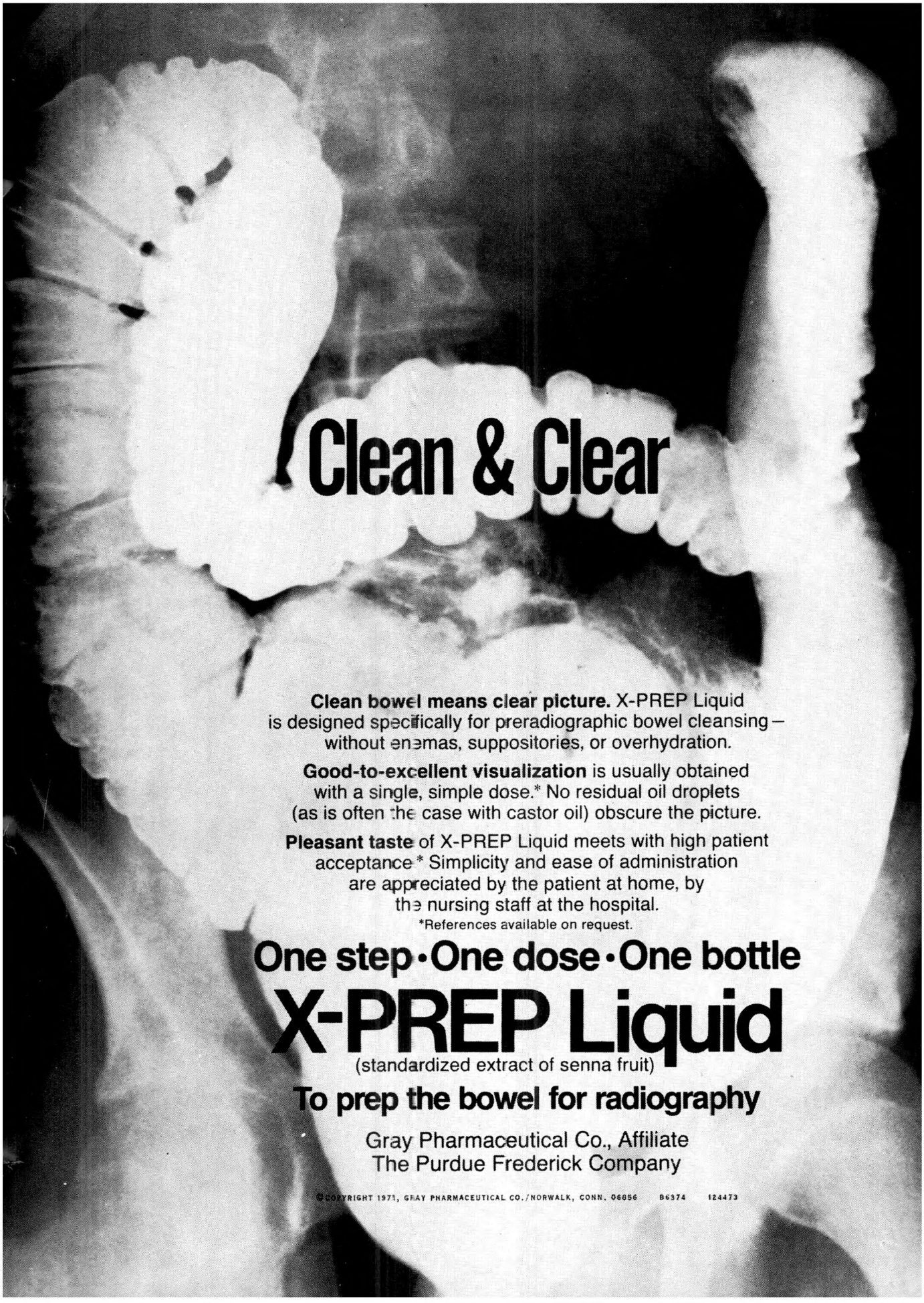
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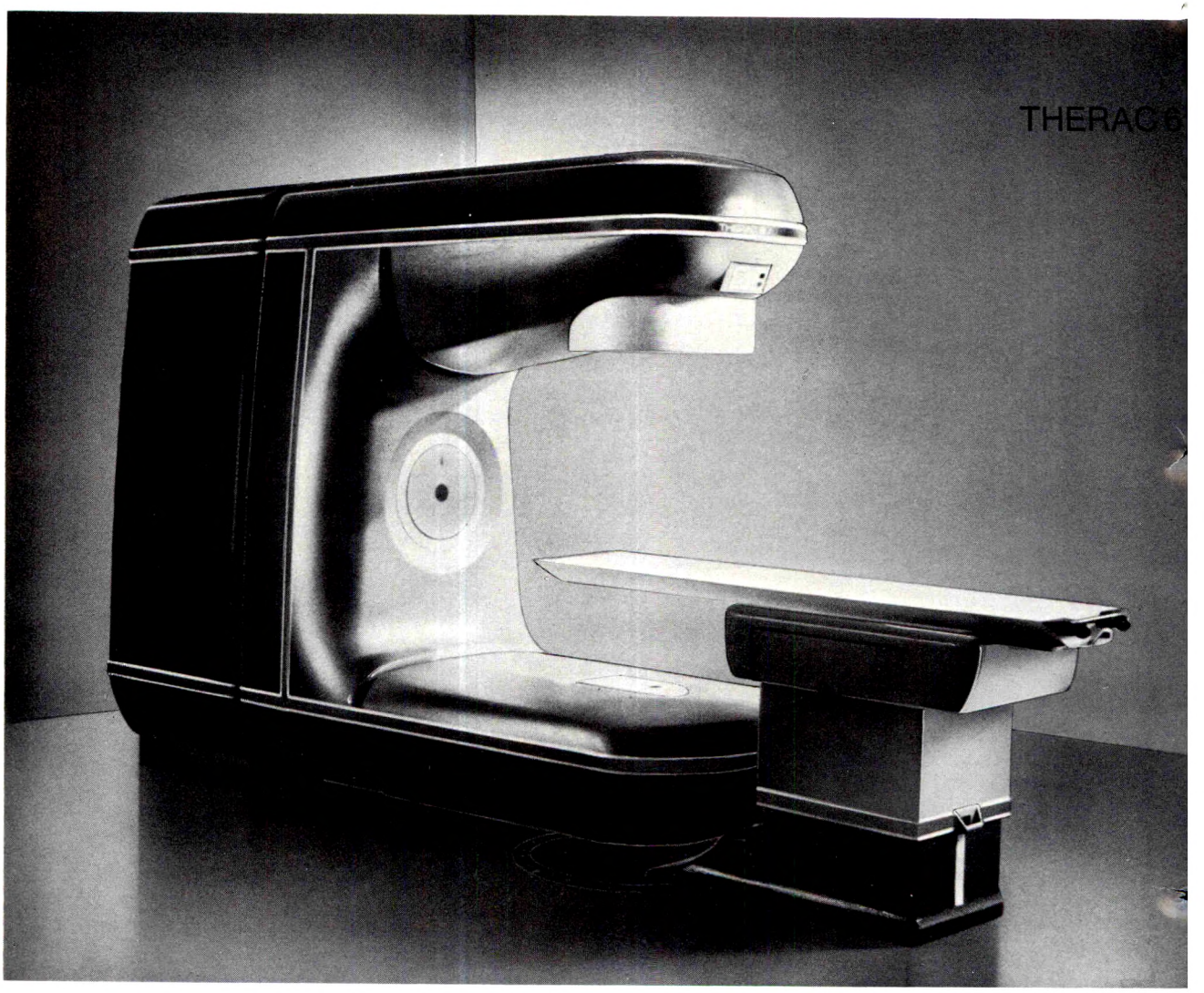
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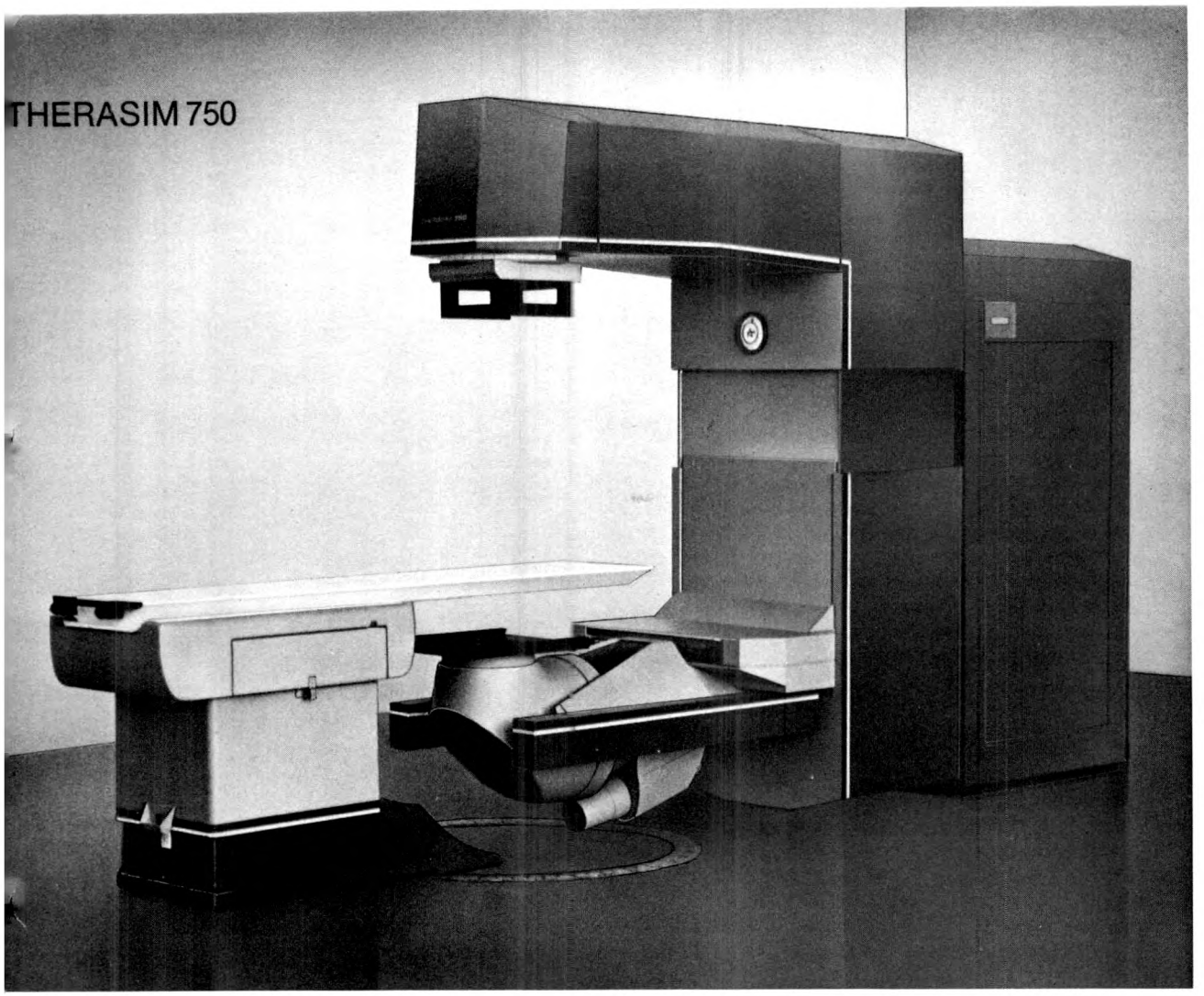
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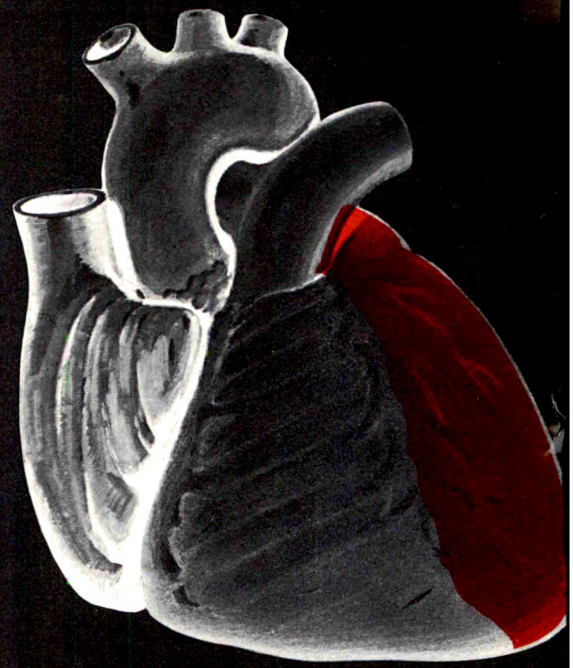
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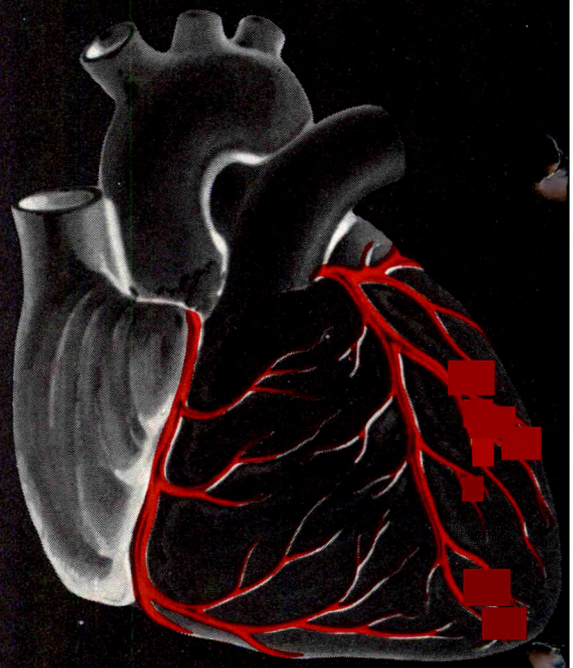
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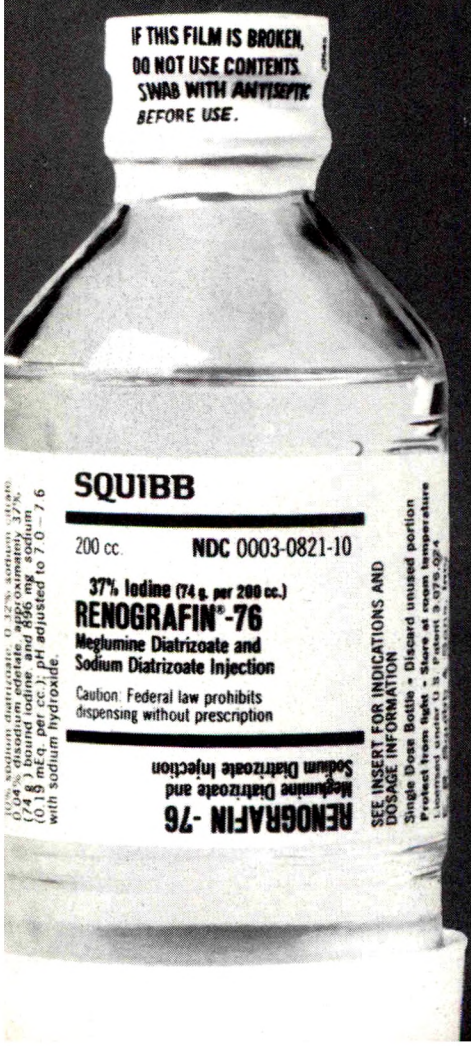
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ontraindications: In patients with a hypersenivity to salts of diatrizoic acid. Urography ntraindicated in patients with anuria.

arnings: A definite risk exists with the use of ntrast agents in excretion urography in paents with multiple myeloma. There has been uria with progressive uremia, renal failure d death. This risk of the procedure in these tients is not a contraindication; however, rtial dehydration in preparation for study is t recommended since it may predispose for ecipitation of myeloma protein in renal bules. No therapy, including dialysis, has en successful in reversing this effect. reloma should be considered in persons over before undertaking urographic procedures. In cases of known or suspected pheochrocytoma, if the physician feels that the posle benefits outweigh the considered risks, diopaque materials should be administered h extreme caution; however, an absolute nimum of material should be injected, the ood pressure should be assessed throughout procedure, and measures for treating a pertensive crisis should be available.

Contrast media may promote sickling in mozygous individuals when injected I.V. or ra-arterially. Although a history of sensitivity iodine *per se* or to other contrast media is an absolute contraindication, administran of diatrizoate requires extreme caution in ch cases. Renografin-76 should be used in gnant patients only when the physician ems its use essential to the welfare of the ient since safe use during pregnancy has been established. Perform thyroid function ts prior to administration of Renografin-76 ce iodine-containing contrast agents may r the test results.

Veigh the inherent risks against necessity performing *angiocardiography* in cyanotic ants and patients with chronic pulmonary physema. In *pediatric angiocardiography*, a e of 10 to 20 cc. may be particularly hazous in infants weighing less than 7 kg.; this is probably significantly increased if these nts have preexisting right heart "strain," t heart failure, and effectively decreased obliterated pulmonary vascular beds. Per- *urography* with extreme caution in pers with severe concomitant hepatic and al disease. Perform *selective coronary arography* only in selected patients and those whom expected benefits outweigh the prural risk.

cautions: Diagnostic procedures involving of contrast agents should be performed er the direction of personnel with prereqte training and a thorough knowledge of the ticular procedure. Appropriate facilities uld be available for coping with situations ch may arise as a result of the procedure for emergency treatment of severe reons to the contrast agent itself; competent sonnel and emergency facilities should be lable for at least 30 to 60 minutes after I.V. inistration since delayed reactions have n known to occur. These severe lifeatening reactions suggest hypersensitivity e contrast agent. A personal or family hisspecial attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 cc. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

Renal toxicity has been reported in a few patients with liver dysfunction who were given oral cholecystographic agents followed by urographic agents; therefore, if known or suspected hepatic or biliary disorder exists, administration of Renografin-76 should be postponed following the ingestion of cholecystographic agents. Consider the functional ability of the kidneys before injecting Renografin-76. Use cautiously in severely debilitated patients and in those with marked hypertension. Bear in mind the possibility of thrombosis when using percutaneous techniques.

In *excretion urography*, adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

Adverse Reactions: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis procedure in presence of preexistent renal disease, retroperitoneal hemorrhage from trans lumbar approach, spinal cord injury an pathology associated with syndrome of trans verse myelitis, generalized petechiae, an death following hypotension, arrhythmia, an anaphylactoid reactions. In *pediatric angio cardiography*, arrhythmia and death have oc curred. During *peripheral arteriography*, hemor rhage from puncture site, thrombosis of th vessel, and brachial plexus palsy (followin axillary artery injection) have occurred. Durin *selective coronary arteriography* and *selective coronary arteriography with left ventriculography*, transient ECG changes (most patients) transient arrhythmias (infrequent); ventricula fibrillation (from manipulation of catheter o administration of medium); hypotension; ches pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in abou 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislodgment o arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have oc curred due to the procedure.

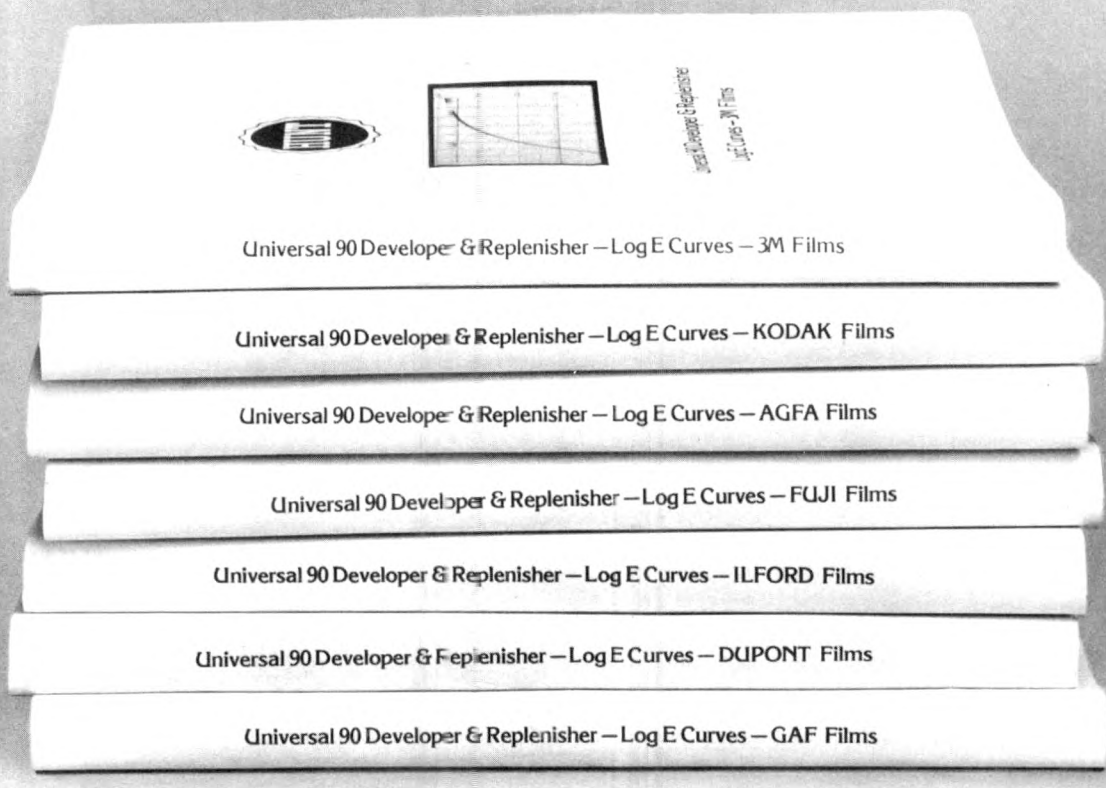
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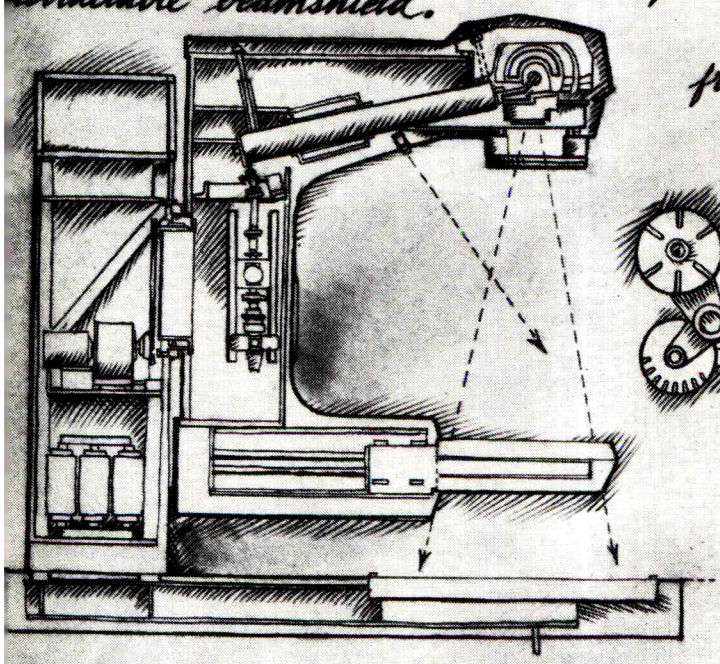
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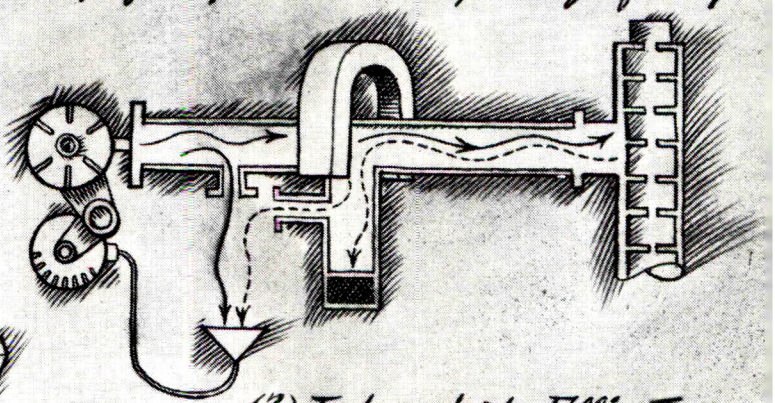
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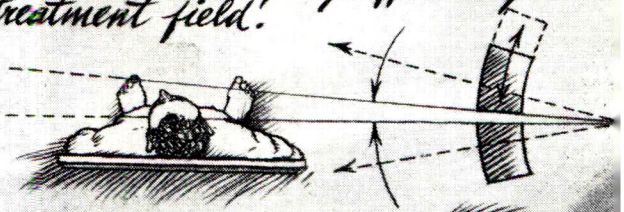
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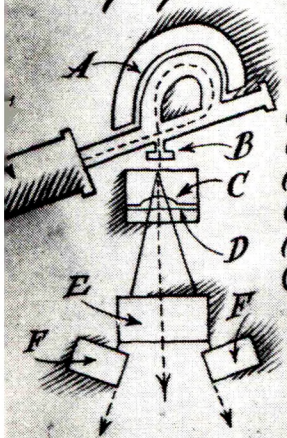
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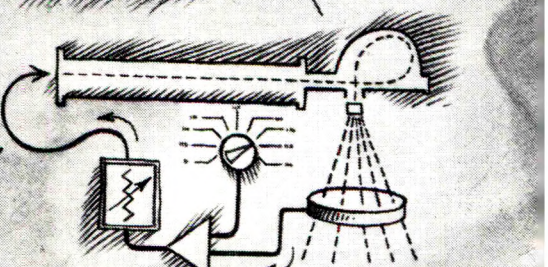
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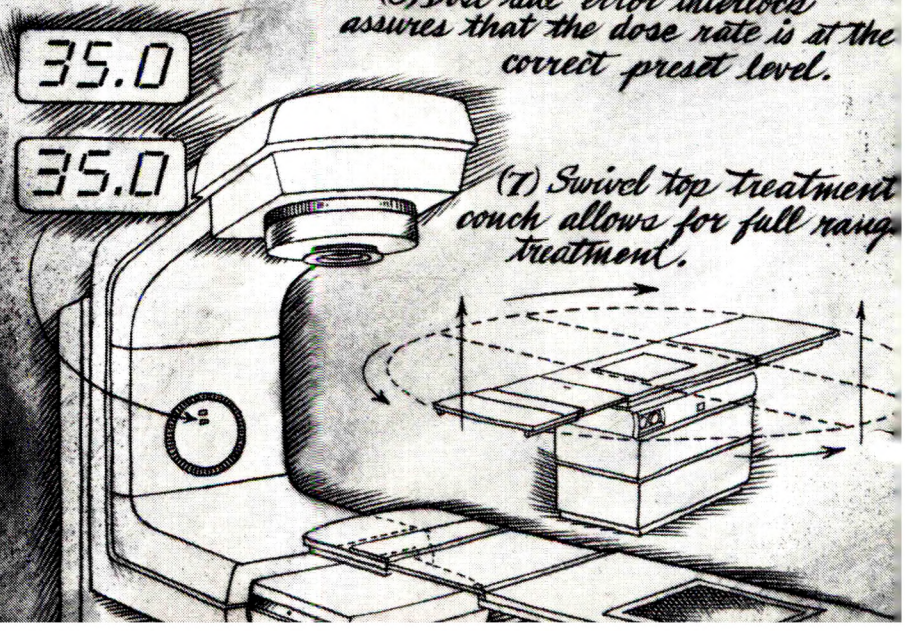


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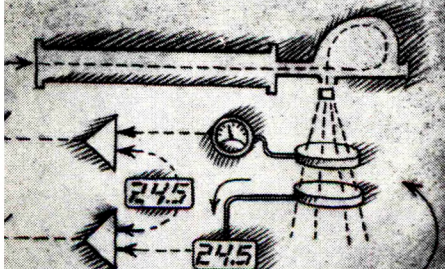


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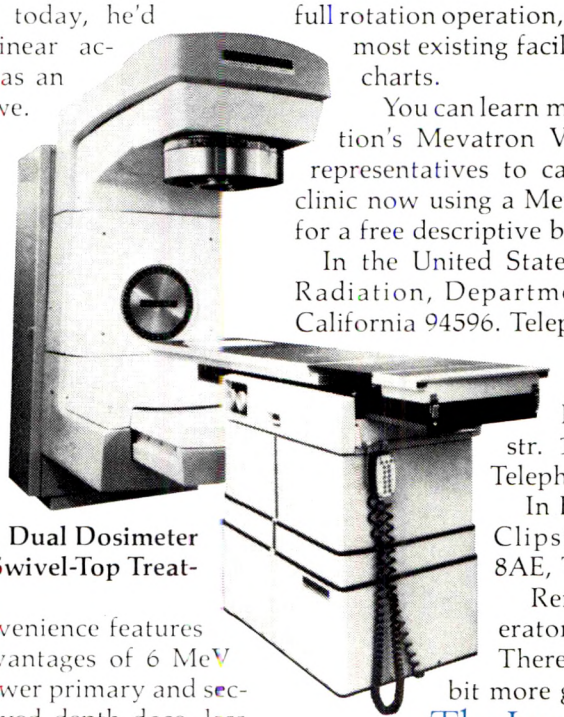
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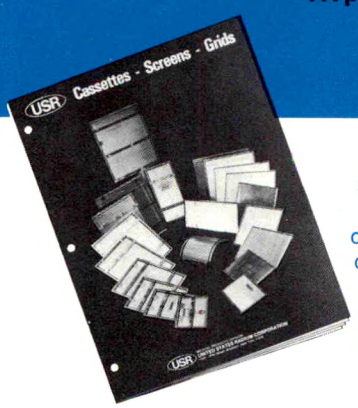
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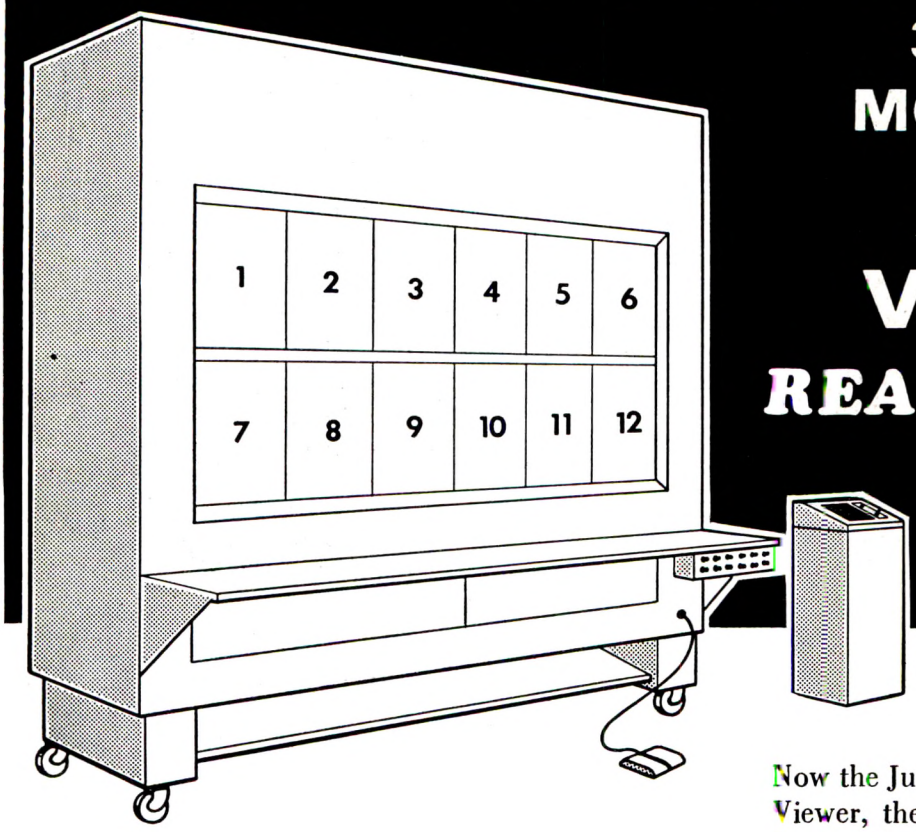
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SMALL BOWEL IN IMMUNOGLOBULIN DEFICIENCY SYNDROMES

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THE frequent occurrence of gastrointestinal disturbances in the various immunoglobulin deficiency syndromes was recognized soon after congenital agammaglobulinemia was first described by Bruton in 1952.[5] In fact, diarrhea and steatorrhea were features of the first reported case of idiopathic acquired (late onset) hypogammaglobulinemia[28] and were estimated in an early report to occur in 20 per cent of such cases.[11] The occurrence of a sprue syndrome[6] and of pernicious anemia and atrophic gastritis[21,30] in association with late onset hypogammaglobulinemia was soon recognized. A group of patients with acquired dysgammaglobulinemia was then identified,[15] characterized by nodular lymphoid hyperplasia of the mucosa and submucosa of the small intestine, with diarrhea and an unusual susceptibility to infection with *Giardia lamblia*. This infection has also been detected with undue frequency in other intestinal immunodeficiency syndromes,[3] as well as in alpha chain disease (Mediterranean lymphoma).

CLINICAL CLASSIFICATION

Recent discoveries in cellular immunology have contributed greatly to our understanding of the pathogenesis of diseases related to immune mechanisms. Of particular help has been the division of lymphocytes into thymus-processed T-cells, responsible mainly for cell-mediated, delayed type immune response, and unprocessed B-cells of bone-marrow origin that are involved in humoral, immunoglobulin-mediated immunity.

This paper is concerned with the immunoglobulin deficiency syndromes, presumably due to a deficiency in B-cell number or function, as opposed to cellular immune defects such as Hodgkin's disease that involve T-cells. Classification of these diseases is made difficult by the wide range of immunologic findings in patients suffering from them. Important factors to be considered in an attempt to classify the primary idiopathic immunodeficiencies are inheritance, association with certain well-defined clinical abnormalities, measure-

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TABLE I

IMMUNOGLOBULIN DISORDERS WITH GASTROINTESTINAL MANIFESTATIONS: CLINICAL CLASSIFICATION

I. *Primary*
- A. Variable immunoglobulin deficiency
  1. Hypogammaglobulinemic sprue
  2. Hypogammaglobulinemia with pernicious anemia and atrophic gastritis
  3. Dysgammaglobulinemia with nodular lymphoid hyperplasia of the small bowel and giardiasis.
- B. Selective I_gA deficiency
  1. I_gA deficient sprue
  2. Nodular lymphoid hyperplasia of the small bowel (uncommon)

II. *Secondary*
- A. Decreased immunoglobulin synthesis or increased synthesis of abnormal immunoglobulins (*e.g.*, plasma cell dyscrasias including Waldenström's macroglobulinemia and alpha-chain disease, lymphosarcoma, chronic lymphatic leukemia)
- B. Increased immunoglobulin breakdown and loss (*e.g.*, exudative gastroenteropathies, intestinal lymphangiectasia)
- C. Amyloidosis

ment of immunoglobulin levels, and estimation of both number and function of T-cells and B-cells.[7] None of these factors taken alone or in combination is satisfactory, however, for a really adequate classification.

The enteropathic immunoglobulin deficiencies are predominantly of acquired adult-onset type, since the infantile forms are rarely associated with gastrointestinal symptoms.[2] They may be either primary (idiopathic), or secondary to some other disease involving the gastrointestinal tract such as the exudative enteropathies and various lymphoreticular malignancies. Primary acquired late-onset immunoglobulin deficiency was initially divided into primary acquired agammaglobulinemia, with low levels of all 3 of the then-known immunoglobulins (I_gA, I_gG and I_gM) and dysgammaglobulinemia, with deficiency of only 1 or 2 of them.[11] Three types of dysgammaglobulinemia were described: Type I, the most common, with deficiency of I_gA and I_gG; Type II with decrease of I_gA and I_gM; and Type III with isolated absence of I_gA.

It is now apparent that these primary, late-onset immunoglobulin deficiency syndromes are a heterogeneous group, and that their classification based on various immunoglobulin patterns has not proved useful. In the classification recommended recently by an international World Health Organization committee,[10] selective immunoglobulin (I_gA) deficiency was classed separately from the other common acquired primary hypo- and dysgammaglobulinemias. These remain unclassified in the group termed by them "variable immunodeficiency," comprising the majority of patients with immunodeficiency.

It may be more useful for the purposes of this discussion to classify the various immunoglobulin disorders associated with gastrointestinal manifestations into the following main groups (Table 1):

I. PRIMARY IMMUNOGLOBULIN DISORDERS WITH GASTROINTESTINAL MANIFESTATIONS

A. Variable Immunoglobulin Deficiency

1. Hypogammaglobulinemic sprue.[6,9] Uniform depression of all immunoglobulin levels, with malabsorption and villous atrophy, but with absence of plasma cells in the lamina propria and no response to gluten exclusion.
2. Hypogammaglobulinemia with pernicious anemia[30] and atrophic gastritis, but with absence of antibodies to parietal cells and intrinsic factor, frequent lack of response of the B_{12} malabsorption to exogenous intrinsic factor, and lack of the expected elevation of the fasting serum gastrin level.[17] There is villous atrophy and lack of plasma cells in the small bowel.
3. Dysgammaglobulinemia with nodular lymphoid hyperplasia of the small bowel.[15] The immunoglobulin defect is incomplete and affects especially I_gA and I_gM in most cases.

There is malabsorption and a high incidence of intestinal infection with *Giardia lamblia*, *Strongyloides*, *Monilia*, bacteria and possibly viruses. Respiratory infections are frequent. The villi appear normal on small bowel biopsy, but there are few or no plasma cells found; lymphoid aggregates are seen.

B. Selective I_gA Deficiency

1. I_gA deficient sprue.[8,23] Isolated I_gA deficiency with gluten enteropathy, villous atrophy and presence of ample plasma cells in the lamina propria, although these are abnormal in that they contain I_gM and no I_gA. There is no unusual susceptibility to infection.
2. Isolated I_gA deficiency with nodular lymphoid hyperplasia also occurs,[13] although it is more commonly associated with dysgammaglobulinemia.

It must be noted that in several instances a single patient will show a combination of features, such as nodular lymphoid hyperplasia and giardiasis together with sprue-like villus atrophy,[18] rendering his disease difficult to classify according to the above scheme. There is an increased incidence of gastric[14] and colonic[16] carcinoma, as well as lymphoma of the bowel,[24] in all these enteropathic types of immunoglobulin deficiency.

One or more of the following factors may contribute to the etiology of the malabsorption that is so frequent in primary hypogammaglobulinemia: intramural intestinal infection; intraluminal bacterial overgrowth; *Giardia lamblia;* gastric achlorhydria; pancreatic insufficiency; and gluten sensitivity. The intestinal mucosal alterations may be secondary to bacterial or parasitic infection and often improve after their eradication.[2,3]

II. SECONDARY IMMUNOGLOBULIN DISORDERS WITH GASTROINTESTINAL MANIFESTATIONS

A. Decreased immunoglobulin synthesis and/or increased synthesis of abnormal immunoglobulin fragments. This disorder is found in Waldenström's macroglobulinemia, alpha-chain disease and other plasma cell dyscrasias. It has also been found in well differentiated lymphosarcoma (lymphocytic lymphoma) and chronic lymphatic leukemia. It is of interest that whereas these latter 2 represent disorders of humoral immunity, Hodgkin's disease reflects a derangement of T-cell function and cell-mediated immunity.[1]

Alpha-chain disease ("Mediterranean lymphoma") is a condition characterized by a diffuse plasma cell proliferation affecting the I_gA secretory system and synthesizing an abnormal immunoglobulin fragment which is part of the heavy chain of I_gA (alpha-chain).[4,25] In nearly all cases the plasma cell infiltrate has involved the whole length of the small intestine and mesenteric lymph nodes, although a respiratory form of the disease has also been reported. The clinical picture is usually that of a severe malabsorption syndrome affecting males or females generally in their second or third decades. The serum contains free alpha-chain with reduced levels of I_gG, I_gM and albumin. The pathologic counterpart is an abdominal lymphoma, originally termed Mediterranean lymphoma because of the peculiar ethnographic distribution of most of the earlier reported cases. Whether alpha-chain disease starts as a benign hyperplastic process or is malignant from its onset has not been determined.

B. Increased immunoglobulin breakdown and loss secondary to exudative gastroenteropathies, including intestinal lymphangiectasia.[29] The effect is similar to the losses that occur with the nephrotic syndrome and exfoliative dermatitides. The major fall here is in I_gG associated with loss of albumin, together with lymphopenia when secondary to intestinal lymphangiectasia.

C. Amyloidosis.[12] Nonspecific immunoglob-

ulin abnormalities such as elevated or depressed serum levels of I_gA, I_gG and I_gM, are frequently encountered in all types of amyloidosis, while M-components are found in the serum or urine of most patients with amyloidosis associated with lymphoproliferative disorders.

ROENTGEN FEATURES

With this clinical classification in mind, we have organized the roentgenographic manifestations of the enteropathic immunoglobulin deficiency-syndromes into the following groups:

1. *Sprue (malabsorption) pattern*, as seen in hypogammaglobulinemic sprue and in I_gA deficient sprue.
2. *Multiple nodular defects*, as seen in dysgammaglobulinemia and lymphoma.
3. *Inflammatory changes* secondary to giardiasis.
4. *Thickening of small intestinal folds*, as in amyloidosis, lymphoma, macroglobulinemia, plasma cell dyscrasias, and intestinal lymphangiectasia.

The roentgen alterations are not necessarily confined to just one of the above findings and a patient may exhibit more than one of these features either simultaneously or on successive examinations. In dysgammaglobulinemia, for example, the findings may be multiple.

1. SPRUE (MALABSORPTION) PATTERN

The roentgen findings in hypogammaglobulinemic sprue and in I_gA deficient sprue are indistinguishable from those seen in celiac sprue, with dilatation of the small bowel, segmentation, fragmentation and scattering of the barium column, and with hypersecretion causing dilution of barium distally (Fig. 1). Transient nonobstructive intussusceptions may be observed.

Rarely a sprue pattern is seen in diffuse intestinal lymphosarcoma. In such patients the distinctive roentgen features of lymphosarcoma are also present, such as nodularity, ulceration and irregular thickening of the folds.

2. MULTIPLE NODULAR DEFECTS

Multiple nodular defects are most commonly seen in dysgammaglobulinemia in association with giardiasis, isolated I_gA deficiency, diffuse immunoglobulin deficiency, and lymphosarcoma.

Nodular lymphoid hyperplasia associated with hypogammaglobulinemia affects predominantly the small intestine. Less frequently the colon and rectum are involved. The characteristic roentgen feature is a uniform distribution throughout the involved segment of intestine of innumerable tiny, smooth, nodular filling defects measuring 1 to 3 mm. in diameter.[16] The nodules are round and regular in outline and are not associated with other mucosal alterations. The small bowel is not dilated and the valvulae conniventes are not thickened or blunted. Microscopic examination of a peroral small bowel biopsy specimen

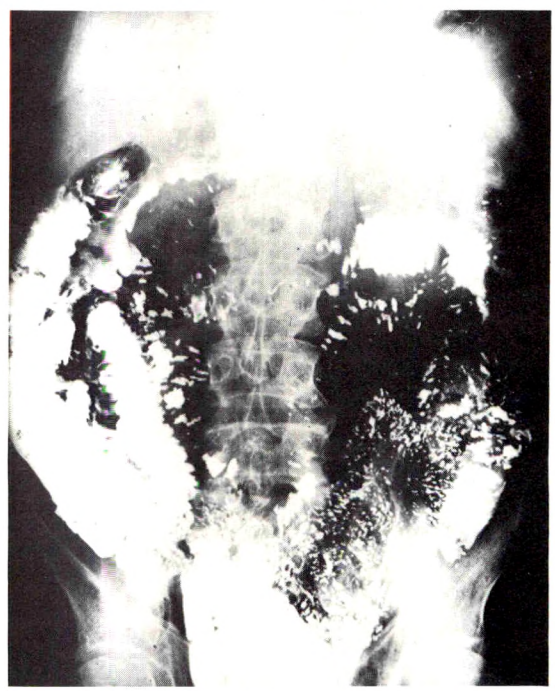


FIG. 1. *Hypogammaglobulinemic sprue.* The roentgen alterations are indistinguishable from celiac sprue. They include dilatation, especially of the jejunum, associated with thinning of the folds, fragmentation, segmentation, and increased secretions. Hypogammaglobulinemic sprue is rare and a diagnosis by roentgen examination alone cannot be made.

reveals the characteristic enlarged lymphoid follicles within the lamina propria causing effacement of the villus pattern of the overlying mucosa and imparting a nodular or polypoid appearance[15] (Fig. 2–9).

Although it is true that nodular lymphoid hyperplasia is usually associated with immunoglobulin abnormalities, it can be seen in the absence of such serum alterations. Normal serum values are more common when it is the colon that is involved. In children and young adults the terminal ileum alone may demonstrate multiple small symmetric nodules as a normal finding (Fig. 10).

Diffuse lymphosarcoma of the small intestine may also result in multiple nodules visualized on the roentgen study. These are usually larger and may produce irregular scalloping of the bowel contour or a cobblestone appearance. Ulcerations can frequently be demonstrated within the nodules. Rarely it can be very difficult to distinguish nodular lymphoid hyperplasia from lymphosarcoma, and in these patients follow-up roentgen studies and small bowel biopsy may be required for differentiation (Fig. 11; and 12).

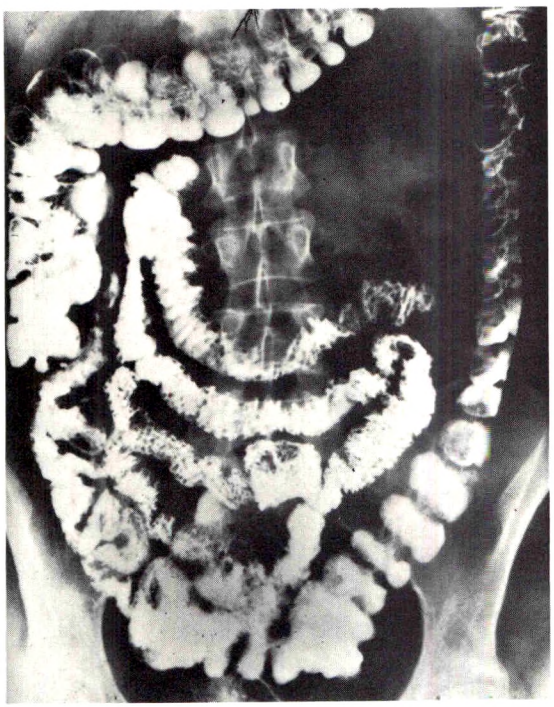


Fig. 2. *Dysgammaglobulinemia.* This patient was investigated because of diarrhea. Her I_gA levels were markedly reduced with borderline I_gG and I_gM. There are numerous small, symmetric 2–4 mm. nodules throughout the jejunum and ileum without evidence of ulceration. It is of interest that despite the administration of 20 fluid ounces of barium, the nodules were easily demonstrated. The colon is normal.

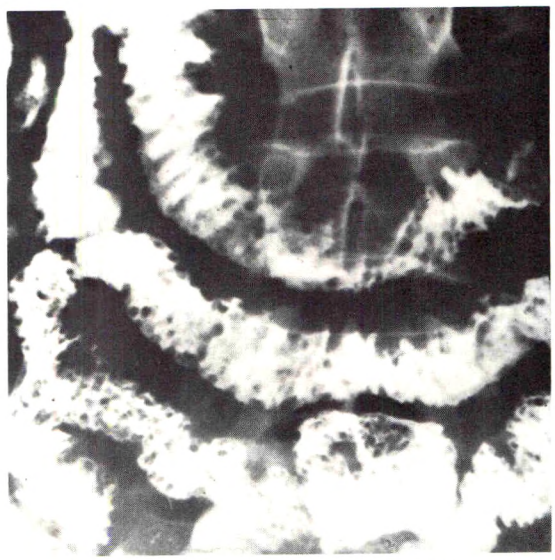


Fig. 3. *Dysgammaglobulinemia* (magnified view of Figure 2). Symmetric small nodules are identified. This is an excellent example of the typical nodules of nodular lymphoid hyperplasia.

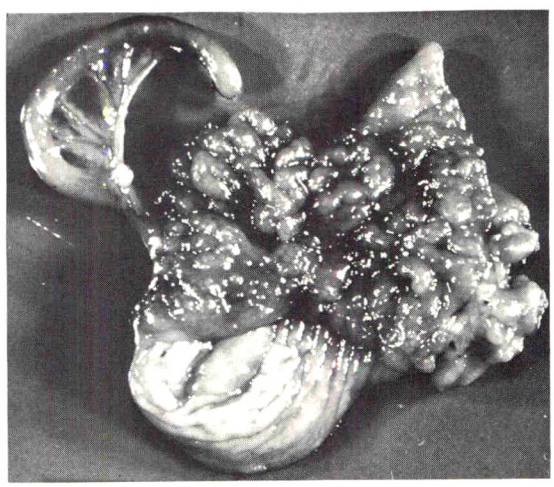


Fig. 4. *Specimen of distal ileum demonstrating nodular lymphoid hyperplasia.* Histologic section revealed marked lymphoid hyperplasia. The appendix is visualized and is normal.

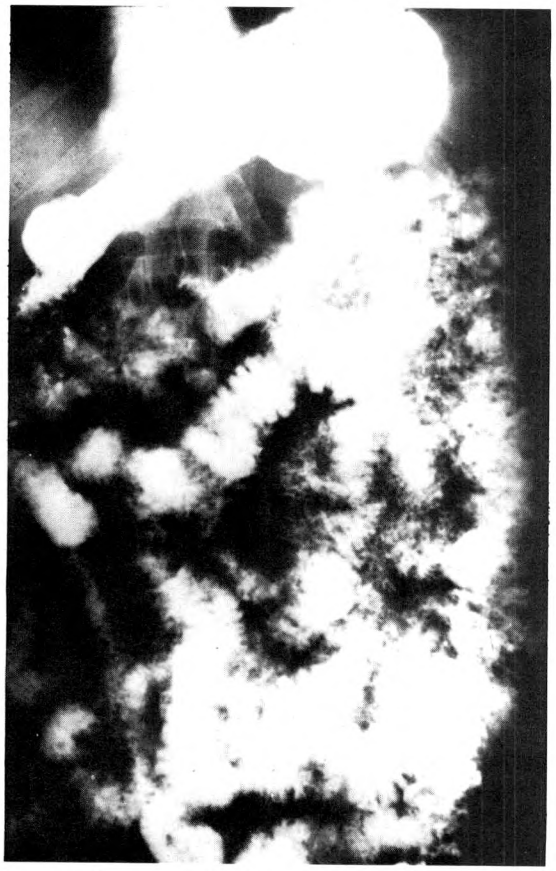


FIG. 5. *Dysgammaglobulinemia with reduced I_gA, I_gG and I_gM.* Numerous small nodules are seen distributed throughout the entire small bowel.

3. INFLAMMATORY CHANGES SECONDARY TO GIARDIASIS

The roentgen findings in *Giardia lamblia* infection are inflammatory in character and are usually limited to the duodenum and jejunum.[22] There is spasm and irritability, slight narrowing of the lumen with thickening and distortion of the folds. Increased intestinal secretions frequently

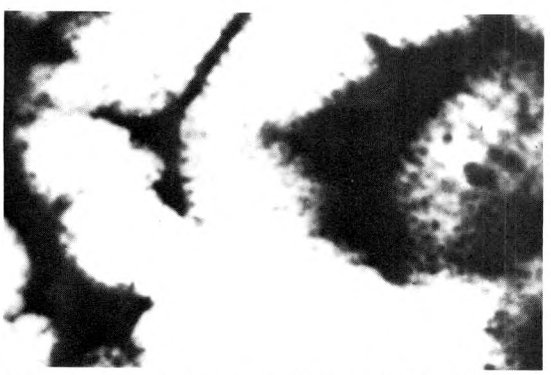


FIG. 6. *Magnified view of Figure 5.* The nodules are well demonstrated. They are characteristically small, round, regular, symmetric and demonstrate no evidence of ulceration.

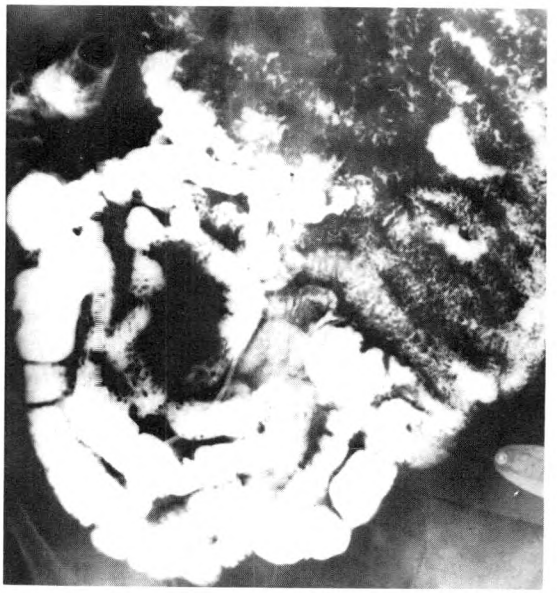


FIG. 7. *Selective immunoglobulin A deficiency.* There are numerous tiny nodules distributed throughout the entire small bowel, best visualized in the distal jejunum and proximal ileum. There is no evidence of increased secretions or ulcerations. This patient had vague abdominal complaints with diarrhea. Follow-up studies revealed no change after a period of 3 years. Small bowel biopsy confirmed the diagnosis of nodular lymphoid hyperplasia.

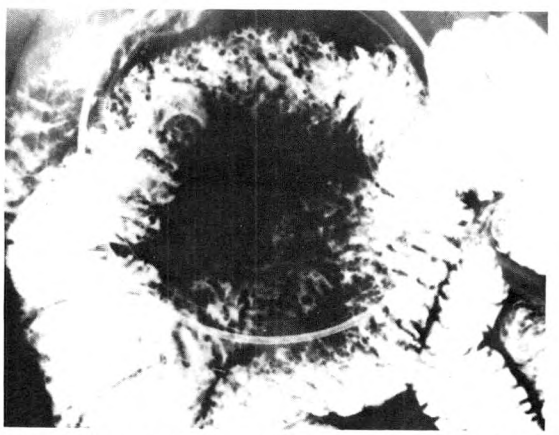


FIG. 8. *Same patient as in Figure 7.* Close-up of distal duodenum and jejunum, exquisitely delineating the tiny nodules typical of nodular lymphoid hyperplasia.

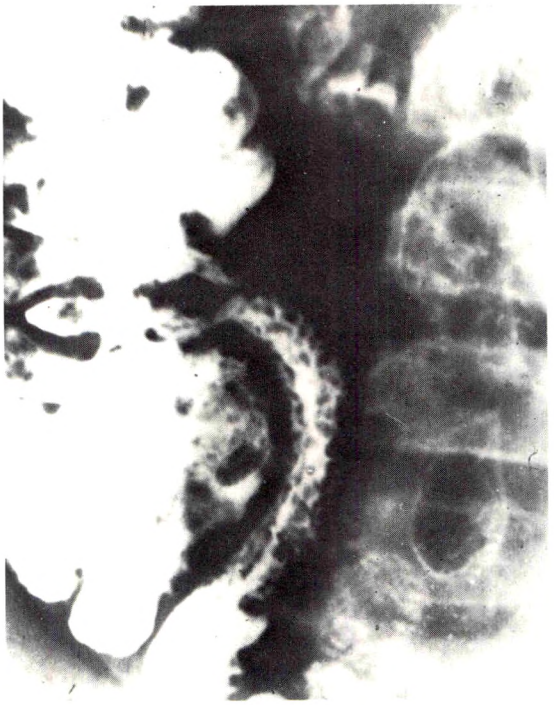


Fig. 10. *Terminal ileum with nodular lymphoid hyperplasia.* This patient had no immunoglobulin deficiencies. The alterations were confined to the terminal ileum and consisted of small nodular filling defects similar to those described above. When nodular lymphoid hyperplasia is restricted to the terminal ileum in young people, it is rarely of significance.

»»→

cause blurring of the folds and sometimes fragmentation and slight segmentation of the barium column. Transit through the jejunum may be rapid. The ileum usually appears normal (Fig. 13; and 14).

When giardiasis complicates a hypogammaglobulinemic enteropathy, the above roentgen changes may be associated with the multiple tiny filling defects of nodular lymphoid hyperplasia (Fig. 15) or, less commonly, with the sprue (mal-



Fig. 9. *Same patient as in Figures 7 and 8.* Magnified view of ileum. The multiple small nodules described above are again seen throughout the ileum.



Fig. 11. *Lymphosarcoma.* This case demonstrates the occasional difficulty in differentiating nodular lymphoid hyperplasia from lymphosarcoma. There are multiple small nodules, varying in size from 3 to 6 mm. in diameter. Some are irregular and others appear submucosal. There is no evidence of ulceration. Because the nodules varied in size, lymphosarcoma could not be excluded and follow-up study was advised.

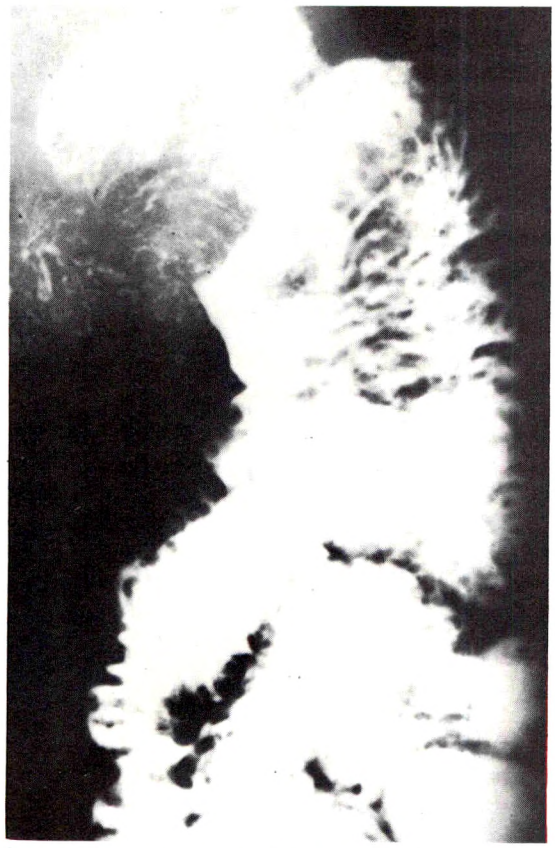


FIG. 12. *Lymphosarcoma.* Same patient as in Figure 11 four months later. There is a segment in the midjejunum measuring 3 inches in length in which the mucosa is ulcerated, with a nodular configuration along the contours. There is no obstruction. Some of the tiny defects previously described are seen in adjacent loops of bowel. At surgery, almost the entire small bowel was involved with lymphosarcoma. (Courtesy of Dr. Roger Blahut.)

absorption) pattern. Eradication of the parasite results in return of the fold pattern to normal, as well as improvement of the villus structure in hypogammaglobulinemic sprue,[3] regression of diarrhea, steatorrhea and malabsorption, and of lactose intolerance and abnormal intestinal protein loss.[2] The number and size of the nodular follicles are not affected, however, by treatment of the *Giardia* infection.

It is important to note that the presence of *Giardia lamblia* in the stool does not necessarily mean that the small bowel roentgen study will be abnormal; indeed, small bowel changes are uncommon in most patients with giardiasis. In epidemics of the infection and in patients with hypogammaglobulinemia, the roentgen studies are more likely to be abnormal. Conversely, *Giardia lamblia* may be absent from the stool, yet be present on duodenal aspirate or small bowel biopsy.

4. THICKENING OF THE SMALL INTESTINAL FOLDS

Thickening of the small bowel folds is not characteristic of the primary enteropathic immunodeficiencies, as classified earlier in this paper, except for superimposed *Giardia lamblia* infection. It is, however, a feature of the secondary immunoglobulin disorders.

Amyloidosis, by infiltration of the bowel



FIG. 13. *Giardiasis.* There is spasm and irritability of the jejunum, associated with narrowing of the lumen and increased secretions. The valvulae conniventes are thickened. The findings are secondary to inflammatory edema. The ileum is within normal limits.

wall, produces conspicuous thickening of the folds and this process tends to be uniform throughout the entire small bowel on roentgen study.[20] Enlargement of the folds in the ileum gives an appearance resembling the normal feathery pattern of the jejunum, a configuration that has been termed "jejunization of the ileum." There is no significant dilatation or increased secretions, and nodules are not present (Fig. 16).

When amyloidosis involves the colon, it produces roentgen alterations that are similar to those of chronic ulcerative colitis, with absent haustral markings but no significant shortening (Fig. 17; and 18). Ascites is often present and can be recognized by separation of the loops of small bowel. In the stomach amyloid infiltration produces a polypoid tumor or features similar to those of scirrhous carcinoma.

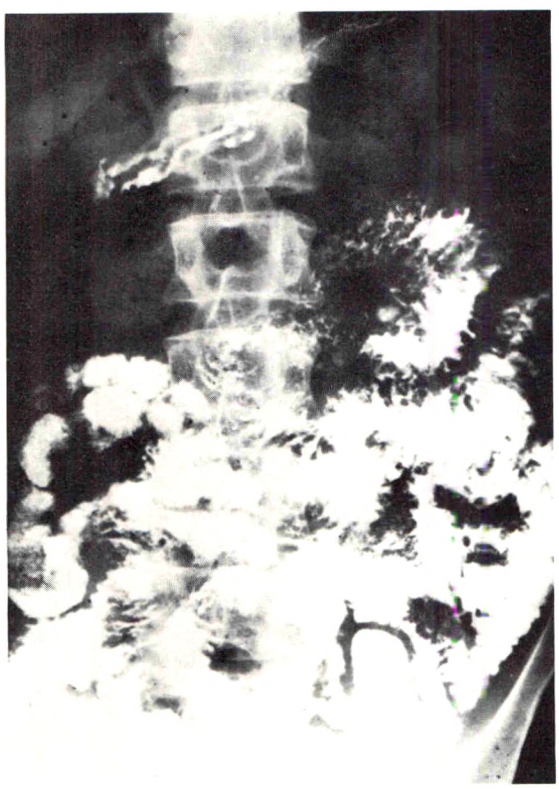


Fig. 14. *Giardiasis.* In this case the roentgen alterations are more pronounced. There is more spasm and irritability and the folds are more thickened and distorted. In this patient the roentgen appearance of the small bowel returned to normal after treatment of giardiasis with atabrine.



Fig. 15. *Giardiasis.* There is thickening of the folds of the jejunum. In the proximal ileum, the small nodular filling defects typical of nodular lymphoid hyperplasia are identified. The correct diagnosis of giardiasis was made and *Giardia lamblia* was identified in the stool. However, at that time the nature of the nodules in the ileum and their association with immunoglobulins was not appreciated. There were no immunoglobulin studies performed in this case.

Lymphosarcoma may also appear as a smooth, diffuse thickening of the intestinal folds throughout the small bowel, without hypersecretion. The thickened folds are more commonly irregularly nodular and coarse marginal scalloping of the bowel contour may be seen (Fig. 19).

The roentgenographic features of *alpha-chain disease* are variable. The small intestinal folds may be thickened in an irregular fashion (Fig. 20). Multiple nodular defects may be demonstrated having the same characteristics as in lymphosarcoma and involving the whole length of the small bowel. A sprue-like pattern may also be seen with dilatation, which may be quite marked, involving both jejunum and ileum, increased secretions, and flocculation and



FIG. 16. *Amyloidosis.* There is a striking, symmetric, diffuse, sharply demarcated thickening of the valvulae conniventes. Dilatation is minimal. Segmentation and fragmentation are absent. There is no evidence of an inflammatory process.

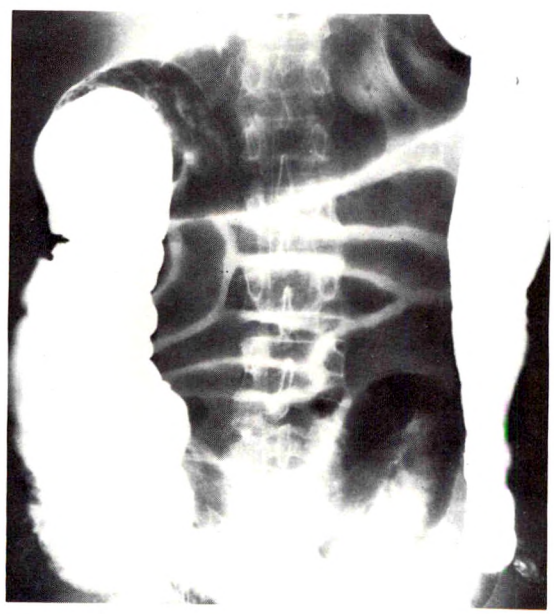




FIG. 18. *Amyloidosis.* Gross specimen reveals marked thickening of the wall. This thickening is responsible for the roentgen findings described above.



FIG. 19. *Lymphosarcoma.* Multiple tumor nodules of varying size are present in the terminal ileum, cecum and ascending colon. The terminal ileum is slightly dilated. The nodules are obviously larger and less symmetric than those associated with nodular lymphoid hyperplasia.

FIG. 17. *Amyloidosis.* There is a complete absence of the haustral markings throughout the colon, and the configuration resembles ulcerative colitis. The air-filled loops of small bowel are separated by the presence of fluid in the abdomen.

segmentation of the barium. Masses of enlarged mesenteric lymph nodes may produce extrinsic compression or dislocation of the small bowel.

A group of patients with marked plasma cell and lymphocyte infiltration of the small bowel has been described in whom the process, over a follow-up of years, has not become lymphoma. Such patients have a variety of serum immunoglobulin defects ranging from deficiency to excessive levels. This group probably represents a heterogeneous collection of abnormalities that await classification. They have in common, however, roentgen alterations of thickening of the folds and at times submucosal or intraluminal nodules. Patients with these findings frequently present clinically with diarrhea and malabsorption[30] (Fig. 21).

Uniform thickening of the folds of the small bowel is also found in some cases of *Waldenström's macroglobulinemia*.[19] The wall of the intestine is thickened, as evidenced by separation of the loops on small bowel series, and the lumen may be moderately dilated. The mucosal surface itself may have a fine granular appearance, perhaps caused by club-like dilatation and distention of the tips of the villi (Fig. 22). The



Fig. 21. *Plasma cell infiltration of the small bowel.* There is no moderate thickening of the valvulae conniventes, especially in the jejunum. Increased secretions are present. The relationship of these cases to alpha-chain disease has not been established.

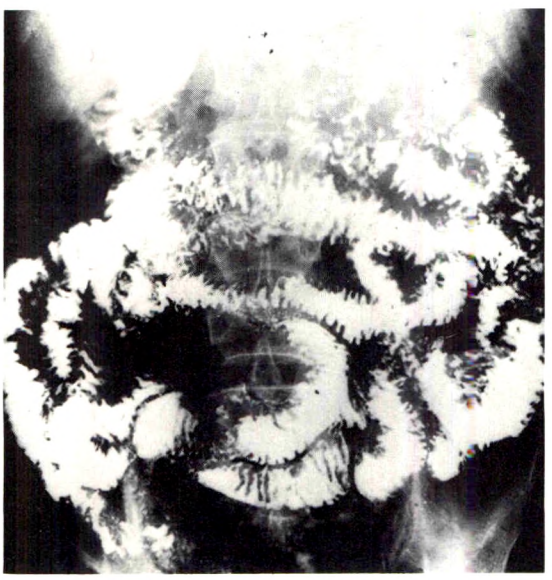


Fig. 20. *Alpha-chain disease.* This entity was previously described as Mediterranean lymphoma. Not enough cases have yet been studied to delineate a definitive roentgen pattern. In this case, there is thickening of the folds, associated in some areas with minimal narrowing of the lumen. No other abnormalities are noted. In some cases, flat nodules along the contour of the bowel have been described.

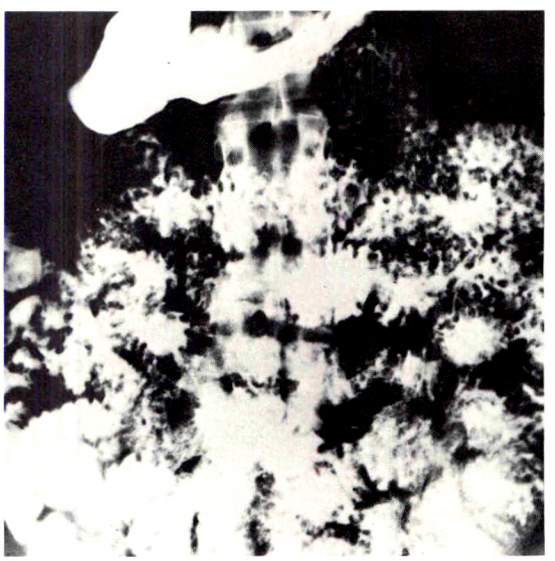


Fig. 22. *Waldenström's macroglobulinemia.* There is diffuse thickening of the valvulae conniventes associated with a granular mucosal surface. In some areas it appears almost nodular. (Courtesy of Dr. Mansho Khilnani.)



FIG. 23. *Intestinal lymphangiectasia.* The entire small bowel is involved. There is minimal segmentation and fragmentation, associated with diffuse thickening of the valvulae conniventes and moderate secretions. There is no dilatation. The serum albumin in this patient was 1 gm. per cent. The alterations described above are probably due to intestinal edema secondary to hypoalbuminemia.

roentgen characteristics of sprue are not present. It must be said, however, that only a few cases of Waldenström's macroglobulinemia involving the intestinal tract have been studied thus far and these observations must be considered provisional.

Intestinal lymphangiectasia is an exudative, protein-losing enteropathy with gross dilatation of the lymphatics of the lamina propria of the small bowel reflecting a generalized disorder of the development of lymphatic channels.[29] Hypogammaglobulinemia, mainly affecting I_gG, is associated with a fall in serum albumin and, to a lesser degree, other proteins, as well as with the loss of lymphocytes into the bowel lumen. Enlargement of folds on roentgen examination usually affects both the jejunum and the ileum. The pattern of fold enlargement is uniform, orderly and symmetrical in a single loop. A stacked-coin appearance is not uncommon and the folds may have a regular biconvex appearance (Fig. 23; and 24). Large folds in the ileum may result in its "jejunization" as described in some cases of amyloidosis. Excessive intestinal secretions result in dilution of the barium column distally in most cases and are an important distinguishing feature. Dilatation is minimal or absent. The thickening of the folds noted roentgenologically in intestinal lymphangiectasia represents mucosal and submucosal edema secondary to hypoproteinemia.

SUMMARY

Recent advances in immunology have permitted recognition of a group of pa-

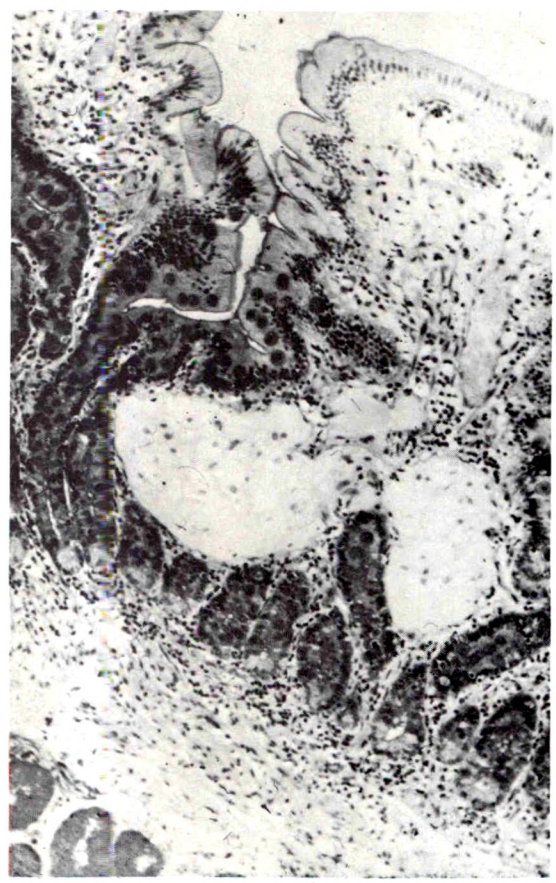


FIG. 24. *Intestinal lymphangiectasia.* Histologic sections demonstrate marked dilatation of the lymphatics as well as edema of the mucosa.

tients who have gastrointestinal manifestations as part of an immunoglobulin deficiency syndrome. Such immunoglobulin deficiency may be primary or may be secondary to a variety of diseases.

We have classified and described the small bowel roentgen features associated with the various immunoglobulin deficiency syndromes as follows: (1) the sprue pattern, as seen in hypogammaglobulinemic sprue and in I_gA deficient sprue; (2) multiple nodular defects, as seen in dysgammaglobulinemia; (3) inflammatory changes secondary to giardiasis, associated with immune deficiency diseases; and (4) thickening of the small intestinal folds, as seen in the plasma cell dyscrasias, lymphoma, intestinal lymphangiectasia, and amyloidosis.

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REFERENCES

1. Aisenberg, A. C. Malignant lymphoma. *New England J. Med.*, 1973, *288*, 883–890.
2. Ament, M. E., Ochs, H. D., and Davis, S. D. Structure and function of gastrointestinal tract in primary immunodeficiency syndromes: study of 39 patients. *Medicine*, 1973, *52*, 227–248.
3. Ament, M. E., and Rubin, C. E. Relation of giardiasis to abnormal intestinal structure and function in gastrointestinal immunodeficiency syndromes. *Gastroenterology*, 1972, *62*, 216–226.
4. Bonomo, L., Dammacco, F., Marano, R., and Bonomo, G. M. Abdominal lymphoma and alpha chain disease. *Am. J. Med.*, 1972, *52*, 73–86.
5. Bruton, O. C. Agammaglobulinemia. *Pediatrics*, 1952, *9*, 722–728.
6. Cohen, N., Paley, D., and Janowitz, H. D. Acquired hypogammaglobulinemia and sprue: report of case and review of literature. *J. Mt. Sinai Hosp.*, 1962, *28*, 421–427.
7. Cooper, M. D., Faulk, W. P., Fudenberg, H. H., Good, R. A., Hitzig, W., Kunkel, H., Rosen, F. S., Seligmann, M., Soothill, J., and Wedgwood, R. J. Classification of primary immunodeficiencies. *New England J. Med.*, 1972, *288*, 966–967.
9. Eidelman, S., Davis, S. D., and Rubin, C. E. Immunologic studies in "hypogammaglobulinemic sprue." *Clin. Res.*, 1968, *16*, 117.
10. Fudenberg, H., Good, R. A., Goodman, H. C., Hitzig, H., Kunkel, H. G., Roitt, I. M., Rosen, F. S., Rowe, D. S., Seligmann, M., and Soothill, J. R. Primary immunodeficiencies: report of World Health Organization Committee. *Pediatrics*, 1971, *47*, 927–946.
11. Gitlin, D., Gross, P. A. M., and Janeway, C. A. Gamma globulins and their clinical significance. II. Hypogammaglobulinemia. *New England J. Med.*, 1959, *260*, 72–76.
12. Glenner, G. C., Ein, D., and Terry, W. D. Immunoglobulin origin of amyloid. *Am. J. Med.*, 1972, *52*, 141–147.
13. Gryboski, J. D., Self, T. W., Clemett, A., and Herskovic, T. Selective immunoglobulin A deficiency and intestinal nodular lymphoid hyperplasia: correction of diarrhea with antibiotics and plasma. *Pediatrics*, 1968, *42*, 833–837.
14. Hermans, P. E., and Huizenga, K. A. Association of gastric carcinoma with idiopathic late-onset immunoglobulin deficiency. *Ann. Int. Med.*, 1972, *76*, 605–609.
15. Hermans, P. E., Huizenga, K. A., Hoffman, H. N., II, Brown, A. L., Jr., and Markovitz, H. Dysgammaglobulinemia associated with nodular lymphoid hyperplasia of small intestine. *Am. J. Med.*, 1966, *40*, 78–89.
16. Hodgson, J. R., Hoffman, H. N., II, and Huizenga, K. A. Roentgenologic features of lymphoid hyperplasia of small intestine associated with dysgammaglobulinemia. *Radiology*, 1967, *88*, 883–888.
17. Hughes, W. S., Brooks, F. P., and Conn, H. O. Serum gastrin levels in primary hypogammaglobulinemia and pernicious anemia: studies in adults. *Ann. Int. Med.*, 1972, *77*, 746–750.
18. Hughes, W. S., Cerda, J. J., Holtzapple, P., and Brooks, F. P. Primary hypogammaglobulinemia and malabsorption. *Ann. Int. Med.*, 1971, *74*, 903–910.
19. Khilnani, M. T., Keller, R. J., and Cuttner, J. Macroglobulinemia and steatorrhea: roentgen and pathologic findings in intestinal tract. *Radiol. Clin. North America*, 1969, *7*, 43–55.
20. Korelitz, B. I., and Spindell, L. N. Gastrointestinal amyloidosis: report of case and review of clinical and radiologic aspects. *J. Mt. Sinai Hosp.*, 1956, *23*, 683–696.
21. Lewis, E. C., II, and Brown, H. E., Jr. Agammaglobulinemia associated with pernicious anemia and diabetes mellitus. *A.M.A. Arch. Int. Med.*, 1957, *100*, 296–299.
22. Marshak, R. H., Ruoff, M., and Lindner, A. E. Roentgen manifestations of giardiasis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, 104, 557–560.
23. Mawhinney, H., and Tomkin, G. H. Gluten

enteropathy associated with selective I_gA deficiency. *Lancet*, 1971, *2*, 121–124.

24. PETERSON, R. D. A., COOPER, M.D., and GOOD, R. A. Pathogenesis of immunologic deficiency diseases. *Am. J. Med.*, 1965, *38*, 579–604.
25. RAMBAUD, J. C., and MATUCHANSKY, C. Alpha-chain disease: pathogenesis and relation to Mediterranean lymphoma. *Lancet*, 1973, *1*, 1430–1432.
26. ROSEN, F. S., and JANEWAY, C. A. Gamma globulins. III. Antibody deficiency syndromes. *New England J. Med.*, 1966, *275*, 709–715; 769–775.
27. RUOFF, M., LINDNER, A. E., and MARSHAK, R. H. Malabsorption syndrome with plasma cell infiltration of small bowel. *Am. J. Gastroenterol.*, 1971, *55*, 602–608.
28. SANFORD, J. P., FAVOUR, C. B., and TRIBEMAN, M. S. Absence of serum gamma globulins in adult. *New England J. Med.*, 1954, *250*, 1027–1029.
29. SHIMKIN, P. M., WALDMANN, T. A., and KRUGMAN, R. L. Intestinal lymphangiectasia. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *110*, 827–841.
30. TWOMEY, J. J., JORDAN, P. H., JARROLD, T., TRUBOWITZ, S., RITZ, N. D., and CONN, H. O. Syndrome of immunoglobulin deficiency and pernicious anemia. *Am. J. Med.*, 1969, *47*, 340–350.

Vol. 122, No. 2

TRANSIENT MALABSORPTION IN INFECTIOUS MONONUCLEOSIS*

CASE REPORT

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INFECTIOUS mononucleosis (IM) is usually a mild disease presumably caused by a viral agent. Of the many symptoms ascribed to the disorder, diarrhea is uncommon, occurring in only 5 to 10 per cent of patients.[1] In the few autopsy studies available, little mention is made of pathologic changes in the small bowel or colon.[2]

We have recently had the opportunity to study a patient with IM who had clinical, biochemical and roentgenologic evidence of malabsorption. No other cause for the malabsorption was found and over a period of 16 weeks all measurements of small bowel function returned to normal.

To our knowledge this is the first case of documented small bowel involvement with malabsorption secondary to IM.

REPORT OF A CASE

A 19 year old Caucasian male presented with a 2 week history of pharyngitis, malaise, 12 pound weight loss and diarrhea. The diarrhea was usually preceded by abdominal cramps and consisted of 3–5 loose stools per day. He denied hematochezia and melena and his only trip outside the continental United States had been to Hawaii.

Physical examination was unremarkable except for an exudative pharyngitis with tender posterior cervical lymphadenopathy. There was no organomegaly or abdominal distention noted.

Leukocyte count was 11,000 per ml.[3] with 64 per cent lymphocytes, many of which were atypical. A mono spot test was positive and a heterophil titer was 1:1,774. The serum carotene was 30 mg. per cent, d-xylose excretion, (25 gm. oral dose), was 2 gm. in 5 hours and a 72 hour stool fat was 32 gm. The stools were persistently guaiac-negative and no ova or parasites were found. A duodenal aspirate revealed no pathogens. A small bowel roentgenogram showed extensive thickening of the mucosal folds (Fig. 1) and a biopsy of the jejunum showed blunting of some villi with an inflammatory infiltrate in the lamina propria (Fig. 2; 3; and 4). There was no evidence of lymphangiectasia and the brush border appeared intact.

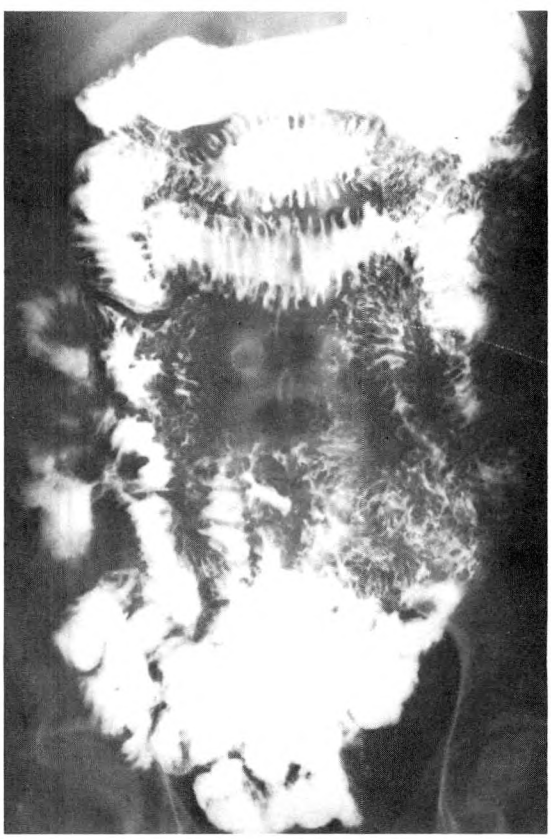


FIG. 1. Small bowel series. Thickened mucosal folds are seen throughout the entire small bowel.

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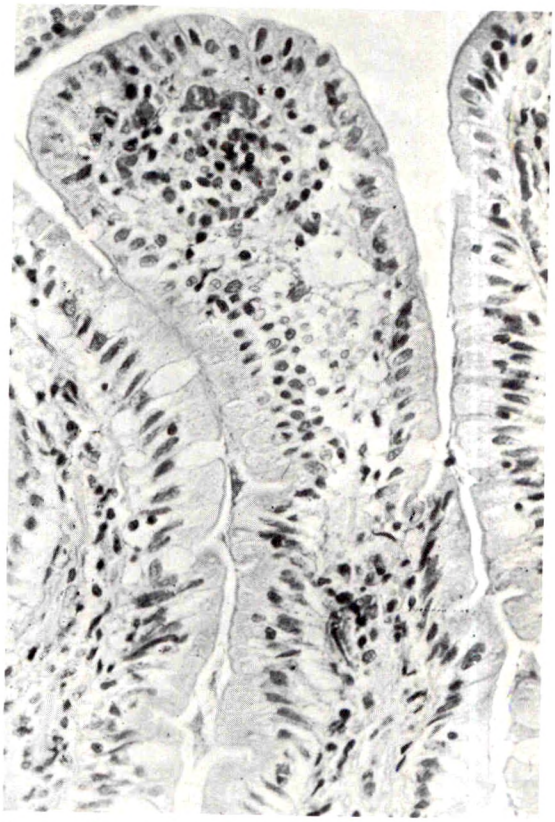


Fig. 2. Intact intestinal villus with an increased cellular infiltrate in the lamina propria (×95).

Acute Ebstein-Barr viral titers were not obtained; however, convalescent titer was 1:20. Other laboratory data are presented in Table 1.

Over the next 4 months the patient gained weight, recovered his sense of well-being and had resolution of his diarrhea without any drug or diet therapy. The mono spot test reverted to negative after 8 weeks and a subsequent small bowel series done 2 months after his initial hospitalization showed a return toward normal (Fig. 5). A repeat small bowel biopsy obtained 3 months after the onset of symptoms showed no histologic abnormalities (Fig. 6). At follow-up 6 months after admission the patient had no complaints.

DISCUSSION

Despite the voluminous amount of material written regarding IM, there is little evidence to suggest that the disease involves the small bowel. Sheehy *et al.*[6] performed jejunal biopsy in 6 patients with IM during the acute phase of the disease. They noted no abnormalities except for

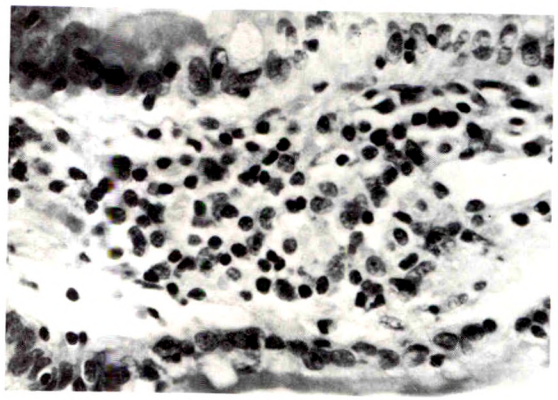


Fig. 3. Higher magnification of the same area as in Figure 2, showing large, somewhat atypical lymphocytes in the lamina propria (×370).

"numerous atypical and vacuolated lymphocytes within the lamina propria of four biopsy specimens." The epithelial cells appeared normal, as did the villous height, and none of their 6 patients had abnormal d-xylose excretion or steatorrhea.

Roentgenologic findings in IM are nonspecific. Splenomegaly, which occurs in 50 to 60 per cent of patients, can often be seen on chest or abdominal roentgenograms.[1] In perhaps 10 per cent of cases, roentgenologic evidence of hilar or mediastinal lymph node enlargement is found[4] and parenchymal pulmonary involvement occurs in 3 to 5 per cent.[1] In this latter group, the findings range from interstitial changes to alveolar consolidation, which rarely may cavitate.[7]

The current literature cites no cases of small intestinal abnormalities in patients with IM. The roentgenologic pattern in our patient demonstrated thickened mucosal



Fig. 4. Low power view showing some clubbing of villi and cellular infiltrate (×51).

Table I

INVESTIGATIVE DATA

	On Admission	10 wk. Later	Normal Values
Hb (g./100 ml.)	13.6	13.0	(14–18 gm. per cent)
Serum B_{12} (pg./ml.)	940	—	(170–700 pg./ml.)
Calcium (mg./100 ml.)	7.5	8.8	(8.5–10.5 mg. per cent)
Total proteins (g./100 ml.)	6.2	7.2	(6.0–8.0 gm. per cent)
Albumin (g./100 ml.)	3.0	4.2	(56–68 per cent of total)
D-xylose (g./5 hr.)	2.0	5.2	(4.0–8.8 gm./5 hr.)
Carotene (mg./100 ml.)	30	53	(40–100 mg. per cent)

folds extending throughout the small bowel as well as moderate dilatation. Nodularity, separation of loops, and increased secretions were not prominent features and motility was normal. The differential diagnosis included causes of small bowel edema, such as hypoproteinemia, and other causes of thickened folds, especially lymphoma.[3] The latter was less probable without nodularity.

The occurrence of the Ebstein-Barr (EB) virus antibody in a dilution of 1:20 is compatible with the diagnosis of IM, but does not "prove" a recent attack. Indeed, the EB virus may be a "fellow traveler" rather than the etiologic agent in patients who have IM.[5]

The possibility that the patient had a serious underlying disease, particularly lymphoma, was considered and justified the involved diagnostic approach taken. The subsequent return of clinical, biochemical and histologic parameters to normal rules out other more ominous causes of malabsorption.

In view of the rarity of reports of bowel involvement in IM, the authors thought it advisable to report this patient and currently are engaged in a prospective study of patients with IM and gastrointestinal complaints, to further evaluate the frequency of such involvement.

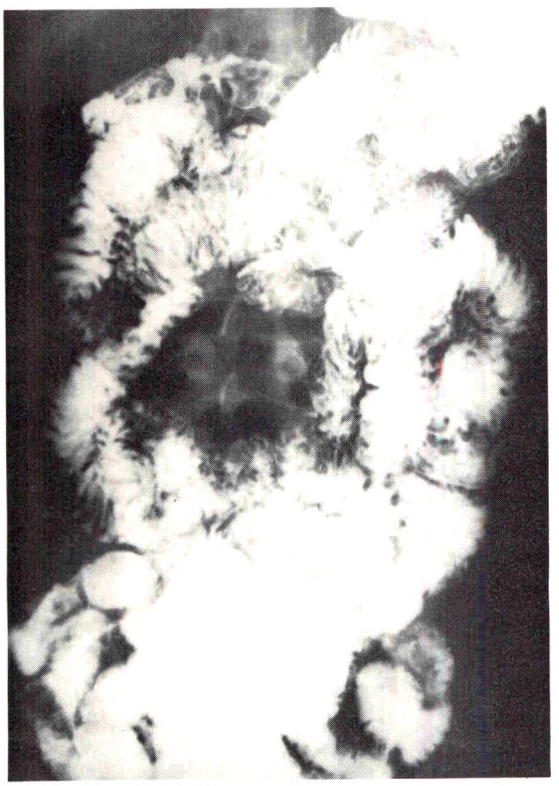


Fig. 5. Two months after the original study, the small bowel folds have decreased in thickness, and the entire small bowel pattern has nearly returned to normal.

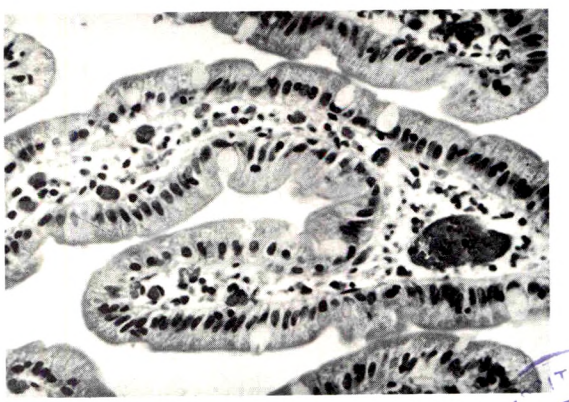


Fig. 6. Intestinal villus appears normal. The atypical lymphocytes are no longer present (×370).

L BRARY

SUMMARY

A patient with infectious mononucleosis and diarrhea was studied and found to have clinical, biochemical and roentgenologic evidence of malabsorption.

Small bowel biopsy showed infiltration of the lamina propria with atypical mononuclear cells as well as clubbed villi.

No other cause for the malabsorption was found and over a 16 week period all parameters of small bowel function returned to normal as did the jejunal biopsy.

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REFERENCES

1. Carter, R. L., and Penman, H. G. Infectious Mononucleosis. Blackwell Scientific Publications, Oxford, 1969, p. 23.
2. Hoagland, R J. Clinical manifestations of infectious mononucleosis: report of 200 cases. *Am. J. M. Sc.*, 1960, *240*, 21–29.
3. Marshak, R. H., and Linder, A. E. Radiology of Small Intestine. W. B. Saunders Company, Philadelphia, 1970, p. 365.
4. Mundy, G. R. Infectious mononucleosis with pulmonary parenchymal involvement. *Brit. M. J.*, 1972, *1*, 219–220.
5 Pullen, H. Infectious mononucleosis. *Brit. M. J.*, 1973, *1*, 350–352.
6. Sheehy, T. W., Artenstein, M. S., and Green, R. W. Small intestinal mucosa in certain viral diseases. *J.A.M.A.*, 1964, *109*, 1023–1028.
7. Teplick, J. G., Haskin, N. E., and Schimert, A. P. Roentgenologic Diagnosis. W. B. Saunders Company, Philadelphia, 1967, p. 746.

VOL. 122, No. 2

INTESTINAL AMEBIASIS*

A ROENTGEN ANALYSIS OF 19 CASES INCLUDING 2 CASE REPORTS

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INTESTINAL amebiasis often presents a difficult differential diagnostic problem, since it mimics a large variety of disease entities of the colon. At the one end of the spectrum, the diffuse amebic dysentery resembles acute idiopathic ulcerative colitis,[4,18] and at the other end, the localized granuloma, the ameboma, resembles carcinoma of the colon.[1,9,24,28]

The diagnosis of intestinal amebiasis may be readily made in some cases by the history of the patient and by the demonstration of *Entamoeba histolytica* in the stools or in the rectal biopsy. However, quite often the lesions are inaccessible to biopsy, and the results of the stool examinations, the rectal scrapings, the rectal biopsies and cultures may not be conclusive. A negative result does not rule out amebiasis and the presence of amebas does not exclude other ulcerative diseases of the colon.[23,24,28,32] Laparotomy as a means of diagnosis or for resection of the lesion is dangerous and contraindicated in the untreated patient.[15,21] The radiologist, therefore, has an important role to play in the differential diagnosis of this disease.

Therapeutic tests with antiamebic drugs for the purpose of conclusive diagnosis have long been advocated,[23,28] but reports of the roentgen diagnosis based on the response of the amebic lesions to the new fast-acting antiamebic drug, metronidazole, is lacking in the literature.[6,17,22,27] Although detailed comparative roentgen analysis of some ulcerative diseases of the colon such as ulcerative colitis *versus* granulomatous colitis has been presented,[2,18,20,29,30] reports of a similar approach in amebiasis are rarely found in the literature.[9,24,32]

The purpose of this paper is to report 2 cases of intestinal amebiasis to demonstrate the importance of the therapeutic test, the "metronidazole test," in the conclusive roentgen diagnosis of intestinal amebiasis and to present a comparative roentgen analysis of the features of amebiasis based on a review of the literature and on 19 of our cases of amebiasis that had positive roentgenologic studies.

A combination of features in amebiasis, as in ulcerative and granulomatous colitis, may raise the strong possibility of intestinal amebiasis. A dramatic roentgen change in 5 to 10 days of treatment with the new antiamebic drug metronidazole may be a most gratifying and unique feature of amebiasis in the complex differential diagnosis of ulcerative diseases of the colon.

REPORT OF CASES

CASE 1. A 45 year old man was admitted with the complaint of bouts of diarrhea alternating with constipation of 1 month's duration. This was accompanied by a 5 kg. weight loss, anorexia and occasional abdominal pain. On physical examination, the liver was not enlarged, but there was mild tenderness in the periumbilical region. The stool examination was negative for ameba and ova. The guaiac test was ++ positive, erythrocyte sedimentation rate was 57 mm./hour, and the remainder of the laboratory data were negative.

The barium enema examination showed an area of spasm in the midtransverse colon (Fig. 1*A*) which was also tender to palpation. In subsequent views, a persistent lesion was present in this area with eccentric loss of haustration at

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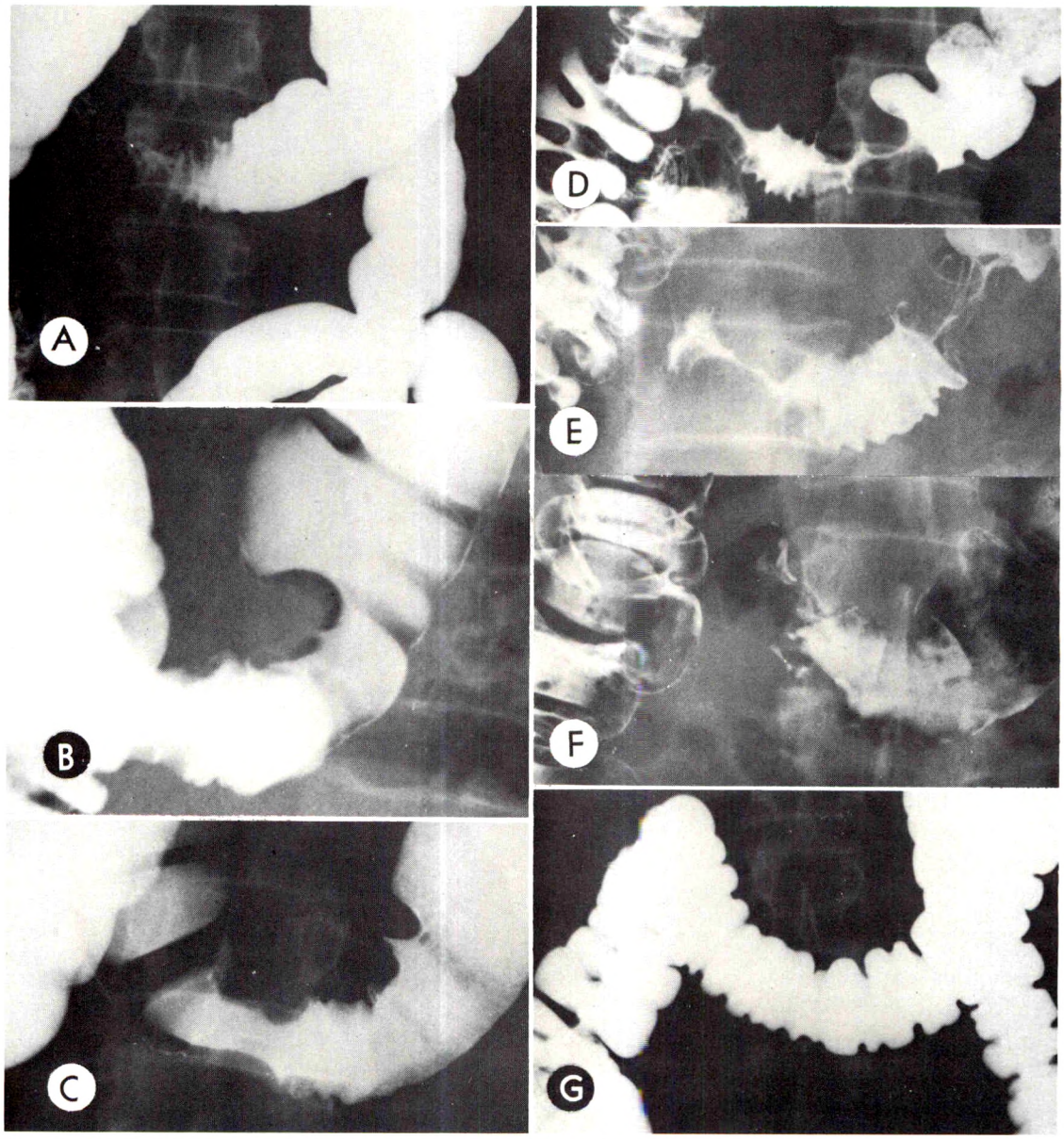


FIG. 1. Case 1. Roentgenograms show: (*A*) spasm in the midtransverse colon; (*B* and *C*) concentric and eccentric narrowing of the lumen; (*D* and *E*) thumbprinting at the ends of the lesion and exaggeration of haustral markings in the intervening fairly well preserved area; and (*F*) lack of distensibility as demonstrated under air pressure. (*G*) Ten days after the beginning of metronidazole therapy, there is complete reversal of all the roentgenologic signs of the disease except for minimal edema of the haustral folds.

one end of the lesion and concentric narrowing at the other end (Fig. 1, *B* and *C*). On the post-evacuation roentgenogram, thumbprinting was present at both ends with an intervening area of milder involvement that showed exaggeration of the haustral markings (Fig. 1, *D* and *E*). The air contrast study showed some distensibility of the narrowed lumen and disappearance of the intervening exaggerated haustral markings (Fig. 1*F*). The patient was placed on metronidazole therapy, 3 times 500 mg. a day for 7 days. He improved clinically within 48 hours of therapy, and a follow-up barium enema examination showed complete disappearance of all the roent-

genologic signs of disease except for minimal edema of the haustra (Fig. 1*G*).

Case II. A 70 year old man was admitted with the complaint of diarrhea and fever of 5 days' duration. On physical examination, he was pale and sick-looking. Proctoscopy revealed a fungating mass in the rectum covered with pus and debris. A barium enema examination showed a 10 cm. area of narrowing in the rectum (Fig. 2*A*). There were multiple ragged ulcerations of varying sizes. One of the deep ulcers perpendicular to the wall penetrated about 2 cm. deep and appeared to lead to a perirectal mass on the left. The upper edge of the lesion showed sharp transition to normal colonic wall, but there was no shelving. The rectal scrapings and biopsy showed *Entamoeba histolytica* trophozoites. The patient was placed on metronidazole—2 tablets (500 mg.) 3 times daily for 7 days. His symptoms improved remarkably and a follow-up barium enema examination at the end of 7 days of therapy showed almost complete disappearance of the ulcers and the perirectal mass. The single penetrating deep ulcer had reduced in size and had clean smooth borders (Fig. 2*B*).

Comment. The 2 cases reported herein illustrate the value of therapeutic response as a means of roentgen diagnosis of amebiasis. Both of the cases at the initial examination presented a roentgen appearance which was indistinguishable from other ulcerative diseases of the colon, but the response to metronidazole in 7–10 days established the diagnosis of amebiasis.

On conventional therapy, the great majority of lesions of intestinal amebiasis disappear completely within a month.[7,32] With the introduction of the new antiamebic drug, metronidazole, the treatment of amebiasis is greatly simplified and consists of about 1 week of therapy.[22] The response to therapy is rapid; signs and symptoms of amebic dysentery disappear within 48 hours, the amebic granulomas are greatly

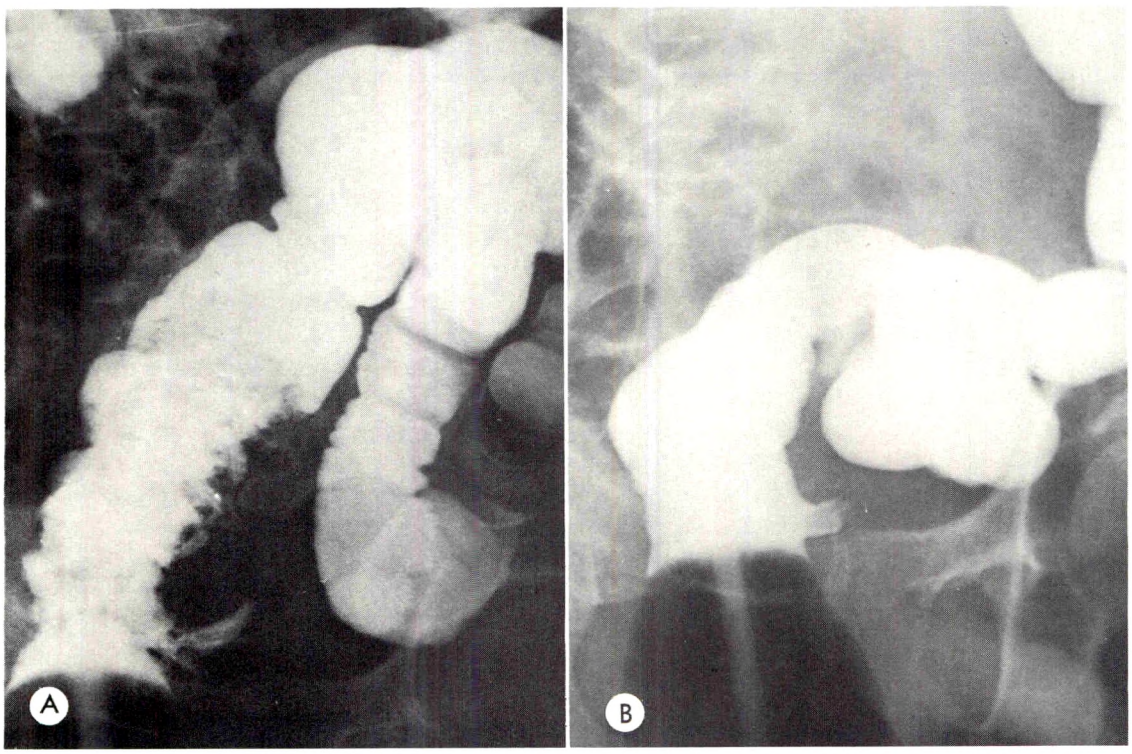


Fig. 2. Case II. Roentgenograms show: (*A*) a long segment of the rectum to be concentrically narrowed with ragged superficial and deep ulcers. A penetrating ulcer is noted leading to a pericolic abscess. (*B*) Seven days after the beginning of the metronidazole therapy, the penetrating ulcer and the pericolic abscess have reduced in size and the ulcer tract now has a smooth border. The remainder of the ulcers have disappeared completely.

reduced in size in 1 week and are no longer detectable on sigmoidoscopy in 14 days.[22,27]

A large ameboma may disappear within a few weeks, but, if the process has burrowed deep enough and lasted long enough, the fibrosis cannot be dissolved, although the ragged mass may shrink to become smooth and narrow.[24] Surgery may then be indicated for cure.[7] Surgery, in general, is dangerous in the untreated patients. However, some amebomas have been resected, in the untreated patients, without complications.[1] Failure to response to antiamebic therapy should redirect the radiologist's attention to the possibility of other ulcerative diseases of the colon, keeping in mind the possibility of excessive fibrosis due to amebiasis.

ROENTGEN FEATURES OF AMEBIASIS

The roentgen features of amebiasis are analyzed on the basis of 16 of our cases who had positive findings on the barium enema examination and 3 cases of acute abdomen who had only plain roentgenographic studies, but who at operation and histologic studies showed extensive intestinal amebiasis.

ULCERS IN AMEBIASIS

1. *Fine granularity or minimal irregularity of the bowel margins.* The early ulcers of amebic colitis are small and shallow, have raised margins and are covered with excessive mucus and pus. Roentgenograms at this stage show an irregular bowel margin and fine granularity of the mucosa, which are better demonstrated on air contrast studies. These findings were seen in 3 of our cases (Fig. 3*A*) and had to be differentiated from the crowded shallow ulcers of ulcerative colitis,[2,10,29] from finely distributed fecal material on air contrast study,[18] from fine granularity with target appearance in trichuriasis,[24] and from fine pseudopolypoid hyperplasia.[2]

2. *Fine serrations and coarse serrations.* Serrations are encountered in almost any infectious disease that involves the colon acutely or chronically.[18,29] Fine serrations were seen in 3 cases with multiple small spike-like projections which were irregular in size and distribution, had fuzzy borders and disappeared on antiamebic therapy (Fig. 3, *B* and *C*). This was in contrast to the sharp and regular appearing innominate grooves of the clean normal bowel.[20,33] Coarse serrations resembling "torn-blotting" paper[29] were seen in 5 cases (Fig. 3*D*).

3. *Collar-button ulcers.* These ulcers are slightly deeper than the above mentioned superficial ulcers and are known to be the result of submucosal proteolysis by amebae.[18,24] They may be difficult to demonstrate on barium enema examinations.[24] We could demonstrate this type of ulcers in 3 of our cases (Fig. 3*E*). They occur commonly in ulcerative colitis and probably as commonly in granulomatous colitis; and may be seen in tuberculosis, shigellosis, salmonellosis and staphylococcal infections.[10,18,20]

4. *Deep ulcers.* These are 2 mm. or more in size measured on the roentgenograms and are a characteristic feature of granulomatous colitis; they are much more common than in ulcerative colitis but can also occur in other ulcerative diseases of the colon.[18,29] The deep ulcers of amebic colitis are generally believed to be produced by ulcerative necrosis of the amebae and secondary bacterial infection.[32] Deep ulcers, in our series, ranged between 3–13 mm. in depth and were of the following types: *deep spike-like* ulcers in 4 cases (Fig. 3*D*); *deep saccular* in 2 cases (Fig. 3*F*); and *deep penetrating* in 2 cases (Fig. 2*A*). Seven of these cases had ulcers of varying sizes. This is known to occur in 20 per cent of cases in granulomatous and ulcerative colitis.[20]

5. *Transverse and longitudinal ulcers with cobblestoning.* This appearance was seen in 2 cases (Fig. 4*B*). It is approximately twice as common in granulomatous colitis as in ulcerative colitis and can occur in any other severe long lasting inflammatory disease.[15]

Coalescence of deep ulcers paralleling the lumen. Histologic studies show that superficial and deep abscesses of amebae often

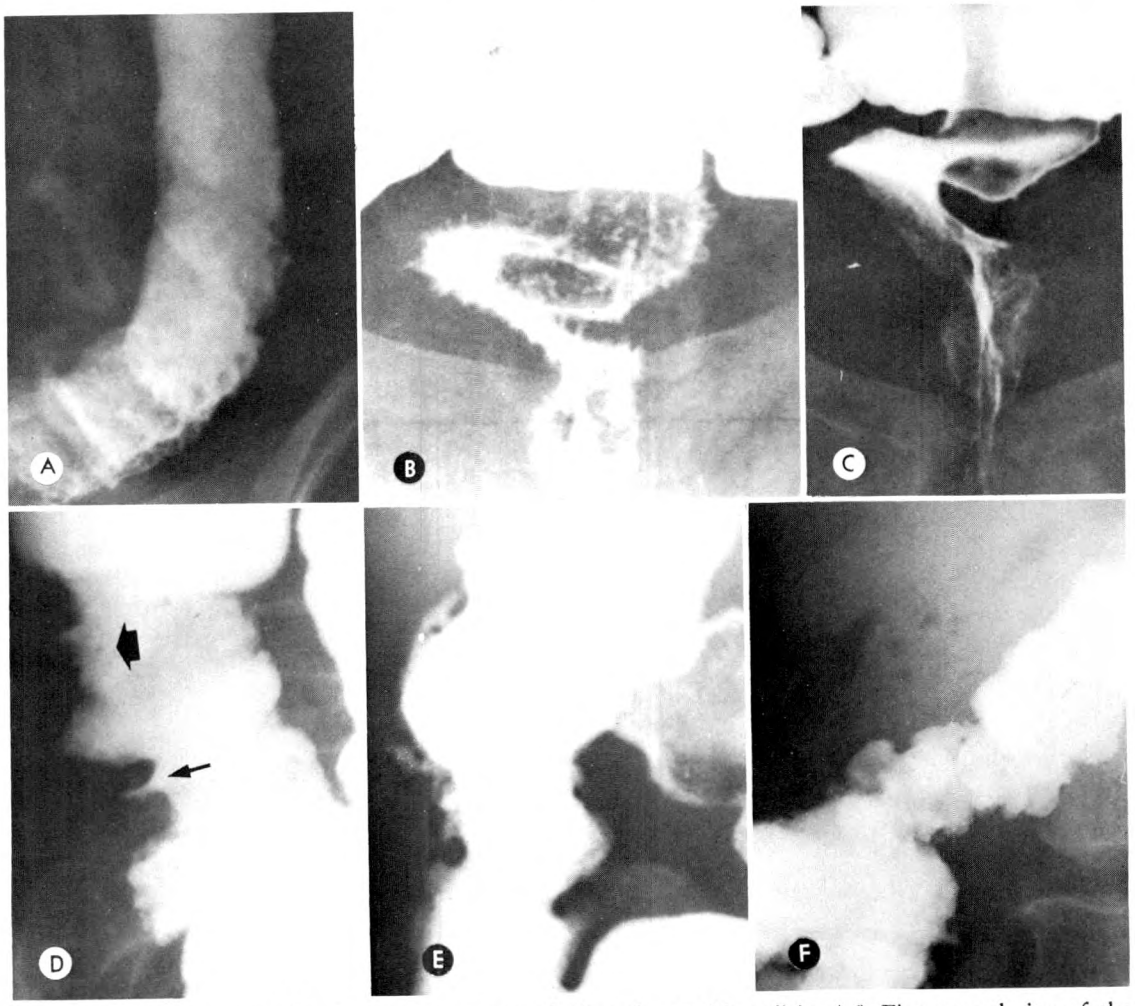


FIG. 3. Roentgenograms showing various types of ulcers in amebic colitis. (*A*) Fine granularity of the mucosa representing crowded shallow ulcers. (*B*) Superficial fine serrations. (*C*) Same after antiamebic therapy. (*D*) Superficial coarse serrations (thick arrow) and a deep spike-like ulcer (narrow arrow). (*E*) Collar-button ulcers with coalescence of a few paralleling the lumen. (*F*) Shallow and deep saccular ulcers.

communicate with each other[28] and sometimes result in large mucosal sloughs which appear as filling defects in the intestinal lumen.[23] We have seen coalescence of 2 or 3 collar-button ulcers (Fig. 3*E*), but they were not as extensive as are seen in granulomatous colitis.[20]

FILLING DEFECTS AND OTHER MUCOSAL CHANGES

Coarse linear, coarse crinkled and disorganized mucosal patterns. These patterns were seen in 10 cases (Fig. 4*A*; and 5*C*). They are nonspecific findings and indicate edema.[10,18,29,30]

Cobblestoning. Cobblestoning was noted with intersecting ulcers in 2 cases (Fig. 4*B*).

Pseudopolyps. Pseudopolyps in the form of discrete radiolucencies, circular in shape, were noted in 1 case only (Fig. 4*A*). They have been described in occasional cases of amebiasis,[13,24,31] but are commonly seen in ulcerative colitis, granulomatous colitis and schistosomiasis.[5,18,24]

Thumbprinting. Thumbprinting in the form of large clear-cut indentations was noted in 5 cases (Fig. 4*C*). It has been occasionally noted in amebiasis.[31] It is commonly seen in ischemic colitis[19,26] and can

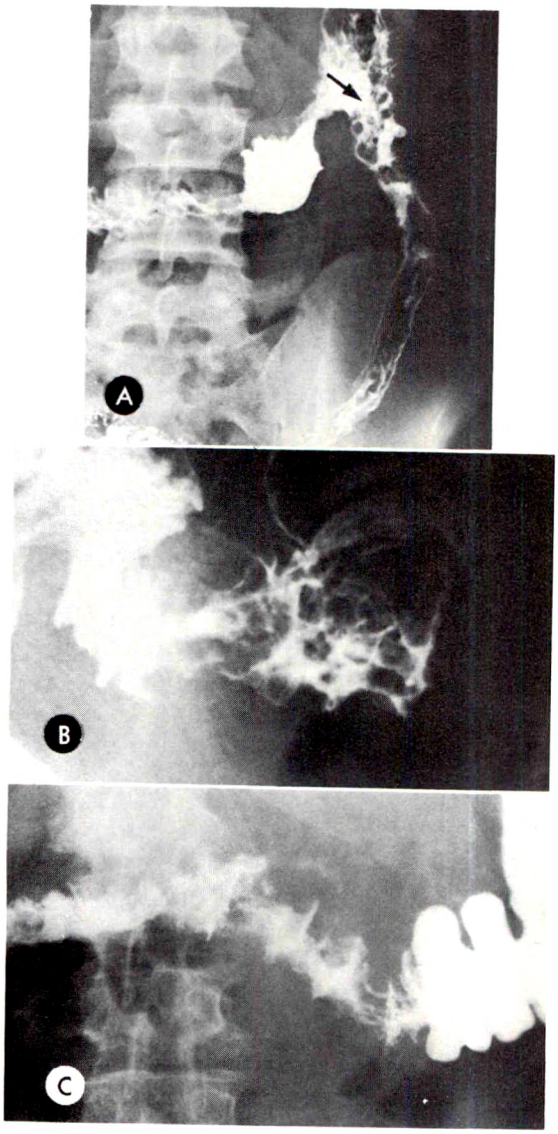


FIG. 4. Roentgenograms showing mucosal changes. (*A*) Coarse linear and disorganized mucosal folds and a few pseudopolyps (arrow). (*B*) Cobblestoning of the mucosa with crisscrossing of ulcers. (*C*) Thumbprinting in the midtransverse colon.

also occasionally be seen in granulomatous colitis and ulcerative colitis.[29] It indicates inflammation irrespective of cause.[18]

HAUSTRAL CHANGES

Spasm and irritability. This is a nonspecific feature of amebiasis. It is said to be less marked in granulomatous colitis than in ulcerative colitis.[20] Mild to severe transient spasm and irritability about the site of the lesion were best noted at fluoroscopy and were recorded on the roentgenograms of 10 out of 16 positive barium enema examinations (Fig. 1, *A* and *B*).

Exaggeration or irregularity of haustral pattern. These were commonly observed by Delahaye *et al.*[7] in the form of umbilicated, bifid, serrated and stacked coin haustra, in a study of 169 cases of amebiasis. Similar appearances were also noted in early granulomatous colitis[25] and are considered to be a sign of spasm and inflammatory thickening.[32] These appearances were noted in 8 cases (Fig. 1, *E* and *F*).

Reduction or loss of haustral patterm. This is a common and prominent finding in all ulcerative phases of amebiasis, but is a nonspecific reaction to any inflammatory process.[24] It was present in 11 cases (Fig. 1*B*; and 7, *A–D*).

Distensibility and narrowing of the lumen. Lack of distensibility to normal caliber and narrowing of the lumen are said to be the most common findings in amebiasis.[32] Some degree of distensibility is known to be present even with the tumor-like narrowing seen with amebomas,[24] and this may help its differentiation from carcinoma[9] (Fig. 1*F*; and 8, *A–C*).

Thickening of the colonic wall. Thickening of the colonic wall is a prominent finding in amebiasis.[9,23,32] However, the acute fulminant or necrotizing form of amebiasis[9,23,32] causes a paper thin necrotic bowel wall.[24] Bowel wall thickening of varying degrees was noted in 13 cases (Fig. 7, *A–D*; and 8, *A–C*).

OTHER ROENTGEN CONSIDERATIONS

Concentric narrowing of the cecum—the "coned cecum." The cecum is said to be involved in about 90 per cent of cases of chronic amebiasis.[32] The concentric narrowing of the normal sac-like cecum results in the coned cecum. The coned cecum of amebic colitis has a shaggy contour and the terminal ileum is very often intact.[24,32] We could demonstrate coned cecum in 7 cases, 6 of which had intact terminal ileum but

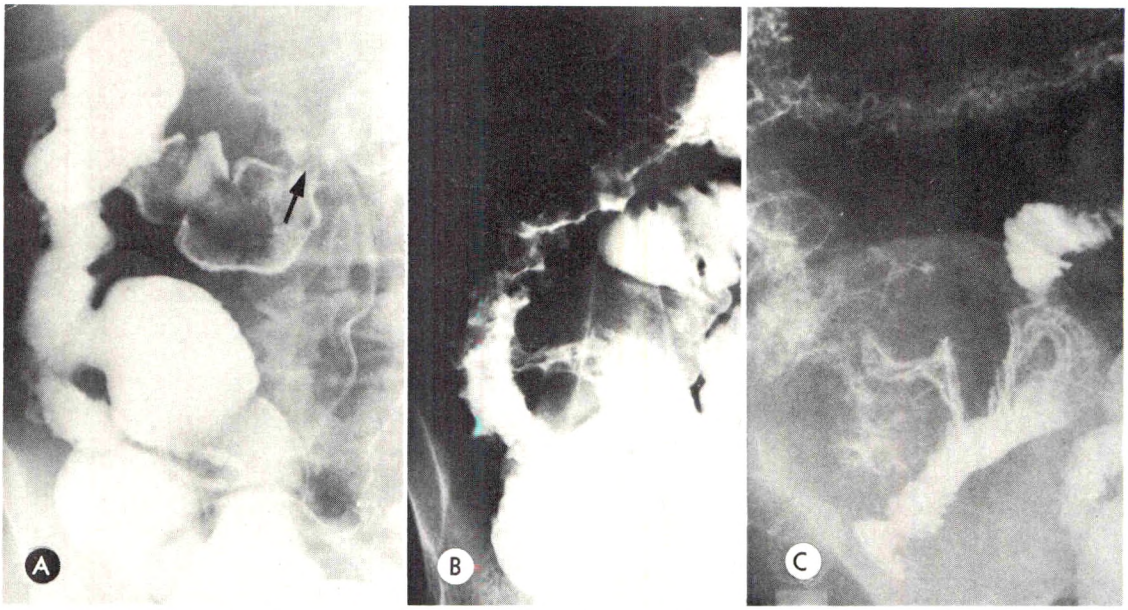


Fig. 5. Roentgenograms show: (*A*) the classical "coned-cecum" of amebic colitis. Note the gaping ileocecal valve and the distended ileum. Also note a pseudodiverticulum in the midtransverse colon (arrow). (*B*) Slight prominence of the mucosal folds of same on postevacuation film. (*C*) Severe involvement of the cecum and ascending colon with coarse crinkled mucosa but intact ileum.

had skip lesions elsewhere in the colon. This combination of findings of coned cecum, intact ileum and skip lesions was the most helpful diagnostic feature of amebiasis (Fig. 5, *A–C*; and 6). We have seen only 2 cases of eccentric lesion of the cecum, which appeared plaque-like. The differential diagnosis included ulcerative colitis, granulomatous colitis, tuberculosis, carcinoma of the cecum, appendicitis and diverticulitis.[7,24,32] The first three affect the ileocecal valve and the terminal ileum to varying degrees as is described below. The cecal carcinoma is often asymmetric, irregular and polypoid; appendicitis usually shows an eccentric defect at the base of the cecum; and diverticulitis is essentially a pericecal abscess.

The ileocecal valve. The cecum and the ileocecal valve are involved in 60–90 per cent of cases of amebiasis.[24,32] The roentgen changes of ileocecal valve are not described but the patency of the ileocecal valve with easy filling of the terminal ileum is stressed as an important sign in amebiasis.[7] Gaping ileocecal valve with well distended terminal ileum was present in all the 7 cases of "coned" cecum (Fig. 5*A*). The ileocecal valve was thickened but otherwise smooth in 2 cases. The ileocecal valve is also known to be commonly gaping in ulcerative colitis; it is thickened and crisscrossed by ileocecal fistulas in granulomatous colitis,[20] and "in-

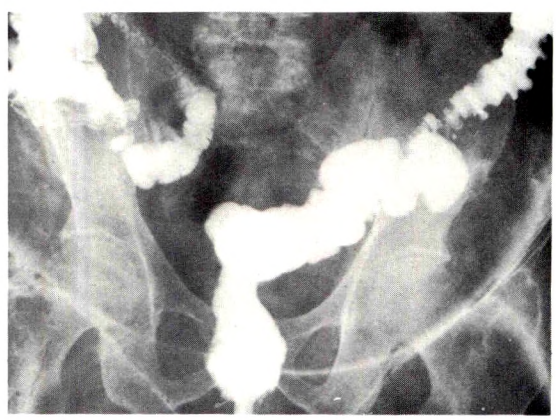


Fig. 6. Roentgenogram of a case of amebic colitis and amebic diverticulitis. There are constricting lesions of the rectum and the cecum (coned cecum). Few amputated diverticula are noted in the sigmoid colon. Resection of rectosigmoid showed amebae in the submucosa and in and around the wall of the diverticula with severe serosal reaction.

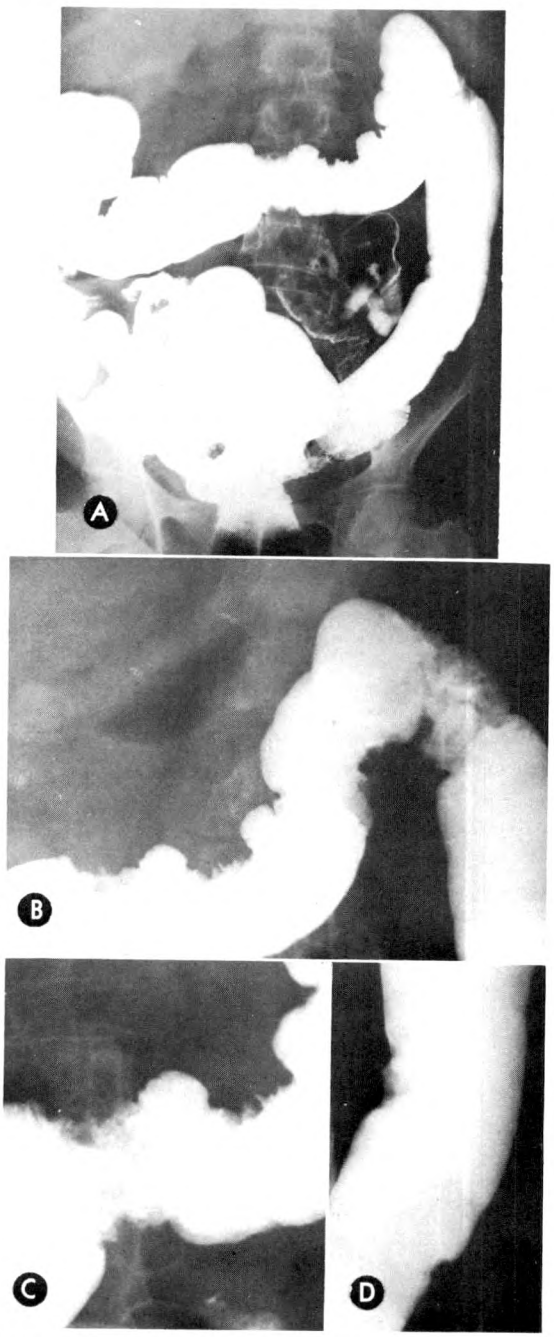


FIG. 7. Roentgenograms of a case of multiple skip lesions. (*A*) Multiple skip lesions (about 10) of amebic colitis. Note the coned cecum with distended ileum and ulcerations of the rectum. (*B*) The distal transverse colon shows the 2 concentric and 2 eccentric lesions. (*C*) Close-up of same, showing details of a short concentric lesion and an eccentric lesion with coarse serrations. (*D*) Close-up view of the very small plaque-like lesion of the descending colon.

verted umbrella-like" with spasm of the ileocecal valve in tuberculosis.[3,11]

The terminal ileum and the appendix. Kean *et al.*[15] reported 7 cases of involvement of the terminal ileum in 148 fulminant fatal cases of amebiasis, but Delahaye *et al.*[7] failed to find a single case of ileal involvement in the roentgen study of 171 cases of chronic intestinal amebiasis. Other reports[13,25] mention occasional cases showing some roentgen changes of the terminal ileum. One of our fatal cases had invasion of the terminal ileum and the appendix at necropsy, but we have no histologic proof of terminal ileal involvement in the 2 cases of barium study which showed some prominence of the mucosal folds (Fig. 5*B*). The terminal ileum is involved much more commonly in tuberculosis (85 per cent)[32] and granulomatous colitis (80 per cent).[20] The terminal ileum is severely affected in tuberculosis, with coarse mucosal folds, rigid walls and very irregular contour, more severe than seen in granulomatous colitis.[14,20] The backwash ileitis of ulcerative colitis is a short segment involvement with minimal narrowing.[20]

Concentric narrowing of areas other than the cecum. Concentric lesions with mild thickening of wall, measuring 3–30 cm. in length, were seen in 6 cases (10 lesions) (Fig. 7, *A–D*), and thick-walled apple core and shelf-like lesions, measuring 7–12 cm. in length, were seen in 4 cases (5 lesions) (Fig. 8, *A–C*). These apple core lesions represented amebomas which closely simulate carcinoma of the colon.[1,9]

Of the several criteria mentioned[9] to differentiate the chronic amebic lesions from carcinoma, we found that tapering of the lesion, at least at one end, and fairly well preserved mucosa in the area were the 2 most important differential diagnostic signs, short of response to antiamebic therapy (Fig. 8, *A–C*). Fleishner and Ming[12] described concentric narrowing with funneling at both ends in occasional cases of diverticulitis of the sigmoid colon. This should easily mimic amebiasis. The situation was made worse in one of our cases in

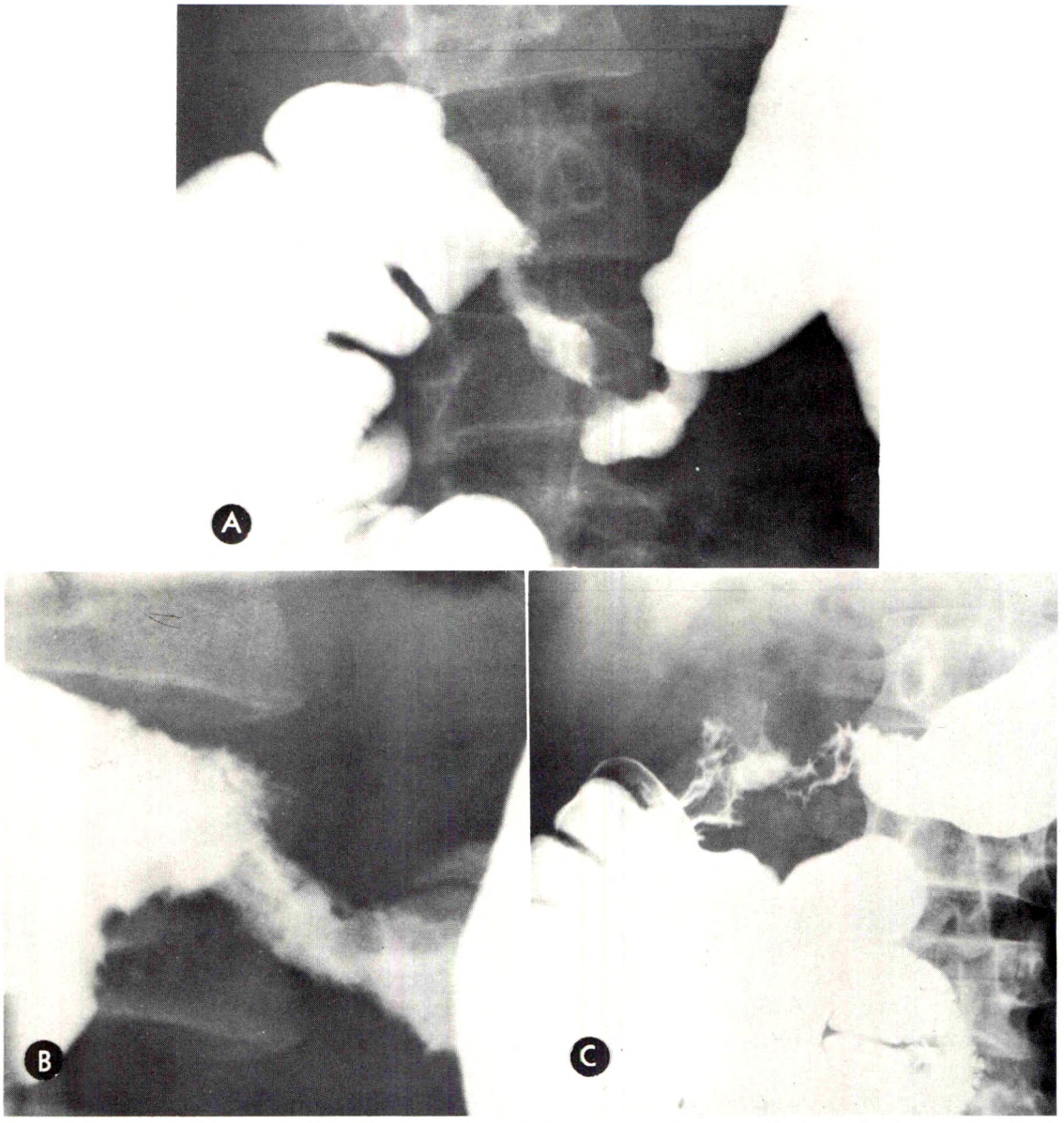


FIG. 8. Roentgenograms of a case of ameboma show: (*A*) a long annular constricting lesion (3 cm. thick wall at operation) with a central large ulcer; (*B*) tapering and ragged end; and (*C*) fairly well preserved mucosa surrounding the large ulcer as seen on the postevacuation roentgenogram.

which amebiasis was superimposed on diverticulosis. Histologic sections showed amebae invading colonic submucosa and the serosa of the tip of diverticula showed severe serosal reaction. The roentgen appearance was a combination of amebic colitis and amebic diverticulitis (Fig. 6).

Eccentric lesions of the colon. Plaque-like lesions with shaggy outlines were noted in 6 cases (Fig. 7, *A–D*). Some were as small as 2 cm. (Fig. 7*D*). These lesions had to be differentiated from the "typical" eccentric lesions of granulomatous colitis which have obvious mucosal changes,[16,20] from metastatic implants which usually have smooth colonic contour and intact mucosa,[16] from the so-called "solitary ulcer" of the colon which may present as a plaque-like mass

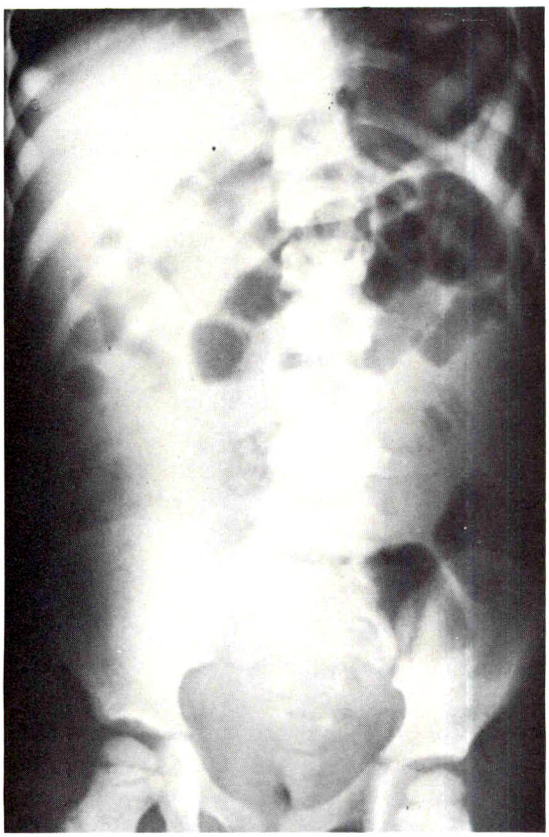


FIG. 9. Roentgenogram of a fatal case of amebiasis in a child showing evidence of peritonitis and ileus. The patient had acute amebiasis with perforation of the cecum and peritonitis. (The terminal ileum and the appendix were invaded by amebae.)

with or without an ulcer,[14] and from lymphoma of the colon which often has a nodular appearance.

Length and site of the lesions. The lesions varied between 2–30 cm. in length. Right-sided colitis occurred in 7, left-sided colitis in 6 and total colitis occurred in the form of multiple skip lesions in 5 (Fig. 7, *A* and *B*). Total diffuse colitis as seen in ulcerative colitis was not encountered in our barium enema studies. The cecum and ascending colon were involved in 9, the rectosigmoid in 8, the transverse colon in 7, and the descending colon in 4.

Skip lesions. Multiple sites of involvement occurred in 7 cases. The most severe had about 10 skip lesions (Fig. 7*A*). Skip lesions are known to be present in about 50 per cent of cases of amebiasis, but are also a characteristic feature of granulomatous colitis.[18,24,32]

COMPLICATIONS OF AMEBIASIS

Toxic megacolon. The extreme stage occurs in 1–3 per cent of those with overt amebic colitis and frequently terminates fatally with multiple perforations, peritonitis and septicemia.[13,24] Barium enema study is contraindicated.[24] Toxic megacolon is a typical complication of ulcerative colitis, but can also occur in granulomatous colitis and ischemic colitis.[18]

Perforation and ileus. We encountered 3 cases of perforation with peritonitis and ileus who died in the postoperative period. One of the cases was a 3 year old child who had extensive invasion of the colon, appendix and the ileum by amebae (Fig. 9). Kean *et al.*[15] reported 148 fatal cases of amebiasis, 45 of which (commonest cause of death) died because of perforation and peritonitis.

Sinuses, fistulae and pericolic abscesses. Perforations in amebiasis may lead to fatal peritonitis or to formation of sinuses, fistulae and pericolic abscesses.[24] We encountered 1 case with a small pericolic abscess which responded to therapy (Fig. 2, *A* and *B*). The differential diagnosis includes granulomatous colitis, diverticulitis, actinomycosis, tuberculosis and salmonellosis.[18]

Pseudodiverticula and strictures. In 1 of our cases a pseudodiverticulum was noted in an area of constriction of the midtransverse colon (Fig. 5*A*). Pseudodiverticula are known to occur in granulomatous colitis, scleroderma and ischemic colitis.[19,20]

Amebic liver abscess. We had 2 cases of amebic liver abscess in our series of 19 cases of positive intestinal amebiasis. They manifested with an elevation of the right leaf of the diaphragm, small to large pleural effusion and were shown on liver scans. Amebic liver abscess occurred in about 12 per cent of the 119 cases of amebic granulomas reported by Radke.[23]

Intussusception and amebic colitis. Intus-

susception is not uncommon in amebic colitis.[23] The only case we had was a 4 year old child who had severe amebiasis. The intussusception was reduced by a cautious barium enema examination.

Amebic colitis with amebic diverticulitis. Amebic colitis with amebic diverticulitis occurred in 1 of our cases as is described above (Fig. 6).

Concomitant amebic colitis and ulcerative colitis. One of our cases with a total diffuse colitis had *Entamoeba histolytica* in the stools and in rectal biopsy. He responded only slightly to antiamebic treatment, but did very well on corticosteroids. The patient subsequently was managed as a case of ulcerative colitis.

Carcinoma and amebiasis. Carcinoma is sometimes seen to coexist with amebiasis. Its significance is that the lesion suspected of amebiasis should be completely subsided before it can be considered to be due to amebiasis alone.[8,28]

SUMMARY

Various roentgen features of 19 cases of amebic colitis are analyzed and compared with granulomatous colitis, ulcerative colitis and other ulcerative diseases of the colon.

The lesions of amebic colitis, in general, had a shaggy contour; the eccentric lesions were almost as common as the concentric lesions. The left-sided colitis was as common as the right-sided colitis and diffuse colitis was present only in the form of multiple skip lesions. Skip lesions were common. Deep and superficial ulcers of varying types were noted and had, in general, a ragged appearance. Various types of unspecific haustral and mucosal changes were noted. The annular lesions of amebomas were distinguished from carcinomas in most cases by their gradually tapering ends, relatively well preserved mucosa and response to antiamebic therapy.

The diagnosis of amebic colitis was strongly considered when the cone-like cecum was accompanied by an intact terminal ileum and a skip lesion elsewhere in the colon. The shaggy contour of the lesions, the ragged ulcers, the multiplicity of the lesions, and a fairly well preserved mucosa on the postevacuation roentgenograms rendered further support to the diagnosis of amebic colitis.

Two case reports are included to demonstrate the unique value of the therapeutic test (metronidazole), in the final and conclusive roentgen diagnosis of intestinal amebiasis.

Included in the series also are cases of concomitant amebic colitis and ulcerative colitis, amebic colitis with amebic liver abscess, amebic colitis with intussusception, and amebic colitis with amebic diverticulitis.

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REFERENCES

1. Balikian, J. P., and Garabedian, M. M. Ameboma of transverse colon simulating carcinoma: report of two cases with their roentgenologic manifestations. *Lebanese M. J.*, 1965, *18*, 259–263.
2. Brahme, F. Granulomatous colitis: roentgenologic appearance and course of lesions. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 35–44.
3. Brown, L., and Sampson, H. L. Intestinal Tuberculosis. Second edition. Lea & Febiger, Philadelphia, 1930.
4. Brown, C. H., and Winkelman, E. I. Amebiasis and ulcerative colitis. *Cleveland Clin. Quart.*, 1965, *32*, 61–67.
5. Chait, A. Schistosomiasis mansoni: roentgenologic observations in nonendemic area. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 688–708.
6. Chketri, M. K., and Chakravarty, N. C. Metronidazole in treatment of intestinal amebiasis. *J. Indian M. A.*, 1969, *50*, 312–313.
7. Delahaye, R. P., Jolly, R., Laaban, J., and Boursiquot, P. L'amibiase intestinale chronique. *J. de radiol., d'électrol. et de méd. nucléaire*, 1964, *45*, 117–132.
8. Doshi, J. C. Amebic granuloma: review. *Indian J. M. Sc.*, 1969, *23*, 61–67.
9. Druckmann, A., and Schorr, S. Roentgenologic manifestations of amebiasis of large intestine. Am. J. Roentgenol. & Rad. Therapy, 1945, *54*, 145–148.

10. FENNESSY, J., SPARBERG, M., and KIRSNER, J. Early roentgen manifestations of mild ulcerative colitis and proctocolitis. *Radiology*, 1966, *87*, 848–858.
11. FLEISHNER, F. G., and BERNSTEIN, C. Roentgen anatomical studies of normal ileocecal valve. *Radiology*, 1950, *54*, 43–58.
12. FLEISHNER, F. G., and MING, S. C. Revised concept on diverticular disease of colon. II. So-called diverticulitis: diverticular sigmoiditis and perisigmoiditis, diverticular abscess, fistula and frank peritonitis. *Radiology*, 1965, *84*, 599–609.
13. HILL, M. C., and GOLDBERG, H. I. Roentgen diagnosis of intestinal amebiasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 77–83.
14. HODGSON, J. R. Miscellaneous diseases of colon. *Seminars in Roentgenol.*, 1968, *3*, 94–105.
15. KEAN, B. H., GILMORE, H. R., JR., and VAN STONE, W. W. Fatal amebiasis: report of 148 fatal cases from Armed Forces Institute of Pathology. *Ann. Int. Med.*, 1956, *44*, 831–843.
16. KHILNANI, M. D., MARSHAK, R. H., ELIASOPH, J., and WOLF, B. S. Roentgen features of metastases to colon. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 302–310.
17. LEVINE, S. M., STOVER, J. F., WARREN, J. G., CHAPPELKA, A. R., and BURKE, E. L. Ameboma, forgotten granuloma. *J.A.M.A.*, 1971, *215*, 1461–1464.
18. MARGULIS, A. R. Radiology of ulcerative colitis. *Radiology*, 1972, *105*, 251–262.
19. MARSHAK, R. H., and LINDNER, A. E. Ischemia of colon. *Seminars in Roentgenol.*, 1968, *3*, 81–93.
20. MARSHAK, R. H., and LINDNER, A. E. Radiology of the Small Intestine. W. B. Saunders Company, Philadelphia, 1970.
21. OCHSNER, A. Surgical significance of amebiasis. *Dis. Colon & Rectum*, 1960, *3*, 297–304.
22. POWELL, S. J. Therapy of amebiasis. *Bull. New York Acad. Med.*, 1971, *47*, 469–477.
23. RADKE, R. A. Ameboma of intestine: analysis of disease as presented in 78 collected and 41 previously unreported cases. *Ann. Int. Med.*, 1955, *43*, 1048–1066.
24. REEDER, M. M., and HAMILTON, L. C. Tropical diseases of colon. *Seminars in Roentgenol.*, 1968, *3*, 62–80.
25. RUDHE, U., and KEATS, T. E. Granulomatous colitis in children. *Radiology*, 1965, *84*, 24–32.
26. SCHWARTZ, S., BOLEY, S. J., ROBINSON, K., KRIEGER, H., SCHULTZ, L., and ALLEN, A. C. Roentgenologic features of vascular disorders of intestines. *Rad. Clin. North America*, 1964, *2*, 71–78.
27. SCOTT, F., and MILLER, M. J. Trials with metronidazole in amebic dysentery. *J.A.M.A.*, 1970, *211*, 118–120.
28. SPICKNALL, C. G., and PIERCE, E. C. II. Amebic granuloma: report of four cases. *New England J. Med.*, 1954, *250*, 1055–1062.
29. STANLEY, P., FRY, K., DAWSON, A. M., and DYER, N. Radiological signs of ulcerative colitis and Crohn's disease of colon. *Clin. Radiol.*, 1971, *22*, 434–442.
30. STEIN, G. N., ROY, R. H., and FINKELSTEIN, A. R. Roentgen changes in ulcerative colitis. *Seminars in Roentgenol.*, 1968, *3*, 3–26.
31. TCHANG, S. Amebiasis in northern Saskatchewan: radiological aspects. *Canad. M. A. J.*, 1968, *99*, 688–695.
32. WEINFELD, A. Roentgen appearance of intestinal amebiasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 311–322.
33. WILLIAMS, I. Innominate grooves in surface of mucosa. *Radiology*, 1965, *84*, 877–880.

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RADIOLOGIC OBSERVATIONS ON INTESTINAL AMEBIASIS

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IBADAN, NIGERIA

AMEBIC colitis is a common medical problem at the University College Hospital (U.C.H.), Ibadan. A report of the analysis of the hospital series drew attention to the varying clinical forms and the complications commonly associated with the disease.[6] In it, it became apparent, although not often appreciated, that roentgenographic studies along with clinical examination are most valuable in the assessment and management of the cases.

This paper attempts to emphasize the value of radiology in the diagnosis, assessment and management of 8 cases of amebic colitis in which radiologic studies were carried out. It also includes radiographic guidelines in differential diagnosis, particularly with reference to idiopathic nonspecific ulcerative colitis which it mimics very closely.

MATERIAL AND METHOD

Patients diagnosed as having amebic colitis with or without associated complications over a period of 6 years (1960–1966) at the U.C.H., Ibadan, and who were subjected to radiographic examinations were selected to illustrate the types of intestinal lesions encountered. The diagnosis of amebic colitis was based on the demonstration of the trophozoites of *Entamoeba histolytica* in the stool specimens. Findings at laparotomy and/or autopsy together with relevant histology reports were included.

REPORT OF CASES

CASE 1. B.S. was a 40 year old Yoruba male patient, who was admitted to the hospital in 1960 with history of dysentery for months. He passed 2 to 3 stools per day, but 1 week prior to admission the diarrhea became severe. On examination he was very thin and dehydrated. There was marked tenderness in the abdomen, particularly in the right iliac fossa, and a mass was felt in the left side of abdomen. The microscopic examination of the stool revealed trophozoites of *Entamoeba histolytica*. Sigmoidoscopy up to 20 cm. showed few shallow ulcers, and the mucosa was granular and bled easily. Barium enema study demonstrated lack of haustra and distensibility of the rectum, the colon and the cecum. There was total effacement of the mucosa. There was short annular filling defect in the midportion of the descending colon. A few deep ulcers were seen in the rectosigmoid region (Fig. 1).

Treatment with fluid replacement and eme-

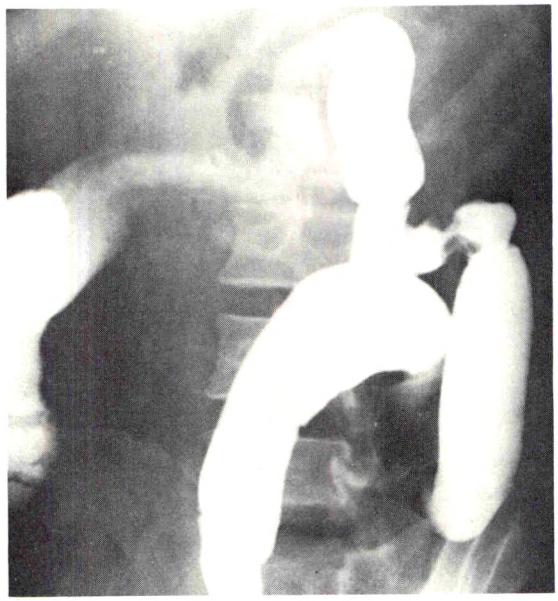


FIG. 1. Case 1. The rectum, colon and cecum show a lack of haustra and distensibility. A few ulcers are seen in the rectosigmoid region. There is a short annular filling defect with overhanging edges in the midportion of the descending colon shown to be due to ameboma (autopsy).

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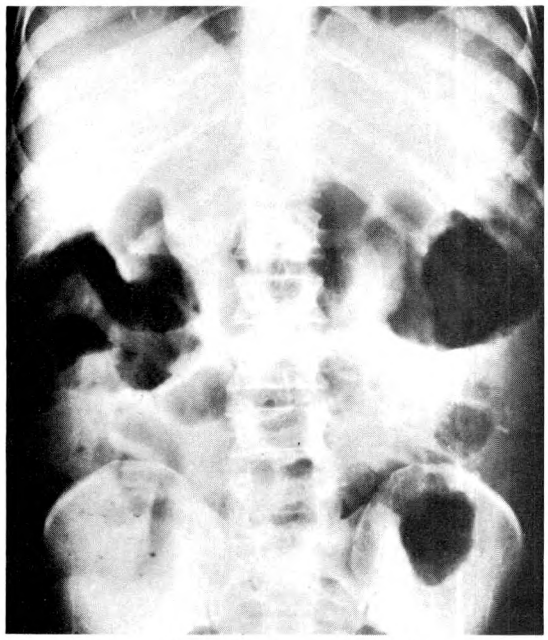


FIG. 2. Case III. Gaseous distention of the transverse colon with thumbprinting of its edges.

tine bismuth iodide at that time was unsuccessful.

At postmortem examination there were extensive confluent ulcerations with punched-out ulcers in the large bowel, and an ameboma in the midportion of the descending colon.

CASE II. J.O. was a 42 year old mechanic who was admitted on account of intermittent bloody diarrhea for 5 months. He had some right-sided lower abdominal pain and fever. On examination he was dehydrated and slightly pale. There was a tender large mass on the right side of the abdomen. Microscopy of the stool showed numerous trophozoites of *Entamoeba histolytica*. Barium enema examination revealed almost complete obliteration of the cecum by a filling defect which extended into the midportion of the ascending colon. The overlying mucosa was effaced.

Laparotomy was performed and a huge inflammatory mass was found extending from the middle of the ascending colon down to the cecum.

The resected specimen consisted of the cecum and a portion of the ascending colon. The cecum adjacent to the appendicular opening had a deep ulcer, 2.5 cm. across. In the distal dilated colon were small ulcers. The mucosa of the dilated part was abnormally smooth. Histology of the ulcer showed complete destruction of the mucosa and the division of the mucosa and submucosa could not be defined. There was intense plasma cell infiltration and a few degenerated *amoebae*. The muscle layer was thickened and also infiltrated with plasma cells, lymphocytes and eosinophils. The ulcers were typical amebic ulcers. The lymph nodes showed nonspecific hyperplasia.

CASE III. J.O. was a 54 year old native doctor who complained of diarrhea, abdominal discomfort and occasional vomiting for 20 days. On examination he was ill-looking, and had a palpable mass in the right iliac fossa. Microscopy of the stool specimen showed trophozoites of *Entamoeba histolytica*. Plain film roentgenograms of the abdomen showed dilated portions of the colon, especially the transverse colon which also showed thumbprinting (Fig. 2). A barium enema examination demonstrated irregularities and collar-stud ulcers along the margins of the right half of the colon and rectum (Fig. 3). The cecum and ascending colon were deformed with filling defects. There was evidence of ileocecal incompetence (Fig. 4).



FIG. 3. Case III. Multiple deep ulcers showing as spiky irregularities along the margins of the rectum and sigmoid colon.

Case iv. S.B. was admitted to the hospital with a 4 week history of dysentery and abdominal pain. Trophozoites of *Entamoeba histolytica* were found in the stool specimen. Treatment with emetine hydrochloride injections, followed with a course of tablets of emetine bismuth iodide, cleared the infection. The barium enema examination showed extensive destruction of the whole colon with abnormal mucosal and haustral patterns. A 3 cm. long stricture was present high up in the rectum and other strictures were noted at the proximal end of the sigmoid and in the descending colon (Fig. 5). The entire colon, especially the ascending colon and transverse colon, would not dilate, suggesting a generalized fibrosis.

Case v. L.A. was a 30 year old housewife, who was admitted with a history of abdominal pain, dysentery and fever for about a week. On examination she looked ill and slightly dehydrated. There was a palpable mass in the right iliac fossa. The plain film roentgenograms of the abdomen showed the presence of a mass in the right iliac fossa and signs of small bowel obstruction. At laparotomy the cecocolic intussusception which extended far into the splenic flexure was confirmed with an ameboma at the apex.

Case vi. J.O. was a 25 year old policeman who complained of abdominal pain and dysentery for about 3 weeks. On examination he was thin and wasted. There was tenderness in the right iliac fossa. Stool examination showed the presence of trophozoites of *Entamoeba histolytica*. Sigmoidoscopy revealed a granular and friable mucosa. Barium enema examination demonstrated marked loss of haustration of the descending colon. He was treated with Flagyl (menonindazole) but the diarrhea continued unabated. Having eradicated the *amoebae*, a short course of steroids was tried with some improvement. He was discharged feeling better and having gained weight, but still passed 2 to 3 soft stools per day. He returned 18 months later with his bowel habits having returned to normal. At this time the barium enema examination showed extensive lack of distensibility, effacement of the mucosa and loss of the haustra from the rectum to the colon. The ileocecal valve was patulous and incompetent (Fig. 6).

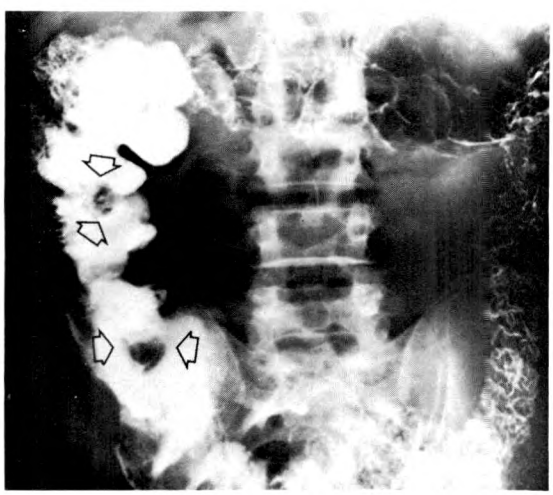


Fig. 4. Case iii. Round filling defects (amebomata) in cecum and ascending colon (arrows).

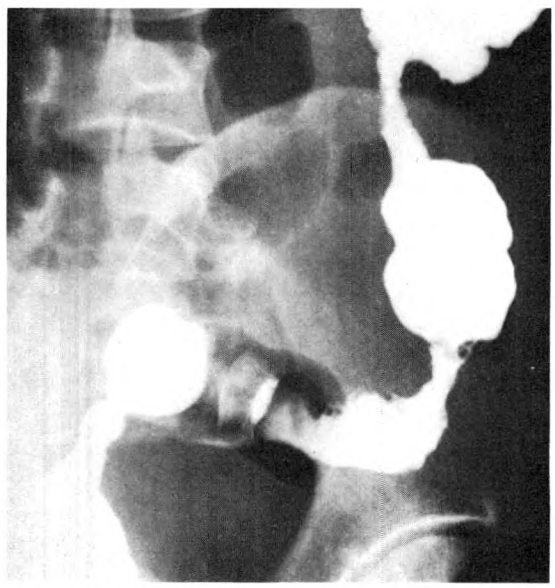


Fig. 5. Case iv. Generalized narrowing of the rectum, sigmoid and descending colon with long strictures between the rectum and sigmoid colon, the distal portion of the descending colon and sigmoid colon, and in the midportion of the descending colon.

Case vii. B.O., a 52 year old man, was admitted to the hospital with a 2 week history of abdominal pain and diarrhea. On examination, he was dehydrated and had generalized abdominal distention, with maximum tenderness in the right iliac fossa.

The specimens of the stool were watery and contained blood and mucus. Trophozoites of



FIG. 6. Case VI. Smooth nondistensible large bowel with lack of haustra the (the "pipe-stem" colon). The terminal ileum is normal, but there was ileocecal incompetence of screening.

Entamoeba histolytica were also found on microscopy.

Plain film roentgenograms of the abdomen showed distention of the ascending and transverse colon. Barium enema examination demonstrated no large bowel obstruction, but thumbprinting and irritability of the right half of the colon.

The postmortem examination revealed multiple undermined ulcers with purulent exudates involving the entire large bowel. There were trophozoites in the exudates and the muscle coats adjacent to the ulcers.

CASE VIII. B.A. was a 40 year old man, who was admitted with a 3 week history of right-sided abdominal pain, diarrhea, and progressive loss of weight. He passed about 12 loose stools per day, and the stools contained mucus and blood.

On examination he looked ill and grossly dehydrated. The blood pressure was 100/70 mm. Hg. The abdomen was rigid and tender generally and there was an impression of a mass in the right iliac fossa. Rectal examination was negative. The packed cell volume was 45 per cent, and the white blood cell count 10,400 per cu. mm. The stool microscopy revealed the presence of pus cells, and numerous trophozoites of *Entamoeba histolytica*.

Plain film roentgenograms of the abodmen showed an ill-defined mass in the right iliac fossa with neither air nor feces in the cecum and ascending colon.

Barium enema examination demonstrated marked irregularity and ulceration of the entire colon and cecum. The mucosal pattern was most bizarre with edematous changes and effacement, particularly in the cecum and right half of the colon (Fig. 7). A few intraluminal filling defects were also seen. There was no filling of the appendix or terminal ileum.

The patient was treated with Flagyl, 800 mg. thrice daily, for a week and the barium enema examination was repeated 2 weeks after the commencement of treatment. A third barium enema study was done 1 week after the second barium enema examination. The findings were identical showing lack of distensibility (fibrosis) and lack of haustration in the cecum, ascending and transverse colon. There were irregular strictures in the hepatic flexure and in the midportion of the descending colon. Pseudodiverticula were present in the transverse colon (Fig. 8). There were no ulcers seen. Ileocecal incompetence was also noted, but the terminal ileum and appendix appeared normal.

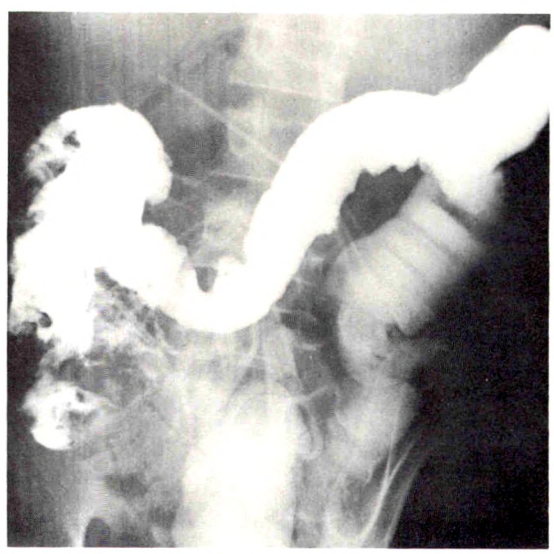


FIG. 7. Case VIII. Gross mucosal ulcerations with filling defects in the cecum, the ascending colon, and the hepatic flexure. Two outpouchings are seen in the midportion of the transverse colon.

RESULTS

The distribution of the sites of the lesions affecting the large bowel from our series is shown in Figure 9. The types of lesions and their numbers are listed in Table I. The cecum was involved in all cases; 5 of these cases had total colonic involvement. The appendix and the terminal ileum were not affected.

ULCERATION CHANGES
(5 cases)

Five of the cases (Cases I, II, III, VII, and VIII) showed either radiologic or microscopic evidence of ulcers. Ulceration is a prominent radiologic feature of amebiasis. The ulcers may be shown radiologically as shallow fine serrations, that is, a "saw-toothed" appearance. These may later become confluent (Cases I, and III) to form collar-stud abscesses under the mucosal lining of the gut. Some ulcers were deep (Fig. 2), but no definite evidence of intraperitoneal perforation was seen. The mucosa may be denuded and eroded (Fig. 8). A long segment of the colon and/or rectum was usually involved (Fig. 3). The portions of colon showing ulcerations are usually spastic and irritable during the filling phase of the barium enema examination.

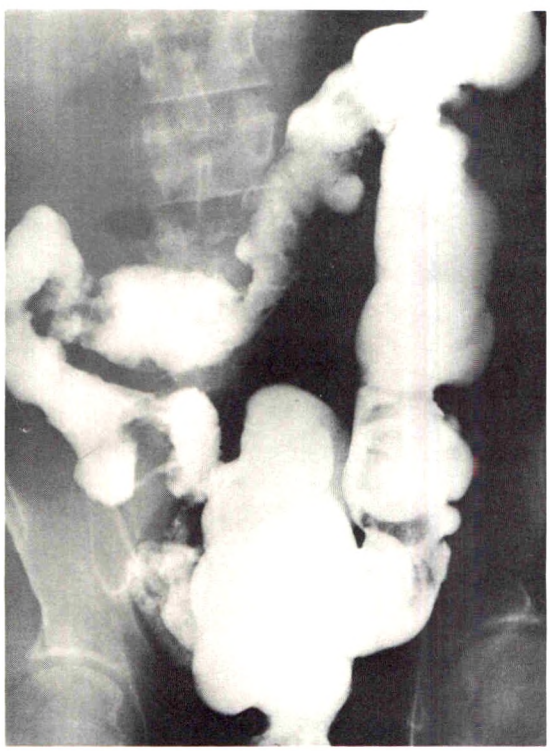


Fig. 8. Case VIII. Two weeks later, the entire cecum, the ascending and transverse colon have become nondistensible and fibrotic. Strictures are seen in the proximal part and midportion of the transverse colon as well as in the distal part of the ascending colon. The previously noted pseudodiverticula in the midtransverse colon have become more prominent. There was ileocecal incompetence. The terminal ileum and appendix appeared normal.

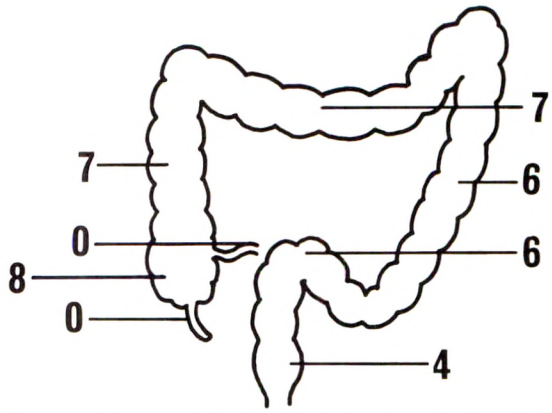


Fig. 9. Diagram illustrating the numerical distribution of lesions in the ileum and large bowel.

TOXIC DILATATION
(2 cases)

Toxic dilatation occurred in 2 cases (Cases III, and VII; Fig. 2). The transverse

Table I

TYPES OF LESIONS SEEN ON RADIOLOGIC EXAMINATION

	No. of Lesions	Case No. of Lesions
Ileocecal incompetence	5	II, III, IV, VI, VIII
Ameboma	4	I, II, III, V
Ulcers	5	I, II, III, VII, VIII
Toxic dilatation	2	III, VII
Intussusception	1	V
Multiple strictures	2	IV, VIII
Pipe-stem colon	1	VI
Pseudodiverticula	1	VIII

colon and hepatic flexure were involved in each case. The affected portions of the colon also showed pseudopolyposis and thumbprinting of the mucosa as well as exaggeration of the haustra.

FIBROTIC CHANGES

This feature is represented by total loss of mucosa and haustra, lack of distensibility and decreased length and caliber of the portion of large bowel affected.

(a) *"Pipe-stem" colon.* Case VI initially showed a "pipe-stem" appearance in the rectum, the descending and sigmoid colon initially; 18 months later, the lesion had involved the entire colon and cecum, suggesting a progression of the lesion from the left half of the colon toward the right. In this case the ileocecal valve was incompetent and the cecum contracted (Fig. 6).

(b) *Multiple strictures.* Two cases (Cases IV, and VIII) showed multiple strictures and "skip lesions." Case IV mainly involved the left half of the colon, while Case VIII involved the right half of the colon. The strictures were long, and tapering in character (Fig. 5). There was no poststenotic dilatation seen, but the intervening areas between the strictures were fibrotic and narrow.

MASS LESIONS
(4 cases)

These masses were due to the formation of amebomata. They usually affect the cecum (Cases I, II, III, and V), but may continue into the ascending colon (Case II). Ameboma could also occur elsewhere in the large bowel such as the descending colon (Case I). They may be plaque-like, or show nodular filling defects (Fig. 4) or may be seen as a short, annular mass with overhanging edges, mimicking carcinoma in appearance (Fig. 1). The characteristic lesion of ameboma with its cecal irregular deformity, the so-called "coned cecum," was not seen, but could also be found in tuberculous ileocolitis.[7]

MUCOSAL CHANGES

These include ulcerations, thumbprintings, destruction of the mucosa in the acute stages in amebomata, and total mucosal effacement as in the "pipe-stem" colon. These types have been earlier mentioned.

ILEOCECAL INCOMPETENCE
(Cases II, III, IV, VI, and VIII)

Incompetence of the ileocecal valve was seen in the cases with total colonic involvement, due to the extension of the disease to the cecum and its ileocecal valve (Fig. 6; and 9).

The types of lesions described above occur in all forms of combinations.

DISCUSSION

Most of the cases of amebic colitis in Ibadan are mild and present with symptoms of diarrhea with no constitutional upset. However, in about 25 per cent of the cases and in particular in those patients with pregnancy, malnutrition or other forms of impaired defense mechanisms, acute and severe forms of the disease occur.[1,6] The illness in these cases was often complicated with severe dehydration, metabolic imbalance, toxemia, perforations and peritonitis. The mortality was also high in this group.

There are few cases who continued to have chronic or repeated episodes of diarrhea after the initial acute amebic colitis had been treated; such a case, described as postdysenteric colitis by Hurst,[5] is illustrated by Case VI.

Rarer still are those cases that present with abdominal mass in the right iliac fossa due to ameboma or intussusception.

The radiologic changes tend to reflect the pathologic changes taking place in the colon. In the mild forms of the disease mucosal ulcerations are predominantly found. These are seen radiologically as shallow fine serrations or as "saw-toothed" marginal irregularities. As they enlarge and become confluent they form the "collar-stud" abscess lying underneath the mucosa. In fulminating cases the ulcers become very deep and might perforate, or there may be severe destruction of the

mucosa. Pseudodiverticula formation has not been previously reported and is most probably due to severe "collar-stud" abscesses and/or concomitant destruction and weakness of the muscle layer.

The mucosal lesions occur at multiple sites and varying lengths along the colon. The lesions could also involve the total length of the colon as seen in 5 of our cases; in these cases there was no intervening normal mucosa as usually described.[10]

Amebiasis may involve the appendix simulating acute appendicitis.[2]

In the severe form of the colitis, the radiologic picture may be exactly like that of the idiopathic nonspecific ulcerative colitis with toxic megacolon and extreme distention attributed to the severe necrosis and damage of the muscular coat. When a plain film roentgenogram of the abdomen shows this picture, a barium enema examination might carry the risk of perforation.

The radiologic findings of lack of distention, ileocecal incompetence, strictures, and "pipe-stem" colon are demonstrations of the fibrosis of the lesions at these sites. Multiple strictures could occur. These lesions are found most commonly in the right side of the colon. Case VIII, in which fibrosis occurred within 2 weeks, demonstrates the rapidity by which fibrosis could occur in some fulminating cases.

The "pipe-stem" colon also marks the attempts at repair leading to fibrosis, rigidity and the pipe-stem configuration. This radiologic feature is well known in association with the idiopathic nonspecific ulcerative colitis; it is, however also found in amebic colitis and has been reported in tuberculous colitis.[7] Pseudopolyps are found in both idiopathic nonspecific ulcerative colitis as well as in amebic colitis. It is now shown that the pseudopolyps could also occur in amebic colitis.

Ameboma presents radiologically like a tumor and can also form a nidus for the formation of the intussusception. Stenotic areas or large filling defects in the nonspecific ulcerative colitis denote areas of malignant changes, whereas in intestinal amebiasis they are due to amebic granulomata. However, Reed and Anderson[11] pointed out that some granulomatous lesion of intestinal amebiasis may degenerate into malignancy.

DIFFERENTIAL DIAGNOSIS

The following conditions must be differentiated from amebic colitis:

(i) *Idiopathic ulcerative colitis*. This disease mimics intestinal amebiasis in many aspects. All changes such as "cobblestone" mucosa, toxic dilatation, ulcerations and the pipe-stem colon are seen, but are commoner in nonspecific chronic ulcerative colitis than in intestinal amebiasis. Histology of a biopsy specimen or response to antiamebic treatment is usually the means of resolving the diagnosis if active trophozoites are not seen in the stools. Sigmoidoscopic appearance of the 2 conditions is indistinguishable.[8] Histology of rectal biopsy may provide means of differentiation.[3] In chronic nonspecific ulcerative colitis there is atrophy and degeneration of the rectal mucosa as well as depletion of the goblet cells. These are absent in amebiasis. Colonic stricture formation is commoner with postdysenteric colitis than in the chronic nonspecific ulcerative colitis. The associated conditions such as erythema nodosum, urticaria, iritis, arthritis and pyoderma are not seen with postdysenteric colitis.

The indirect hemagglutination test which is positive for amebiasis will be negative in nonspecific ulcerative colitis. The latter condition, however, will give a positive Schwartzman phenomenon.

Amebiasis will respond to treatment with an amebacide such as Flagyl, but will not affect the nonspecific colitis.[10]

(ii) *Carcinoma*. The classical ameboma is usually cecal in position, with a symmetric deformity, fine marginal irregularities and with good response to antiamebic therapy. Carcinoma is usually asymmetric with completely destroyed mucosa, and shows no response to antiamebic therapy. The annular type of ameboma can only be

differentiated from carcinoma by its response to antiamebic treatment or histology.

(iii) *Tuberculosis.* The fine serration, the "coned cecum," the "pipe-stem" colon and the incompetent ileocecal valve are changes which have not only been described in amebiasis,[14] but also in tuberculosis.[7] However, ileal involvement and pulmonary changes which are rare in amebiasis are common in tuberculosis.

(iv) *Crohn's disease.* The terminal ileum is almost always involved in Crohn's disease, but usually normal in amebiasis. Segmental involvement and multiple strictures are extremely common in Crohn's disease, but rare in amebiasis, even though 2 of our cases showed this.

(v) *Actinomycosis.* This is a rare disease, often involving the cecum and appendix with irregular filling defects. The draining sinus and the histologic appearances help to distinguish it from amebiasis.

(vi) *Schistosomiasis.* This disease is usually a polypoidal left-sided colitis; involvement of the cecum is rare. Stool examination or biopsy of the rectum gives a definitive diagnosis.

Radiologic examination is the method of choice for illustrating the location, type, extent and severity of amebic intestinal lesions. The changes following treatment can also be easily demonstrated radiologically. One of the other important roles of radiology in intestinal amebiasis is in demonstrating complications such as fistulae, perforation, peritonitis, cholecystitis, hepatic abscess and arthritis.[2,14]

There are very similar radiologic changes in idiopathic chronic ulcerative colitis and amebic colitis and only when these are considered along with the other epidemiologic, clinical and laboratory findings can clear distinction between the 2 conditions be made.

SUMMARY

Eight cases of intestinal amebiasis are described: 4 with ameboma (one of which was annular in type simulating a carcinoma, and another presenting as intussusception); 2 with toxic dilatation; 2 with multiple strictures; and 1 with pseudodiverticula.

Intussusception and multiple strictures are rare in amebiasis. Pseudodiverticula are unknown in amebiasis. These changes are reported in this series.

One case presenting with toxic dilatation demonstrated the radiologic progression from the initial ulcerative type of the disease to the late chronic stage of the disease with the classical "pipe-stem" colon. Another fulminating case demonstrated that fibrosis could occur in areas of severe mucosal changes within 2 weeks of treatment.

The disease involved the cecum in all cases, the entire colon in 5 cases, and the rectum in 4 cases.

The close similarities in the radiologic appearances of intestinal amebiasis and nonspecific chronic ulcerative colitis are emphasized.

The lack of involvement of the terminal ileum in amebic colitis is stressed in the discussion of the differential diagnosis of amebic colitis.

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REFERENCES

1. Abioye, A. A. Fatal amoebic colitis in pregnancy and puerperium: new clinico-pathological entity. *J. Trop. & Geog. Med.*, 1973, *1*, 97–100.
2. Davis, F. W. Amebiasis: experience in Johns Hopkins Hospital, 1936–1946. *Am. J. M. Sc.*, 1949, *217*, 505–517.
3. Fung, W. P., Monteiro, E. H., Ang., H. B., Koo, K. M., and Lee, S. K. Ulcerative postdysenteric colitis. *Am. J. Gastroenterol.*, 1972, *57*, 341–348.
4. Hardy, R., and Scullin, D. R. Thumbprinting in case of amebiasis. *Radiology*, 1971, *98*, 147–145.
5. Hurst, A. Medical Diseases of War. Third edition. Edward Arnold & Company, London, 1943.
6. Lewis, E. A., and Antia, A. U. Amoebic colitis

in Ibadan. *Tr. Roy. Soc. Trop. Med. & Hyg.*, 1967, *63*, 633–638.
7. LEWIS, E. A., and KOLAWOLE, T. M. Tuberculous ileo-colitis in Ibadan: clinical-radiological review. *Gut*, 1972, *13*, 646–653.
8. MADANAGOPALAN, N., VEDACHALAM, S. P., SUBRAMANIAM, R., and MURUGESAN, R. G. Rectal and colonic mucosal biopsy findings and feces: sigmoidoscopy, and histopathological correlation in amoebiasis and other colitis. *Gut*, 1968, *9*, 106–110.
9. NICHOLSON, J. H., and DE-DENOICIS, F. A. Amebiasis. *New England J. Med.*, 1947, *237*, 799–805.
10. PARDO-GILBERT, A., PEREZ-ALVARADO, N., and ZAVALA, B. Differential diagnosis of nonspecific and amoebic ulcerative colitis: survey of 100 patients. *Dis. Colon & Rectum*, 1972, *15*, 147–149.
11. REED, A. C., and ANDERSON, H. H. Amebiasis and cancer of colon. *Am. J. M. Sc.*, 1936, *191*, 237–250.
12. STEWART, G. T. Post-dysenteric colitis. *Brit. M. J.*, 1950, *1*, 405–409.
13. TCHANG, S. Amoebiasis in Northern Saskatchewan: radiological aspects. *Canad. M. A. J.*, 1948, *99*, 688–695.
14. WEINFELD, A. Roentgen appearances of intestinal amebiasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 311–322.

INTESTINAL SPECIMEN ROENTGENOGRAPHY: A THREE WAY CORRELATION*

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ALTHOUGH specimens from the intestinal tract lend themselves to easy roentgenography, only scattered reproductions are found in textbooks and journals. Correlation of such specimen roentgenograms with pathology is done sporadically in some institutions, but no planned and systematic studies are reported in the literature. Correlated roentgenographic and pathologic studies of intestinal specimens were carried out by Brahme and Lindström,[3] but the study was limited to the vascularity of the intestine.

The roentgenograms of 12 surgical intestinal specimens, obtained in our Institution in the past 6 months, prompted this study. The radiologist, the surgeon and the pathologist became directly involved. This correlation, revolving around the specimen roentgenogram, brought practical patient management advantages in addition to the obvious learning and teaching values.

Five illustrative cases are presented with comments on their practical or teaching value.

MATERIAL AND METHOD

Twelve cases were involved in the correlative study. Nine patients had granulomatous disease of the intestine, 2 patients had lymphoma of the intestine and 1 patient had a leiomyoma. Three surgeons, 3 radiologists and 2 pathologists were involved in the study. All patients had barium intestinal studies performed in this Institution shortly before surgery.

The initiator in each case was the radiologist who had performed the roentgen examination. Immediately upon completion of the operation, the surgeon brought the unopened specimen to the radiologist, describing in detail the operative findings and the surgical procedure. The specimen was then roentgenographed in the same position as the *in vivo* roentgenogram. Barium or air was usually injected into the specimen with a syringe or enema tip. The surgical clamps were disturbed as little as possible.

Filming was done at the table top by using detail screens and extremity technique. The roentgenograms obtained in this manner had to show good soft tissue detail in order to be of value.

A copy of the specimen roentgenogram was given then to the pathologist together with the specimen. The specimen was oriented in the same way as the roentgenograms and photographed. Pathologic examination of the specimen was carried out in the usual manner.

REPORT OF CASES

CASE 1. *Invasive lymphoma of the ileum.* A 59 year old man was admitted to Northwestern Memorial Hospital with a 10 week history of progressive epigastic cramping pain, occurring 1 to 2 hours after meals and lasting about 2 hours. Positive physical examination findings were limited to the abdomen where a 3×8 cm. nontender mass was palpated to the left of the umbilicus. The stool was 3+ guaiac positive for occult blood and the hematocrit was 30 per cent.

The barium meal small bowel studies (Fig. 1*A*) revealed a dilated loop of ileum approximately 30 cm. in length. The loop was fixed, had lost its normal mucosal pattern and was separated from the other loops by a significant space.

An abdominal laparotomy was performed where a large firm tumor involving the ileum

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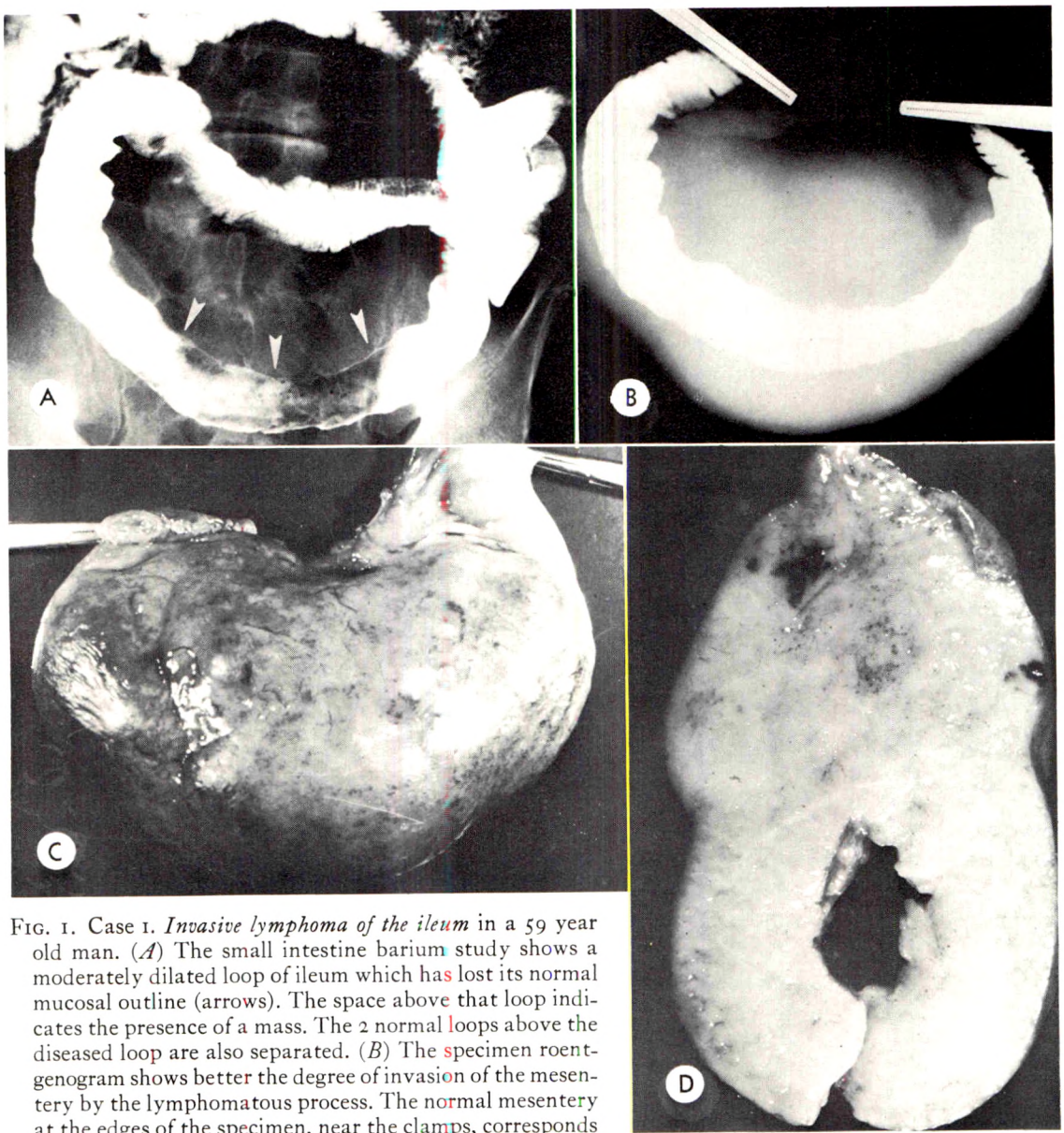


Fig. 1. Case 1. *Invasive lymphoma of the ileum* in a 59 year old man. (*A*) The small intestine barium study shows a moderately dilated loop of ileum which has lost its normal mucosal outline (arrows). The space above that loop indicates the presence of a mass. The 2 normal loops above the diseased loop are also separated. (*B*) The specimen roentgenogram shows better the degree of invasion of the mesentery by the lymphomatous process. The normal mesentery at the edges of the specimen, near the clamps, corresponds to the normal small bowel in the same areas. (*C*) The photograph of the specimen depicts the kidney-shaped, fleshy appearing tumor. (*D*) A transverse section of the specimen shows the wide open lumen, flat-destroyed mucosa and the lymphomatous invasion of the mesentery.

was found. A second mass, consisting of enlarged mesenteric lymph nodes, was found at the base of the small bowel mesentery and a third similar mass was found adjacent to the duodenum. The involved ileal loop with a short segment of normal bowel at each end was resected and an end-to-end anastomosis was performed. The enlarged mesenteric lymph nodes were not resected.

The roentgenogram of the specimen (Fig. 1*B*) showed the same size, appearance and mucosal pattern of the loop of ileum as on the *in vivo* study. The surrounding mass, however, became more obvious. It had a uniform "fleshy" appearance. The mass merged into the normal mesentery at about the level of normal bowel loops, seen at the edges of the specimen.

A photograph of the gross specimen (Fig. 1*C*) revealed a kidney-shaped mass with a short segment of normal bowel at each end. The

mesentery was normal. The entire mass measured 19×12×6 cm. A cross section of the specimen (Fig. 1*D*) showed a patent lumen, surrounded by thickened walls composed of homogeneous, light, tan-gray tissue resembling lymphoid tissue. The mesenteric portion of the bowel had a thickness of about 5 cm.

The microscopic diagnosis was malignant lymphoma, mixed plasmocytic and lymphocytic type, involving the omentum, lymph nodes and the small intestine.

Comment. The involved loop is dilated, fixed and has a flat mucosa, which appears infiltrated or destroyed. The marked thickening of the diseased intestinal wall clearly separates this loop from the surrounding normal bowel as seen on the *in vivo* roentgen examination (Fig. 1*A*). This roentgenogram also reveals a space between the 2 normal loops of small bowel immediately above the diseased loop. This space is caused by the enlarged mesenteric lymph nodes which are seen in 70 to 100 per cent of patients with intestinal lymphoma.[2]

The specimen roentgenogram confirms the maximum distensibility of the diseased loop and gives the exact location of the intestine in relationship to the mass. This helped the pathologist to make the proper section of the gross specimen. The normal appearing segments seen at both ends of the specimen indicated to the surgeon that the excision had been sufficiently wide.

The specimen roentgenogram in this instance indicates that nothing more could have been done roentgenographically *in vivo* to better demonstrate the lesion. The specimen roentgenogram helps to classify the lymphomatous process as invasive and also gives some indications for immediate prognosis.

Case II. *Infiltrative lymphoma of the ileocecum.* An 80 year old woman was admitted to Northwestern Memorial Hospital because of an enlarging mass at the base of her tongue. She gave a 2 year history of diarrhea, crampy abdominal pain and occasional black stools. The physical examination revealed a 2×1 cm. firm, nontender, nonulcerated mass at the base of the tongue. The only other abnormal physical finding was a 6×8 cm. nontender mass in the right lower quadrant of the abdomen. The barium enema and small bowel barium studies revealed a lesion of the terminal ileum extending into or involving the cecum. The lumen of the ileum was narrow and grossly deformed. The surrounding mass was penetrating and causing an impression upon the ileocecal valve and the cecum (Fig. 2*A*). The biopsy of the tongue lesion revealed a malignant lymphoma, lymphocytic type.

An exploratory laparotomy was performed. A large group of firm lymph nodes surrounding the celiac artery was found. In addition, multiple large lymph nodes were discovered throughout the mesentery of the small bowel. In the area of the terminal ileum, a group of lymph nodes forming a mass of approximately 8 cm. in diameter was noted. This mass arose in the mesentery, but infiltrated the terminal ileum and the cecum. The terminal ileum and right colon with its involved mesentery was resected and an end-to-end ileotransverse colostomy was constructed.

The roentgenogram of the barium-filled specimen (Fig. 2*B*) showed large masses consisting of enlarged lymph nodes located in the mesentery of the terminal ileum above and extending into the ileocecal valve area. The lumen of the ileum was still visible but the mucosa was markedly flattened, deformed and had a scalloped appearance. The cecum was not distensible and a mass was seen in the ileocecal valve area.

The external examination of the specimen disclosed a multinodular ovoid tumor at the serosal aspect of the ileocecal valve area. The tumor measured approximately 11×6×6 cm. It appeared to consist of multiple nodules which had coalesced. Section of the tumor (Fig. 2*C*) disclosed a pale "fleshy" appearance of the cut surface. There were multiple nodules which were very discrete in some areas but had coalesced in others. The wall of the ileum was markedly thickened and was infiltrated and partially replaced by fleshy tumor. The most proximal 2 cm. of the ileum had an average wall thickness of 3 mm. and a circumference of 4 cm. Distally, the wall of the ileum became thicker, measuring 1.5 cm. at the ileocecal valve. The distal 5 cm. of ileum proximal to the valve was partially occluded by a nodular appearing overgrowth of pale fleshy tumor. The ileocecal valve was grossly thickened and was replaced by tumor. The remainder of the



Fig. 2. Case II. *Infiltrative lymphoma of the ileocecum* in an 80 year old woman. (*A*) Spot roentgenogram of the terminal ileum shows a patent lumen, which is constricted and distorted in a scalloped fashion (arrows). There may be a surrounding mass. The cecum appears to be infiltrated in the ileocecal valve region. (*B*) The specimen roentgenogram shows normal cecum at the top clamp and a normal ileum at the bottom clamp. The coalesced lymph nodes are well demonstrated above the ileum, infiltrating the terminal ileum and the ileocecal valve. The scalloped appearing lumen is similar to the *in vivo* roentgenogram. (*C*) The sagitally opened specimen shows the tumor (on both sides of the ruler) and the infiltrated ileocecal valve (arrows).

cecum was adherent to the main tumor but was not invaded.

Microscopic examination revealed malignant lymphoma, poorly differentiated lymphocytic, diffuse type, involving the terminal ileum, cecum and mesenteric lymph nodes.

Comment. Contrary to Case I, where the lymphomatous process in the bowel wall extended into the mesentery keeping the lumen of the intestine open, in Case II, the process involving the mesenteric lymph nodes infiltrated the terminal ileum and the ileocecal valve producing stenosis. All the roentgen signs of infiltrative lymphoma, as given by Balikian *et al.*[2] are seen on the specimen roentgenogram, namely: thick folds, scalloped appearance, flattening of the mucosa, and constriction.

The specimen roentgenogram indicated that the lines of excision were satisfactory to the surgeon, it helped the pathologist to orient the specimen and make the proper section to demonstrate the involvement. To the radiologist, it showed that the extent of the lesion was well demonstrated by the spot roentgenograms of the terminal ileum.

Case III. *Leiomyoma of the jejunum.* A 27 year old man was admitted to Northwestern Memorial Hospital with a 10 year history of cramping lower abdominal pain and intermittent gastrointestinal bleeding for the past year. He complained of increased fatigability, cramps and occasionally dark stools, but was otherwise in good health. The physical examination was within normal limits. Stool specimens were 3+ guaiac positive for occult blood and the patient's hemoglobin on admission was 8.5 gm. per cent.

The small bowel barium study revealed retention of barium in a diverticulum-like structure originating from the medial wall of the jejunum (Fig. 3*A*). On some of the roentgenograms, loops surrounding the barium collection suggested the presence of an extrinsic mass.

An exploratory laparotomy was performed. A mass was found in the right upper quadrant of the abdomen. The mass was delivered from

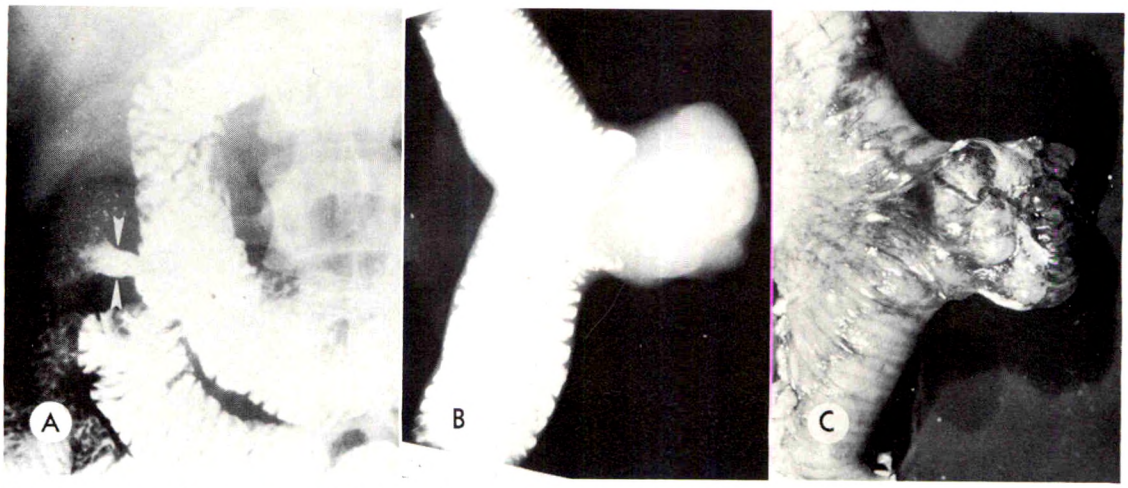


FIG. 3. Case III. *Leiomyoma of the jejunum* in a 27 year old man. (*A*) The gastrointestinal examination shows a glove finger like collection of barium (arrows) in the right upper quadrant, filled from the loop located to the right of the collection. (*B*) The specimen roentgenogram demonstrates the subserosal character of the leiomyoma. The ulceration penetrates deeply into the tumor. The antimesenteric location of the mass is obvious. (*C*) The photograph of the specimen corresponds well to the specimen roentgenogram.

the abdomen and was found to consist of a lobular structure measuring approximately 10×6 cm., which was attached to the antimesenteric border of a loop of proximal jejunum. The mass had pearly, white lobulations as well as densely injected lobulations. It appeared to be leiomyoma. The mass and its attached jejunum were resected with approximately 10 cm. of normal bowel on each side. Intestinal continuity was restored with an end-to-end jejunojejunostomy.

The roentgenogram of the barium-filled specimen (Fig. 3*B*) showed a normal lumen of the segment of jejunum except for slight flattening at the site of an intramural mass which originated on the antimesenteric wall of the bowel extending about 4.5 cm. beyond the wall of the normal bowel. The subserosal location of the mass was well demonstrated. The tumor contained a long ulceration which communicated with the lumen of the bowel and corresponded to the diverticulum-like barium-filled structure seen on the *in vivo* roentgenograms. The lobulated appearance of the mass was well demonstrated.

The gross photograph of the specimen (Fig. 3*C*) revealed the subserosal location of the tumor which extended far beyond the wall of the normal bowel. Areas of hemorrhage were noted. The microscopic examination confirmed the diagnosis of a benign leiomyoma with central necrosis.

Comment. The subserosal type of intestinal leiomyoma is not easily detected roentgenographically, unless there is ulceration connecting the lumen of the intestine with the necrotic center of the tumor.[1] The submucosal types extend into the lumen of the intestine and are more easily detected on a barium examination. Other occasionally encountered roentgen signs of subserosal leiomyoma can be seen on the specimen roentgenogram: flattening of the mucosa at the site of involvement and the presence of a soft tissue mass produced by the protruding tumor which may cause displacement of neighboring organs.

An extraluminal "glove finger" collection of barium may represent ulceration in a subserosal leiomyoma, but may also represent a Meckel's diverticulum when the lesion is in the ileum.

CASE IV. *Granulomatous enteritis involving a gastrojejunostomy.* A 51 year old woman was admitted to Northwestern Memorial Hospital with a 2 month history of sharp, cramping right upper quadrant pain. The pain occurred after meals and was associated with nausea and vomiting. At age 34, the patient underwent a total colectomy with an ileostomy for what was diagnosed as chronic ulcerative disease of the

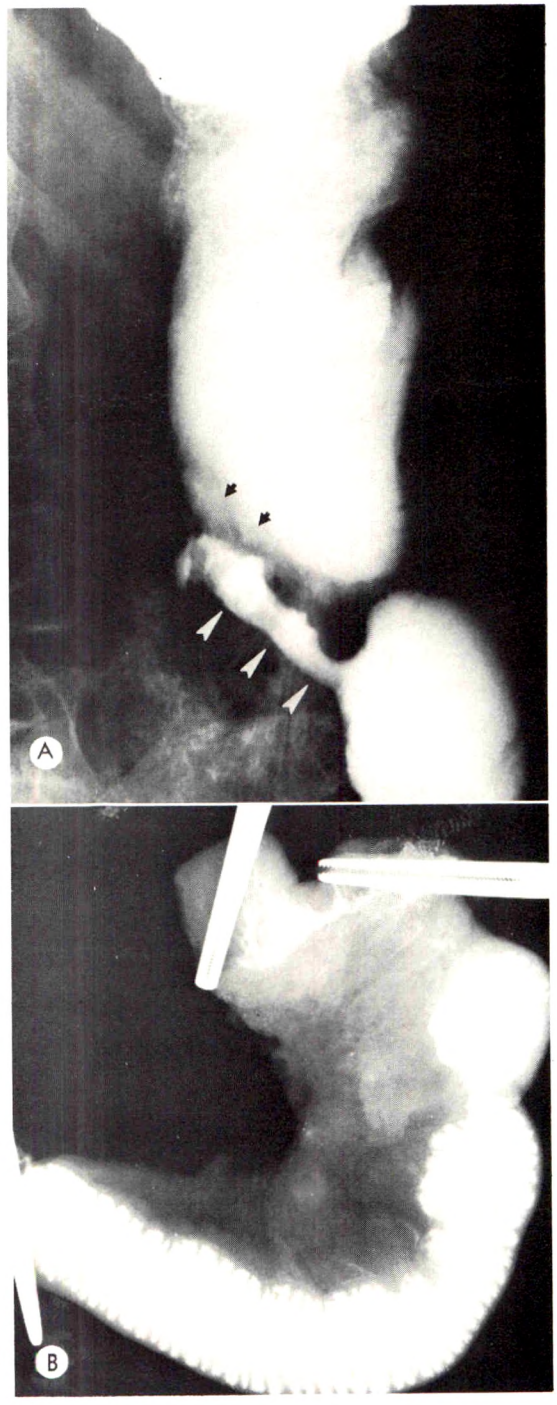


Fig. 4. Case IV. *Granulomatous enteritis involving the gastrojejunostomy* in a 51 year old woman. (*A*) The upper gastrointestinal examination shows a distended stomach draining slowly through a gastrojejunostomy (black arrows). The afferent loop is not filled, the sausage like efferent loop (white arrows) is narrow, rigid and empties into a moderately dilated loop of jejunum. (*B*) The specimen roentgenogram shows well the granulomatous involvement of the jejunum at the anastomosis with the stomach held by the horizontal clamp. The top vertical clamp is on the afferent loop, the bottom vertical clamp is on the efferent loop. The increase in mesenteric fat accounts for the 3 cm. circumference of the involved jejunum. Note the tiny but deep ulceration of the diseased segment and the transition of thick to normal mesenteric fat at the limit of granulomatous involvement.

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rectosigmoid. At age 38, the patient developed anorexia with weight loss of 27 pounds, accompanied by vomiting and epigastric distention. Obstruction of the second portion of the duodenum was found on a barium meal examination. At operation granulomatous duodenitis was diagnosed, and a gastrojejunostomy and vagotomy were performed.

The physical examination was within normal limits at present admission. A barium meal examination (Fig. 4*A*) showed a moderately distended stomach with a gastrojejunostomy. The afferent loop did not fill. A 5 cm. segment of the proximal efferent loop was markedly narrow and deformed. Distal to this stenotic segment the efferent loop became moderately dilated. The stomach emptied very slowly.

At operation, the stomach revealed marked thickening and stenosis of the pylorus. The anticolic gastrojejunostomy which had been placed high on the greater curvature of the stomach was stenotic. Beginning at the ligament of Treitz this afferent limb of the gastrojejunostomy was normal for approximately 10 cm. At this point, 2 cm. from the gastrojejunostomy, the jejunum became thickened and stenotic. The disease extended through the gastrojejunostomy and involved the proximal 5 cm. of the efferent limb. The remainder of the small bowel was normal. The segment of diseased jejunum including the gastrojejunostomy was excised. An end-to-end jejunojejunostomy was constructed 4 cm. from the ligament of Treitz, and a new side-to-side gastrojejunostomy was constructed 8 cm. distally.

The specimen was filled with barium, oriented similar to the roentgenogram taken *in vivo*, and a roentgenogram was taken (Fig. 4*B*). Both ends of the jejunum connected to the gastrojejunostomy were narrow and showed super-

ficial and deep ulcerations with marked thickening of the mesenteric fat.

The pathologic specimen consisted of a loop of jejunum with an opening at the antimesenteric side representing the gastric ostium. There was stenosis on either end of the gastric ostium. The proximal narrow area was located in the afferent limb and was 2 cm. in length, the distal stenotic area was located in the efferent limb and was 13 cm. long. The circumference of the bowel wall at the narrow area was approximately 3 cm. The mucosa was diffusely hyperemic and showed patchy linear and irregular, superficial mucosal ulcerations. The serosal surface had a bluish discoloration.

The microscopic diagnosis was granulomatous disease of the jejunum at the site of the gastrojejunostomy.

Comment. Granulomatous duodenitis is rare and most authors recommend treatment by gastrojejunostomy and vagotomy.[4] Recurrence of granulomatous disease after resection is usually expected proximal to the suture line. In this case no resection was done, but the jejunum was incised on its antimesenteric border in creating a gastrojejunostomy. It is interesting to note that the granulomatous disease recurred at this point.

The pipe stem or sausage appearance of the involved segment is characteristic of the stenotic form of granulomatous jejunitis.[5] The specimen roentgenogram demonstrates the typical findings of active granulomatous disease: the increase in circumference of the loop due to the hyperplastic changes in the submucosa, the increase in mesenteric fat and the tiny but deep ulcerations.

Case v. *Granulomatous ileitis involving a Meckel's diverticulum.* A 29 year old man was admitted to Northwestern Memorial Hospital with a history of 4 weeks of intermittent diarrhea, occasional cramping abdominal pain and weight loss. He had also noted nocturia and pneumaturia, and occasionally his urine contained blood and particulate matter. On 3 occasions, he described his urine as being "just like diarrhea."

The patient had been hospitalized first at age 26 after 6 months of diarrhea and a 35 pound weight loss. Granulomatous ileitis was diagnosed by a small bowel barium examination. He was placed on medical management. He was hospitalized again 9 months later with cramping abdominal pain and a perirectal abscess. The perirectal abscess was incised and drained.

At the present admission physical examination was within normal limits. A small bowel barium examination revealed a long segment of terminal ileum (Fig. 5*A*) which was narrow, markedly irregular and had thickening of the wall. Before the barium reached the most terminal segment of the ileum, a fistulous tract was demonstrated leading into the rectum. The cecum was markedly spastic, irritable and not sufficiently fully distended for evaluation.

Cystoscopy was performed prior to surgery and showed edema of the left posterior wall of the bladder, the apparent site of a bladder fistula.

An abdominal laparotomy was performed, revealing an inflammatory mass involving the distal 20–25 cm. of ileum. This mass was adherent posteriorly and attached to the bladder at its right posterior superior aspect and attached to the sigmoid colon on its medial aspect. The terminal ileum was indurated and inflamed with the mesenteric fat encroaching at the mesenteric border of the ileum. The cecum appeared to be inflamed and thickened at its junction with the ileum. A Meckel's diverticulum arose from the terminal ileum 25 cm. from the ileocecal valve, and its tip was attached to the inflammatory mass 6 cm. from the ileocecal valve. The mass was dissected free of the bladder, and a 3 mm. defect in the bladder was closed in 2 layers. When the mass was dissected from the sigmoid colon, a 1 cm. defect in the sigmoid colon was visualized. A terminal ileostomy and right hemicolectomy were performed with an end-to-end ileotransverse colostomy.

The specimen roentgenograms with barium and air (Fig. 5, *B* and *C*) showed the involved ileum which presented multiple deep ulcerations penetrating into the submucosa. The Meckel's diverticulum was well seen, and was involved by granulomatous disease with its tip adherent to the terminal ileum. The cecum appeared to be uninvolved.

The pathologic specimen showed that the portion of diseased ileum had a dull mucosal surface, purple tan in color, which was firm and indurated. Adjacent to the uninvolved part of ileum, there was a diverticulum measuring 3

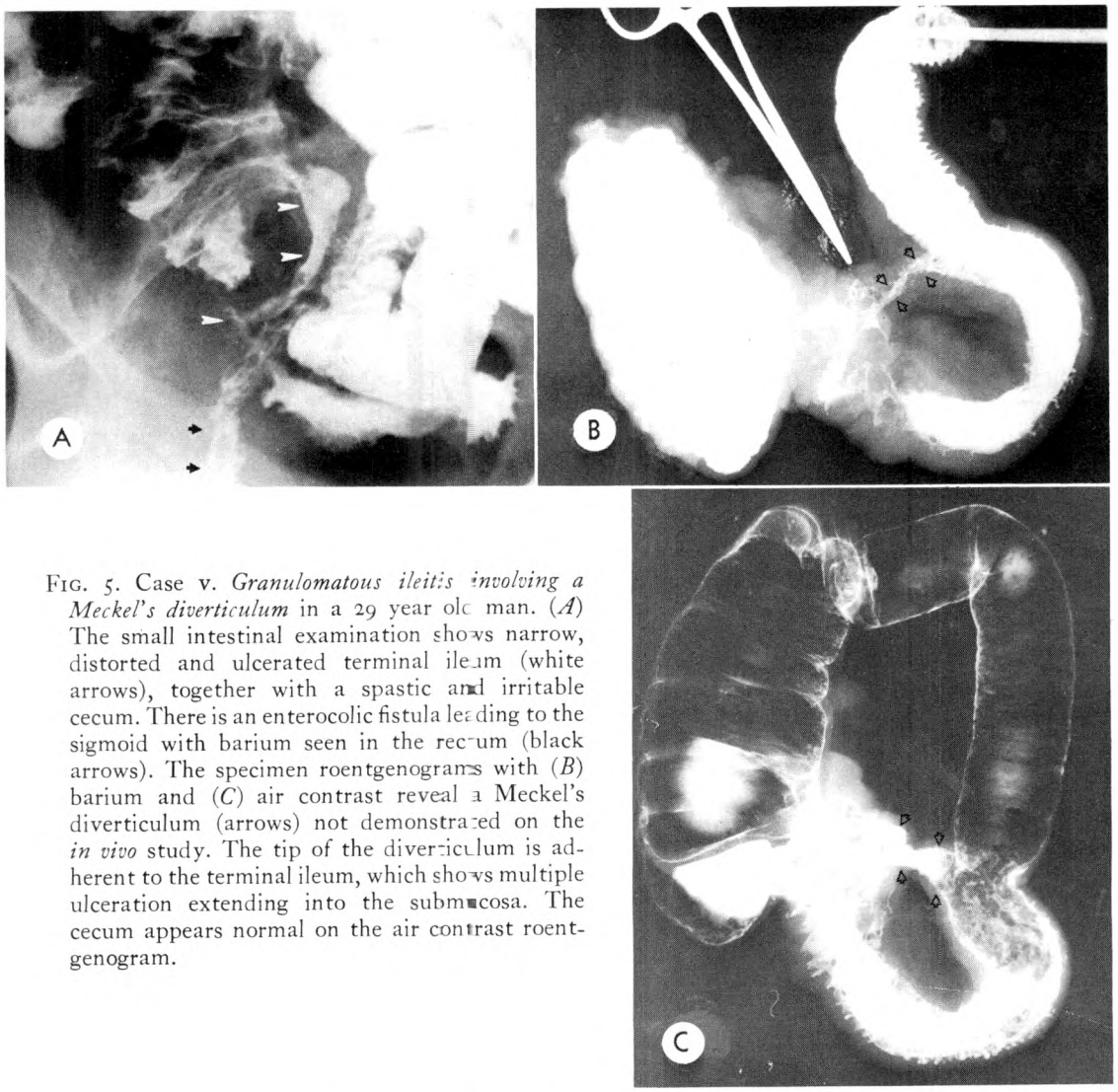


Fig. 5. Case v. *Granulomatous ileitis involving a Meckel's diverticulum* in a 29 year old man. (*A*) The small intestinal examination shows narrow, distorted and ulcerated terminal ileum (white arrows), together with a spastic and irritable cecum. There is an enterocolic fistula leading to the sigmoid with barium seen in the rectum (black arrows). The specimen roentgenograms with (*B*) barium and (*C*) air contrast reveal a Meckel's diverticulum (arrows) not demonstrated on the *in vivo* study. The tip of the diverticulum is adherent to the terminal ileum, which shows multiple ulceration extending into the submucosa. The cecum appears normal on the air contrast roentgenogram.

cm. in length and 1.5 cm. in diameter. The diverticulum was adherent to the terminal portion of the ileum. The terminal ileum from the area of the diverticulum to the ileocecal valve had a hyperemic edematous mucosa and multiple ulcerations. The cecum revealed superficial ulcerations up to 2 mm. in diameter.

The microscopic diagnosis was granulomatous ileitis involving also the Meckel's diverticulum.

Comment. Granulomatous involvement of a Meckel's diverticulum has been reported before. It may coexist with granulomatous ileitis or be confined only to the diverticulum, as reported by Solomons and Halford.[6]

The specimen roentgenograms in this case point out the real value of such a study. The Meckel's diverticulum was identified on the specimen roentgenogram but not on the *in vivo* roentgenogram and helped the pathologist to orient himself in the specimen and to determine the exact areas of involvement. The uninvolved cecum and ileum are well seen on the air study. However, the cecum, although appearing normal on the specimen roentgenograms, had definite superficial ulcerations on the pathologic examination. This probably accounted for the spasticity of the cecum on the *in vivo* barium examination.

DISCUSSION

From the comments given after the cases reported, it is obvious that there are definite benefits from a correlated study of diseased intestinal specimens.

To the pathologist, who is expected to study the specimen in order to supply the surgeon not only with an anatomic diagnosis, but also with data on the extent of the diseased process and line of excision in relationship to the disease, the specimen roentgenogram is of great help. When it is combined with the specimen and supplemented with a photograph of the specimen, the pathologist can refer back to the intact specimen long after it has been cut. The exact location of the diseased process in space and location in the body can easily be determined with the aid of the specimen roentgenogram.

To the surgeon, the immediately obtained specimen roentgenogram can assist in determining if the lesion was completely excised. Although frozen sections of ends of the specimen can be obtained, they may be difficult to interpret and may not reveal minimal disease.

To the radiologist, the value of the specimen roentgenogram can be immeasurable. By comparing the specimen roentgenogram with the roentgenograms done *in vivo*, the following questions can be clearly answered: how well was the *in vivo* gastrointestinal study done; what should have been done in terms of technique to better demonstrate the lesion; was the extent of the lesion well outlined; and finally were all the roentgen signs utilized to make the correct roentgen diagnosis?

The teaching value of this 3 way correlation is considerable. When residents and students are confronted with a gastrointestinal study, a clinical setting, a specimen roentgenogram, a gross specimen photograph and a final microscopic diagnosis, the entire disease process can be appreciated. Rather than memorizing a group of symptoms and the roentgenographic appearance of a lesion, one can recognize why these symptoms are produced, and how the pathologic anatomy results in these characteristic roentgenographic signs.

SUMMARY

A 3 way correlation in handling surgically removed intestinal specimens is proposed. The correlation involves the radiologist, the surgeon and the pathologist and is centered on the specimen roentgenogram taken immediately after surgery.

The advantages and teaching value of this correlation is illustrated by 5 cases: 2 patients with intestinal lymphoma, 2 patients with granulomatous disease, and 1 patient with a leiomyoma of the jejunum.

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REFERENCES

1. Baker, H. L., Jr., and Good, C. A. Smooth-muscle tumors of alimentary tract: their roentgen manifestations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 246–254.
2. Baliklan, J. P., Nabil, T. N., Shamma'a, M. H., and Shahid, M. J. Primary lymphomas of small intestine including duodenum. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 131–141.
3. Brahme, F., and Lindström, C. Comparative radiographic and pathological study of intestinal vaso-architecture in Crohn's disease and in ulcerative colitis. *Gut*, 1970, *11*, 928–940.
4. Colcock, B. P. Operative technique in surgery for Crohn's disease and its relationship to recurrence. *Surg. Clin. North America*, 1973, *53*, 375–380.
5. Marshak, R. H., and Wolf, B. S. Chronic ulcerative granulomatous jejunitis and ileojejunitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *70*, 93–112.
6. Solomons, D., and Halford, M. E. H. Crohn's disease of Meckel's diverticulum occurring in case of jejunal diverticulitis. *Brit. J. Surg.*, 1964, *51*, 910–913.

Vol. 122, No. 2

HYSTERICAL ABDOMINAL PROPTOSIS*

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HYSTERICAL, nongaseous abdominal bloating or proptosis was described as long ago as 1824 by the celebrated John Bright, while a personal experience of 92 cases, with a full and fascinating discussion of the syndrome, was reported by Alvarez.[1]

The standard radiologic texts, even those devoted to abdominal diagnosis, make no mention of it, nor is it included in the differential diagnosis of idiopathic intestinal pseudo-obstruction.[2]

The bloating usually develops rapidly and repetitively and is often painful, so that, as in the case reported here, it may devolve upon the radiologist to save the patient from laparotomy.

REPORT OF A CASE

Agonizing screams alerted the radiologist to the patient on the radiographic table. The request-form stated simply: "Diffuse abdominal pain for several hours, with distention. Any fluid levels?" The 38 year old patient's actions resembled those of a woman in labor. At intervals of several minutes she would announce the onset of another spasm, followed by screaming, facial contortions and breath-holding. The lateral parts of the lower abdomen began to bulge, with rapid spread to the entire lower central abdomen (Fig. 1), but its progression did not resemble peristalsis, nor were there any borborygmi or peristaltic rushes on auscultation. After about 2 minutes swelling and distress abated. The abdomen was somewhat tender.

The supine and erect roentgenograms (Fig. 2, *A* and *B*) showed normal intestinal gas pattern with a solitary fluid level in the ptotic duodenal bulb.

Horizontal-beam lateral views during the attacks (Fig. 3, *A* and *B*) showed the enormous degree of abdominal proptosis obtained. It is apparent that this was accompanied by descent of the diaphragm and displacement of the intestines forward, with some redistribution of the gas in them. The anterior abdominal wall, when protruded, appeared thicker rather than thinner as it would be in true distention; this represents the contribution of abdominal muscular contraction to the swelling.

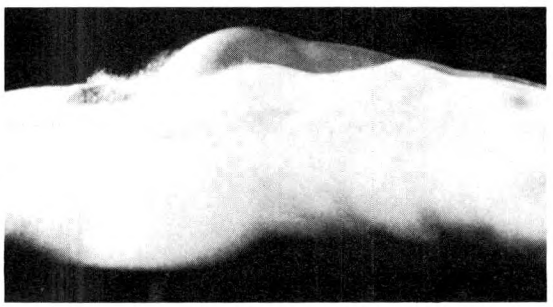


Fig. 1. Double exposure before and during hysterical abdominal proptosis in another case (courtesy of N. J. Roussak), but identical with ours.

Fortunate recall of an association over 20 years earlier[3] resulted in thwarting surgical interest in the case and referral for social and psychiatric help. The radiologist's history-taking revealed that the patient was a young widow, desperately trying to rear 4 small children with inadequate resources; the 'colics' had been occurring for some 2 years and the swelling over the last 3 months.

DISCUSSION

It is axiomatic that the diagnosis of functional disorders should not be merely by the exclusion of the organic, but by recognition of the specific psychologic pathogenesis and symptomatology.

The hysterical personality and the precipitating environmental situation have easily been elucidated in most of these cases. A century ago, when intestinal obstruction used to be relieved by trocar through the abdominal wall, the nongaseous type was directly diagnosed! Subsidence of the bloating without passage of flatus, has been demonstrated by having the end of the rectal tube submerged in water.[3] The bloat-

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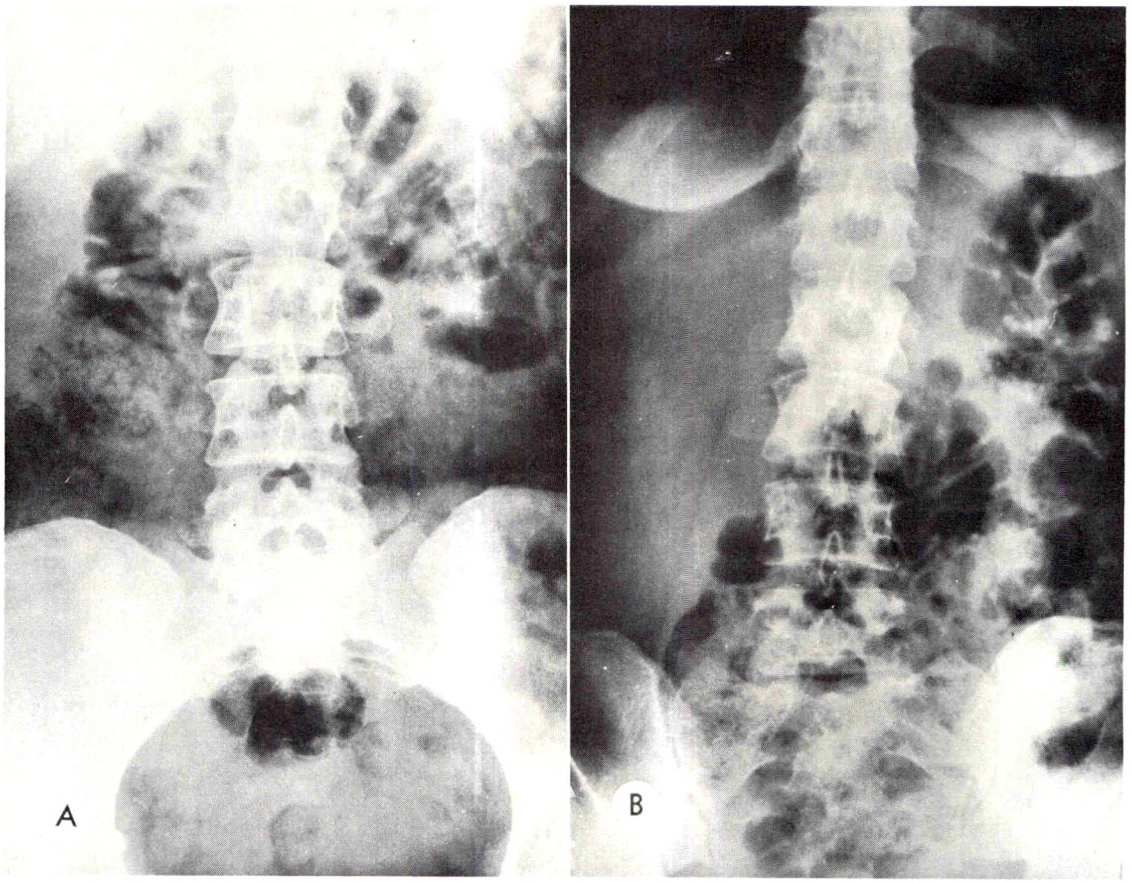
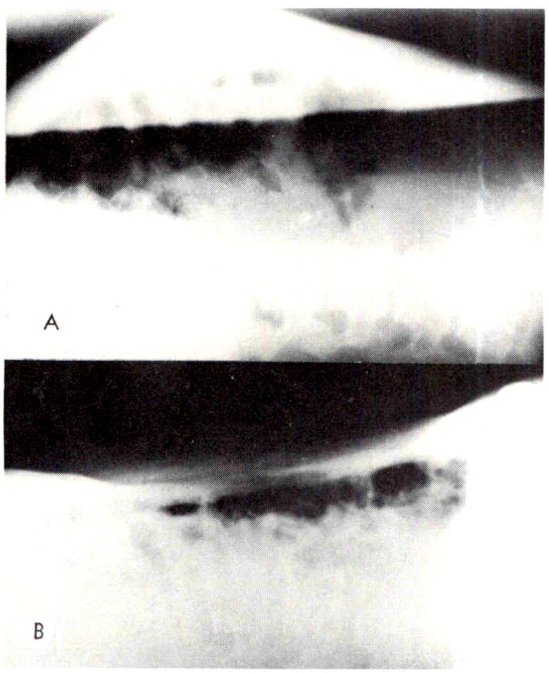


FIG. 2. (*A* and *B*) Supine and erect roentgenograms. No intestinal distention; no stasis.



ing may be prolonged and various measures, even general anesthesia, have been employed to terminate it.

Mechanisms involved in its production include asymmetric contraction of groups of abdominal muscles, often painful, exaggeration of the lumbar lordosis and sustained depression of the diaphragm. The muscular contortions inevitably disturb

FIG. 3. (*A* and *B*) Supine lateral roentgenograms during proptosis and between episodes. The intestines are protruded forward, partly by fixed descent of the diaphragm and by contraction of the lateral abdominal muscles. The contracted anterior muscles add to the proptosis; in true distention the anterior wall would be thinned. (Collimation provides suitable exposure for the anterior parietes.)

respiration and produce sensations of choking and distress, as well as abdominal tenderness. Naturally, peristalsis is normal in all phases of the disturbance.

CONCLUSION

The radiologic diagnosis stems from the absence of gaseous distention and the use of the supine lateral roentgenogram to show the altered contour of the parietes, diaphragm, spine and abdominal musculature producing the proptosis.

The radiologist needs to be cognizant of the radiology of hysteria!

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REFERENCES

1. Alvarez, W. C. Hysterical type of non-gaseous abdominal bloating. *Arch. Int. Med.*, 1949, *84*, 217–245.
2. Moss, A. A., Goldberg, H. I., and Brotman, M. Idiopathic intestinal pseudo-obstruction. Am. J Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 312–317.
3. Roussak, N. J. Hysterical abdominal proptosis. *Gastroenterology*, 1951, *17*, 133–137.

ANGIOGRAPHY IN ACTINOMYCOSIS OF THE ABDOMEN*

REPORT OF 2 CASES

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ACTINOMYCOSIS of the abdomen is fairly rare.[1,3,4,7] It is "the most misdiagnosed disease—one which is most often missed by experienced clinicians."[3] One of the main reasons for this is the varying pattern of the symptoms. The fungus may be silent for long periods in poorly vascularized tissue. It can cause phelgmon with marked hardness and abscesses, sometimes fistulating. The course is often influenced by secondary infections which may have been treated with antibiotics, so that the disease and the symptoms of actinomycosis are partly masked.

As far as we know only 1 case of abdominal actinomycosis has been published, where angiography has been performed.[2] Therefore, it has been thought worthwhile to describe another case. Both patients were examined at our department and the results of the former case have now been re-investigated.

REPORT OF CASES

CASE I. (Previously reported by Boijsen and Reuter, 1966.[2]) Male, 60 years old. Appendectomy at the age of 23. Psoriasis for the past 20 years. The patient came to the hospital after 3 months of tiredness with loss of appetite, voiding urge, and pains in the right groin. A tumor in the right abdominal fossa was palpated. On conventional colon examination a tumor was found to indent the cecum (Fig. 1), but there was no infiltration of the wall of the large bowel.

Angiography of the superior mesenteric, inferior mesenteric, and right common iliac artery was performed. A mass 6×6 cm. close to the cecum was demonstrated (Fig. 2). The mass was moderately hypervascularized with wide, well defined arteries, some of which showed slight changes in caliber. There was some venous pooling, but no arteriovenous shunting or accumulation of contrast medium. The primary diagnosis was malignant tumor.



FIG. 1. Case 1. There is an impression in the cecum and the most distal part of the ileum.

At operation a mass was excised composed of omentum, appendix (sic!), peritoneum, abdominal wall, musculature, and bladder wall. Thus the appendix was still in place despite the supposed appendectomy at the age of 23. Histologic diagnosis was: chronic, fistulating process with colonies of fungus as in actinomycosis. No complications were encountered postoperatively.

CASE II. Male, 50 years old, with lower abdominal pain for the past 10 years. Appendectomy at the age of 47. The patient had a stomach ulcer. At laparotomy a large, hard tumor

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mass was found to occupy the left lower part of the abdomen, extending into the small pelvis. The cranial dorsal part of the bladder and the descending and sigmoid parts of the large bowel were infiltrated. At biopsy no signs of malignancy were found. Transversostomy was performed. On conventional colon examination 8 days postoperatively (Fig. 3), irregular changes of the wall were found with stenosis and diverticula in a 15 cm. long part of the sigmoid. On repeat barium examination 1 month later, the appearance of the bowel was unchanged. At selective angiography of the inferior mesenteric artery (Fig. 4), tortuous and partly poorly demarcated arteries were seen in an 11×15 cm. mass in the sigmoid. No lumen irregularities or displacement of the arteries and no accumulation of contrast medium in the mass were found. The clinical, operative and roentgenologic findings all indicated the probability of an inflammatory lesion.

Three and a half months later another mass appeared in the scar of the previous laparotomy. Resection of the sigmoid and part of the descending colon and of the mass in the scar was undertaken. Histologic diagnosis: actinomycosis.



Fig. 3. Case II. Irregular changes in the wall of the sigmoid with stenosis and diverticula.

DISCUSSION

The angiographic appearance was in the first case very close to that of a malignant lesion, which was described by Boijsen and Reuter as follows[2]: "There were displaced arteries around the lesion and irregular vessels within it. There was no accumulation of contrast material and only slight early venous drainage. The angio-

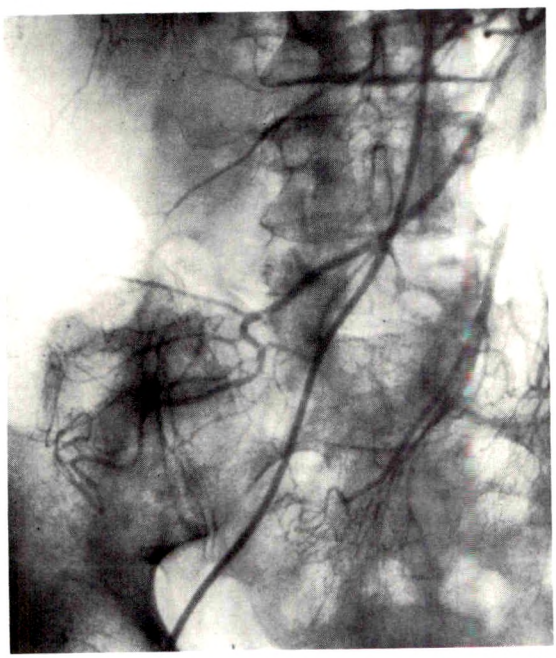


Fig. 2. Case I. A mass 6×6 cm. is supplied by the ileocolic artery. Moderate hypervascularization. Slight changes in caliber of the branches within the mass.

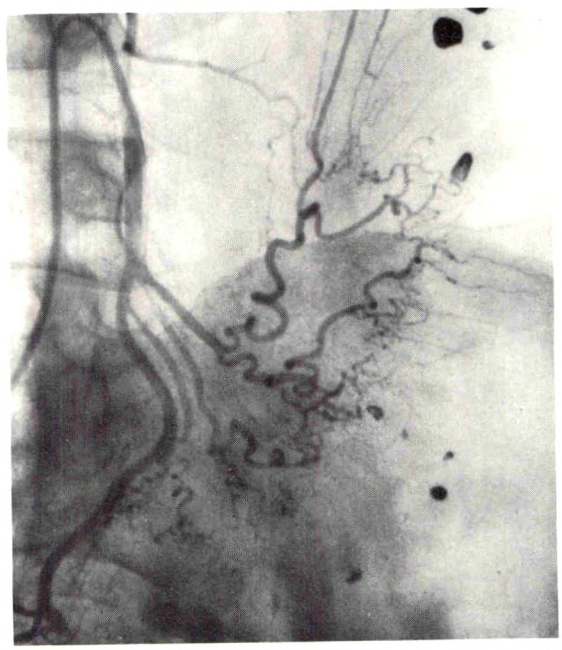


Fig. 4. Case II. Branches from the inferior mesenteric artery are wide, serpentine, partly unsharply demarcated but not infiltrated or dislocated. There is some barium contrast medium retained in the diverticula.

graphic appearance of the lesion was indistinguishable from carcinoma."

On reviewing the case there is no important change to be made in the earlier description except that the dislocated arteries were wide and that there was some venous pooling.

In the second patient the angiographic appearances were in favor of an inflammatory process; wide mesenteric arteries were indicative of a moderately increased flow of blood and the veins emptied the contrast medium early. Due to shrinking, the mesenteric and marginal arteries were tortuous and the vasa recta were close to each other. There was no occlusion of vessels. The conditions might be interpreted as diverticulitis with fibrosis[8] or as tuberculosis,[6] but the possibility of a poorly vascularized malignant tumor could not be ruled out. Ulcerative colitis was considered unlikely as the vasa recta were not dilated and there was no accumulation of contrast medium in the bowel wall. Against the possibility of regional enteritis was the fact that there was no narrowing of the arteries in a thickened mesentery and also that the vasa recta did not originate at right angles from the stems.[5,8]

The angiographic appearances of the 2 patients did not show any changes that could be looked upon as specific for actinomycosis. The differential diagnosis comprised poorly vascularized malignant tumor, regional enteritis, and tuberculosis.

SUMMARY

Two cases with actinomycosis involving the large bowel are reviewed.

At angiography one of the cases simulated a malignant tumor, the other had the characteristics of an inflammatory lesion.

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REFERENCES

1. Adar, R., Antebi, E., David, R., and Mozes, M. Abdominal actinomycosis. *Israel J. Med. Sc.*, 1972, *8*, 148–153.
2. Boijsen, E., and Reuter, S. R. Mesenteric angiography in evaluation of inflammatory and neoplastic disease of intestine. *Radiology*, 1966, *87*, 1028–1036.
3. Cope, Z. Actinomycosis. Oxford University Press, London, 1938.
4. Davies, M., and Keddie, N. C. Abdominal actinomycosis. *Brit. J. Surg.*, 1973, *60*, 18–22.
5. Lunderquist, A., Lunderquist, A., and Knutsson, H. Angiography in Crohn's disease of small bowel and colon. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 338–344.
6. Pinto, R. S., Zausner, J., and Beranbaum, E. R. Gastric tuberculosis: report of case with discussion of angiographic findings. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 808–812.
7. Piper, J. V., Stoner, B. A., Mitra, S. K., and Talerman, A. Ileovesical fistula associated with pelvic actinomycosis. *Brit. J. Clin. Pract.*, 1969, *23*, 341–343.
8. Reuter, S. R., and Redman, H. C. Gastrointestinal Angiography. W. B. Saunders Company, Philadelphia, 1972.

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PERITONEOGRAPHY*

DEMONSTRATION OF NORMAL AND ABNORMAL MURAL THICKNESS

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THE outstanding inherent limitation to conventional contrast studies of the alimentary tract is the inability to accurately evaluate the thickness of the bowel wall. Opacification of the lumen in upper gastrointestinal series and barium enema studies permits delineation of the mucosal pattern, but only indirect signs must be relied upon for the roentgen diagnosis of mural or serosal disease. It is apparent that definition of the wall of the bowel requires contrast on both its internal mucosal surface and its external serosal border. Peritoneography[4] is the double-contrast opacification of the peritoneal cavity, utilizing both gas and a water-soluble contrast medium. The contrast can be easily directed to appose the serosal surface of any portion of the bowel desired. When combined with a conventional intraluminal contrast study, the soft-tissue thickness and contour of the intervening bowel wall is clearly demonstrated. Furthermore, because of the renal excretion of the intraperitoneal water-soluble contrast medium, the mural thickness of the dome of the urinary bladder is precisely visualized.

REPORT OF CASES

CASE 1. A 44 year old white female was admitted because of hematemesis and tarry stools for 3 days, associated with intermittent epigastric pain radiating to the back. She had been taking increasing amounts of aspirin for intermittent headaches.

The patient, a chronic alcoholic, had been hospitalized 2 years previously for a similar episode. An upper gastrointestinal series then showed no abnormalities. Esophagogastroscopy demonstrated esophagitis and mild prominence of a single vein in the lower third of the esophagus on Valsalva maneuver. No abnormality was found in the stomach. Liver biopsy documented alcoholic hepatitis with early cirrhosis.

Physical examination revealed mild icterus but no hepatosplenomegaly. Spider telangiectasias were present on the chest and neck. Stool guaiac tests were 1+, but there was no blood in the gastric aspirate. Complete blood cell counts and serum amylase were within normal limits. Serum bilirubin was 1.9 mg. per cent.

An upper gastrointestinal series showed no evidence of esophageal varices (Fig. 1), but there was a lobulated mass in the fundus of the stomach above the esophagogastric junction (Fig. 2, *A* and *B*). Review of the study done 2 years previously disclosed no signs of a mass then. Endoscopy showed one small varix in the lower third of the esophagus. Examination of the stomach uncovered no abnormalities in the mucosa and no definite evidence of mass in the region of the gastric fundus. A repeat upper gastrointestinal series confirmed the presence of a mass. Because of the appearance of this over a 2 year interval and the confusing findings on esophagogastroscopy, the possibility that it was extrinsic to the stomach was considered.

Peritoneography was then performed simultaneously with the ingestion of barium (Fig. 3). This clearly documented localized thickening of the medial wall of the gastric fundus and established the lobulated mass to be intramural in nature. Varices of the gastric fundus were diagnosed and further substantiated by a splenoportogram (Fig. 4).

Comment. Unequivocal determination of the mass to be truly intramural and not extrinsic in nature was established by peritoneography. With synchronous barium opacification of the gastric lumen, the precise dimensions and contour of the localized thickening of the wall can be visualized.

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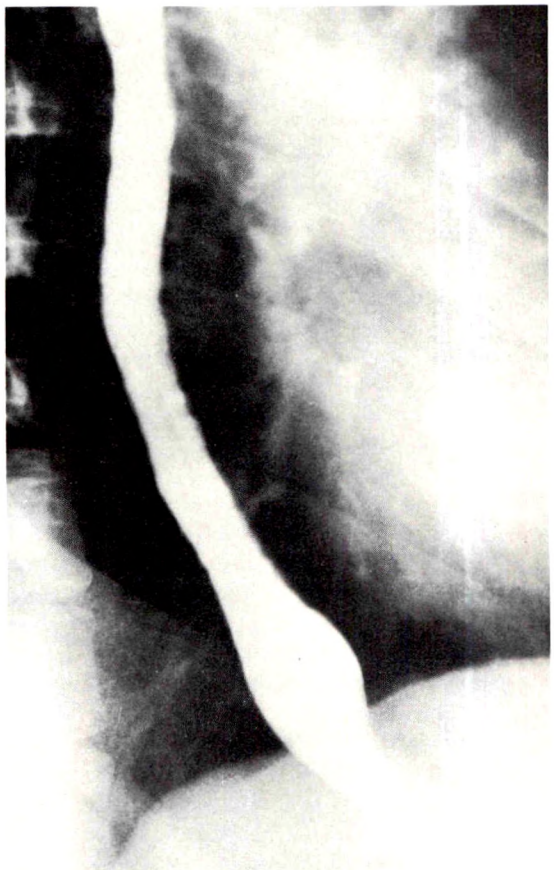


FIG. 1. Case I. Esophagogram. No evidence of esophageal varices.

CASE II. A 52 year old cirrhotic male was hospitalized because of weight loss. During his work-up, a liver scan showed several diffuse areas of decreased uptake suggesting the possibility of hepatic metastases. Peritoneoscopy demonstrated a cirrhotic liver without evidence of secondary deposits. No other definite abnormalities were visualized.

Peritoneography was performed immediately afterward, as a complement to the endoscopic study. The double-contrast examination (Fig. 5) disclosed an unsuspected mass thickening of the wall of the stomach along the distal pars media. It extended not only into the gastric lumen but into the gastrocolic ligament as well. A subsequent upper gastrointestinal series (Fig. 6) confirmed the intraluminal projections of an infiltrating carcinoma. At surgery, exten-

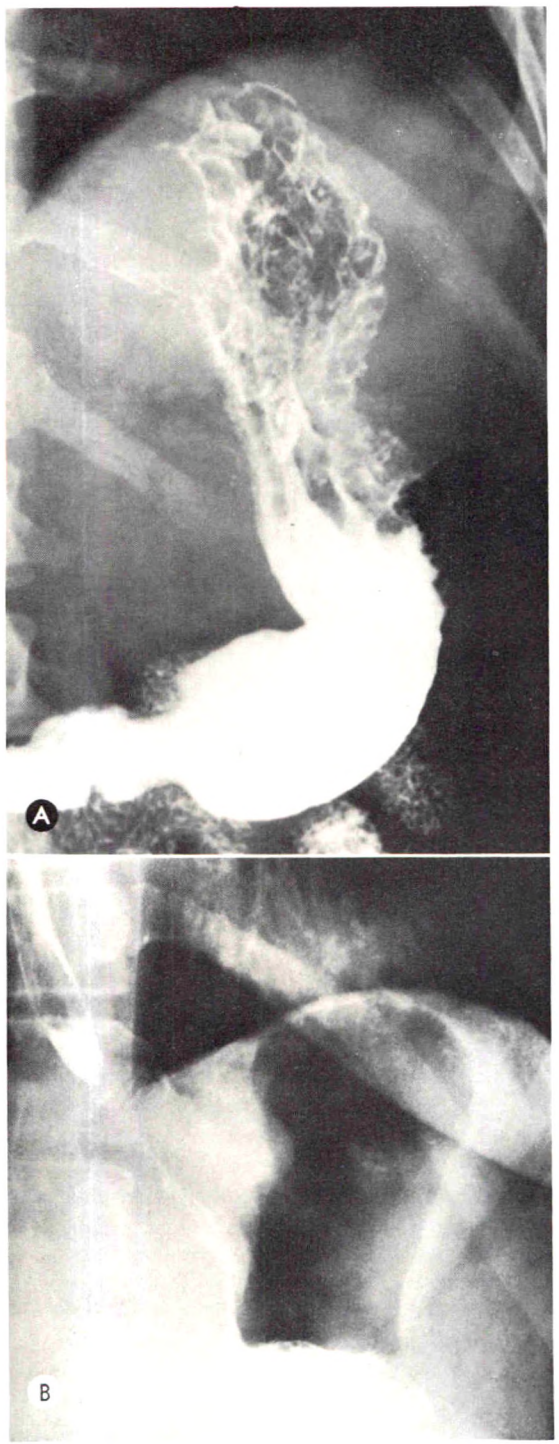


FIG. 2. Case I. (*A* and *B*) Upper gastrointestinal series. There is a lobulated mass along the medial wall of the gastric fundus above the esophagogastric junction.

sion of the mucosal lesion through the wall into the gastrocolic ligament was found.

Comment. This clinically unsuspected lesion was not evident during direct endoscopic visualization of the peritoneal cavity. Its presence was first detected by peritoneography which also demonstrated its full extent. The gastrointestinal series shows only its projection internally toward the gastric lumen, but cannot reveal its infiltration through the serosal aspect into the supporting mesenteries.

Case III. A 39 year old Egyptian farmer, known for many years to be *S. haematobium* and *S. mansoni* positive, was evaluated for diarrhea.

Peritoneography was combined with a conventional barium enema-air contrast study of the colon (Fig. 7). Diffuse colonic polyposis was demonstrated as typical intraluminal filling defects. In addition, however, marked thickening of the colonic wall to 10 mm. was clearly shown as a symmetrically widened soft-tissue density between the mucosal surface demarcated by the intraluminal air and the serosal surface outlined by the intraperitoneal positive contrast.

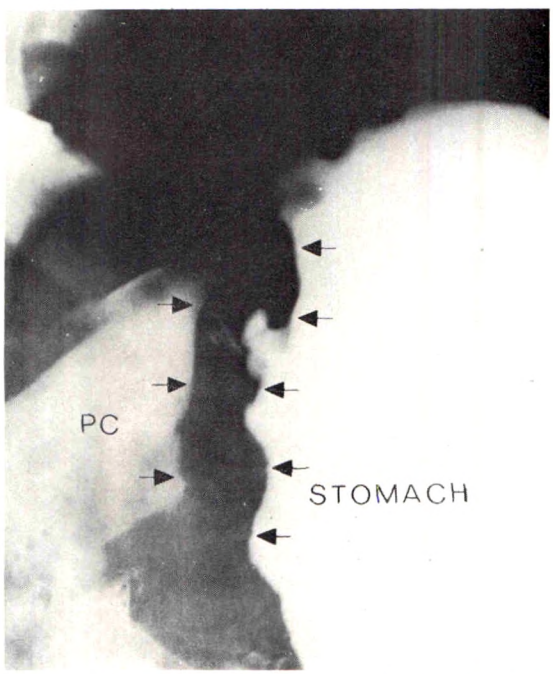


Fig. 3. Case 1. Peritoneogram, with simultaneous upper gastrointestinal series. Left posterior oblique (LPO) Trendelenburg projection. Peritoneal contrast (PC) on the serosal surface of the stomach demonstrates lobulated thickening of the gastric wall (arrows) and documents that the mass is intramural in nature.

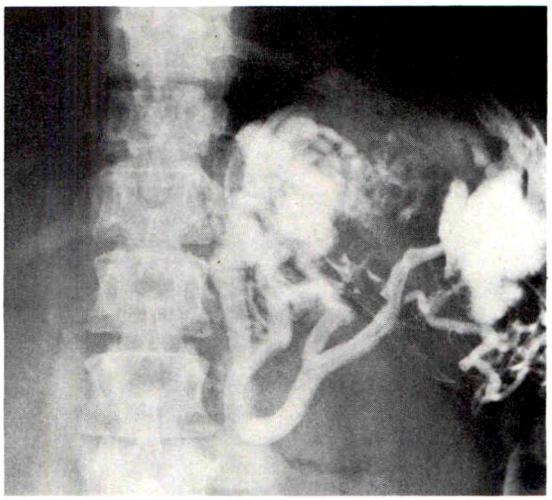


Fig. 4. Case 1. Splenoportogram shows opacification of the huge varices in the gastric fundus, but demonstrates no esophageal varices. A spontaneous spleno-renal shunt is present.

Comment. Significant thickening of the colon wall over a considerable length involved by polyposis is strikingly shown by peritoneography. In active schistosomiasis, this mural thickening indicates the progression from early exudative polypoid lesions to residual inactive intramural fibrosis. Normally, the colonic wall, as demonstrated by peritoneography (Fig. 8), is usually quite thin. A common exception is seen in cases of the prediverticular stage of sigmoid muscle hypertrophy. Primary mural and serosal disease or extension of mucosal lesions through the wall may be determined by peritoneography.

Case IV. A 48 year old Egyptian mason worker was evaluated for an 8 year history of dysuria with recent gross hematuria. Intravenous urography demonstrated multiple filling defects of the urinary bladder consistent with polyps. The patient was *S. haematobium* positive.

Peritoneography permitted, on delay films, evaluation of the urinary bladder and the adjacent peritoneal reflections and recesses. In addition to the multiple, large filling defects within the urinary bladder, the study clearly showed diffuse mural thickening of its dome

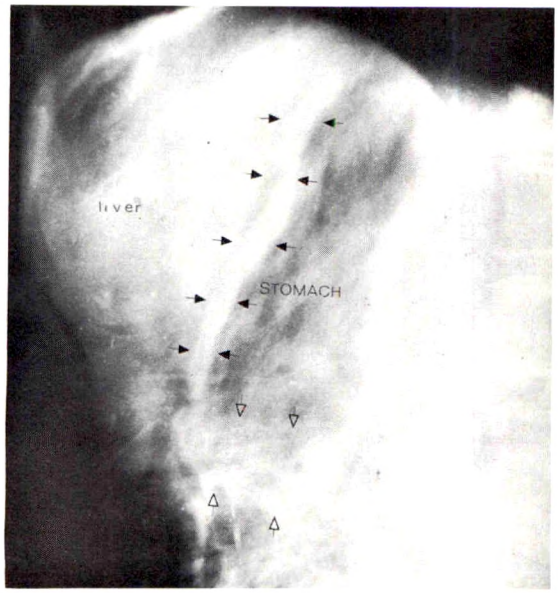


FIG. 5. Case II. Peritoneography. Right lateral projection. Contrast medium in the peritoneal cavity and normally occurring air within the lumen of the stomach discretely outline the normal symmetric thickness of the greater curvature (black arrows). Along the distal pars media (open arrows), the wall is disrupted and irregularly thickened by a mass which projects both into the lumen and externally into the region of the gastrocolic ligament.

(Fig. 9*A*) with infiltration of the adjacent paravesical fossa (Fig. 9*B*). The changes indicated a bladder carcinoma, of at least a Stage C nature. Surgery documented multiple carcinomas with intramural and perivesical extension associated with a few *S. haematobium* polyps of the bladder.

Comment. The absorption by the peritoneal surfaces of the water-soluble contrast medium used in peritoneography not only contributes to the safety of the procedure but, with renal excretion, provides opacification of the kidneys, ureters and urinary bladder. This occurs within 20 to 30 minutes. The intraperitoneal positive contrast medium gravitates to the dependent pelvis in the supine position, so that delayed films consistently demonstrate the thickness of the dome of the urinary bladder. Normally, it is smooth and symmetric, averaging 1–3 mm. in depth (Fig. 10*A*). Its dimensions vary somewhat with the degree of bladder distention and the extent of its curvature presenting parallel with the roentgen beam (Fig. 10*B*). The pelvic recesses of the peritoneal cavity consist of the central pouch of Douglas and the lateral paravesical fossae. These may be clearly seen in the supine projection but are often best visualized in the erect position (Fig. 11). Any disturbance of their symmetry or smooth margins indicates

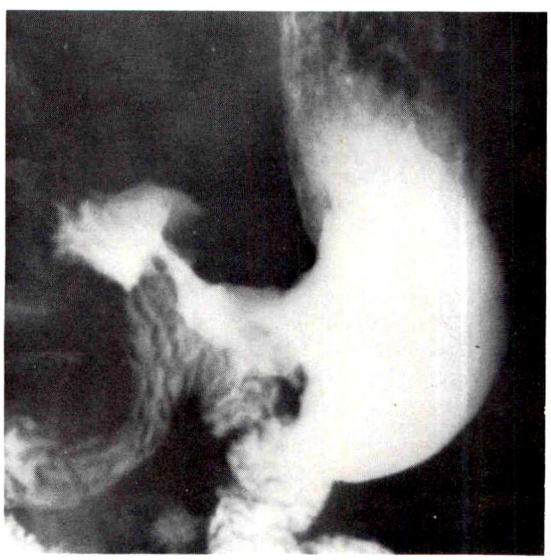


FIG. 6. Case II. Upper gastrointestinal series shows an infiltrating carcinoma of the distal body and antrum.



FIG. 7. Case III. Peritoneogram with barium enema-air contrast examination. Accompanying diffuse polyposis, marked thickening of the colonic wall is shown (arrows) by positive contrast apposing the serosal surface.

a definite abnormality. Their involvement, whether by mass displacement or actual invasion, as in Case IV, is useful in staging a bladder neoplasm.

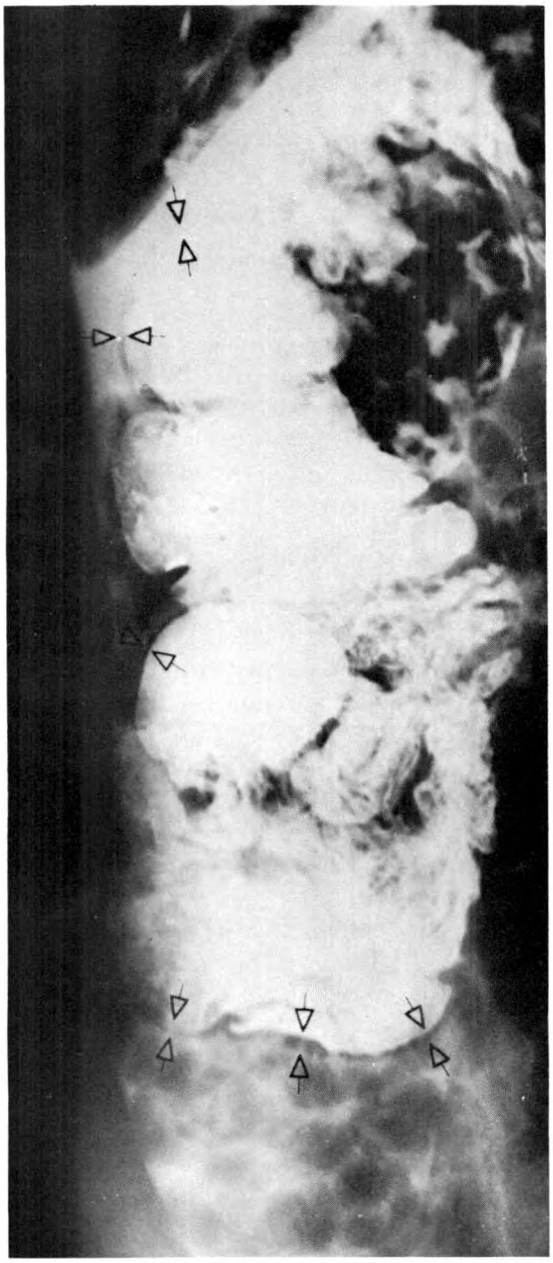


FIG. 8. Peritoneography with simultaneous barium enema study demonstrates the normal thickness and contour of the right colonic wall. The mural soft tissue stripe outlined between the mucosal and external surfaces (arrows) averages 1–2 mm. in thickness and symmetrically parallels the contour of the lumen.

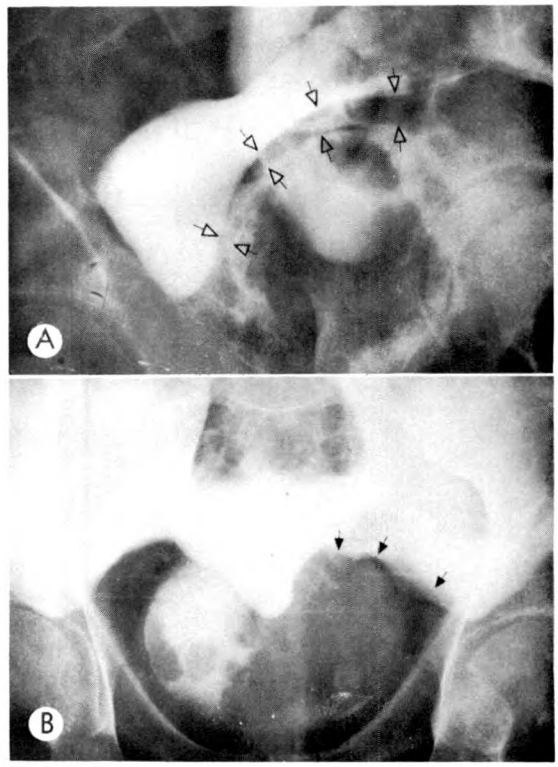


FIG. 9. Case IV. Peritoneography. With renal excretion of the contrast medium following its peritoneal absorption, the lumen of the urinary bladder is opacified. Remaining intraperitoneal contrast medium can be directed to the pelvic cavity. (*A*) Oblique roentgenogram shows markedly thickened wall (arrows) of dome of bladder which contains multiple filling defects. (*B*) Supine view demonstrates gross irregularity of the peritoneal reflection of the left paravesical fossa (arrows), indicating extensive infiltration. (Reproduced with permission of *Radiology*.)

DISCUSSION

Conventional barium studies of the alimentary tract most accurately diagnose lesions resulting in mucosal alterations or viscus displacement. The classic roentgen differential diagnosis between mucosal, submucosal (intramural) and extrinsic lesions is a distinction made on indirect signs only, based upon the contours of a defect. Terminology includes such descriptions as "mural defect," yet the actual wall of the structure studied is rarely visualized roentgenologically. Routine contrast studies permit direct roent-

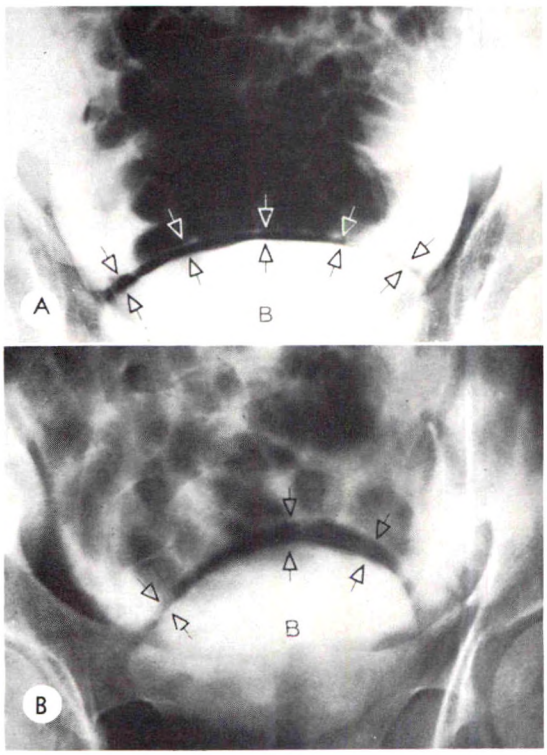


FIG. 10. (*A* and *B*) Peritoneography. Two examples of the normal thickness of the dome of the urinary bladder (B). Its normal range of 1–3 mm. is dependent upon the bladder's distention and contour relative to the roentgen beam.

genologic examination of the mucosal contour alone. Only occasionally, and then often by chance, can the wall of a structure be seen. On plain film roentgenograms in obese patients, rich serosal and omental fat may faintly outline the greater curvature of the stomach, the superior border of the transverse colon, and rarely small bowel loops in the right lower quadrant. Mural densities may be identifiable fortuitously during high dose urography as a phenomenon of total body opacification.[5] Arteriography may demonstrate the wall of a viscus as a mural blush during the capillary phase. Parietography has been used as a method of demonstrating the gastric wall by insufflation of oxygen into the peritoneal cavity with simultaneous inflation of the stomach by an effervescent powder, combined with tomography.[1]

Peritoneography extends the roentgenologic diagnostic capabilities in intraabdominal disease by providing a consistent detailed parameter of wall thickness, serosal contour, and transmural extension of disease. The presence of intraperitoneal gas enhances the positive contrast and compensates to some degree for the effect of gravity in positioning. Contrast medium may thus be easily and consistently apposed to the serosal surface of any viscus projecting within the peritoneal cavity. With the mucosal surface of the bowel outlined by swallowed air, introduced gas, or particularly conventional contrast studies, the mural dimensions are easily appreciated as the intervening soft-tissue density. The coincident urogram obtained is very useful to complement the opacification of the peritoneal cavity itself, and provides accurate assessment of the dome of the urinary bladder. The ability of peritoneography to diagnose occult lesions and to stage lesions preoperatively, otherwise often difficult,[2,3] is apparent.

SUMMARY

Conventional contrast studies of the alimentary tract do not directly demonstrate the wall.

Peritoneography, the intraperitoneal instillation of a water-soluble contrast medium and gas, consistently permits precise detailed evaluation of the mural thickness of the bowel and the dome of the urinary bladder. It extends roentgenologic diagnostic capabilities in intraabdominal disease in demonstrating occult lesions, the

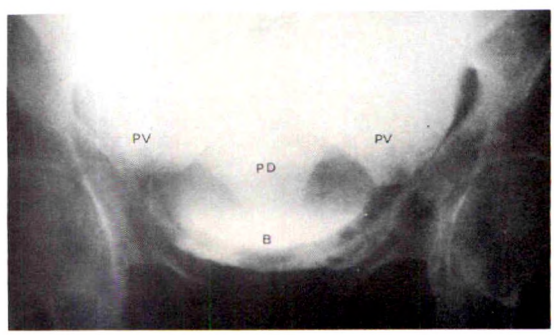


FIG. 11. Peritoneography, erect view. The urinary bladder (B) is opacified. Intraperitoneal contrast medium distends the mid-line pouch of Douglas (PD) and the lateral paravesical fossae (PV).

extent of disease states, and in contributing to accurate preoperative staging of a neoplasm.

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REFERENCES

1. Frimann-Dahl, J., and Traetteberg, K. Parietography of stomach. *Brit. J. Radiol.*, 1962, *35*, 249–254.
2. Marshall, V. F. Relation of preoperative estimate to pathologic demonstration of extent of vesical neoplasms. *J. Urol.*, 1952, *68*, 714–723.
3. Marshall, V. F. Current clinical problems regarding bladder tumors. *Cancer*, 1956, *9*, 543–550.
4. Meyers, M. A. Peritoneography: normal and pathologic anatomy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 353–365.
5. O'Connor, J. F., and Neuhauser, E. B. D. Total body opacification in conventional and high dose intravenous urography in infancy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 63–71.

THE MANY FACETS OF SCLEROSING FIBROSIS

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THE concept of sclerosing fibrosis as an idiopathic histopathologic entity is well established in the medical literature. Depending on its location in various parts of the body, it has been variously described as retroperitoneal fibrosis, sclerosing mediastinitis, sclerosing cholangitis, Riedel's thyroiditis and orbital pseudotumor, to mention the most common examples. More recently there have been several well documented reports of 2 or more of the above mentioned conditions observed in the same patient, either simultaneously or developing at a later date.[2,5,17] This evidence strongly supports the notion that these conditions represent manifestations of a common process having perhaps a systemic basis. The term "multifocal fibrosclerosis"[5] appears to be an appropriate designation for cases of multiple organ involvement.

In the radiologic literature excellent reports of the individual entities mentioned above are numerous; however, attempts to unite them into a common concept have been considerably less frequent.

The following selected cases reflect the most common aspects of multifocal fibrosclerosis.

REPORT OF CASES

CASE 1. This 48 year old man had a history of mild hypertension, a nonfunctioning right kidney, and faint visualization of a partially obstructed left kidney seen elsewhere on intravenous pyelography (IVP) 1 year before admission. Intermittent complaints of low back pain and bilateral flank pain were elicited at that time. Surgery was refused by the patient.

In the year preceding this admission, intermittent flank pain remained a problem. Repeat IVP, followed by retrograde pyelography showed marked dilatation of the left kidney collecting system with dilatation of the left ureter down to its midportion (Fig. 1; and 2).

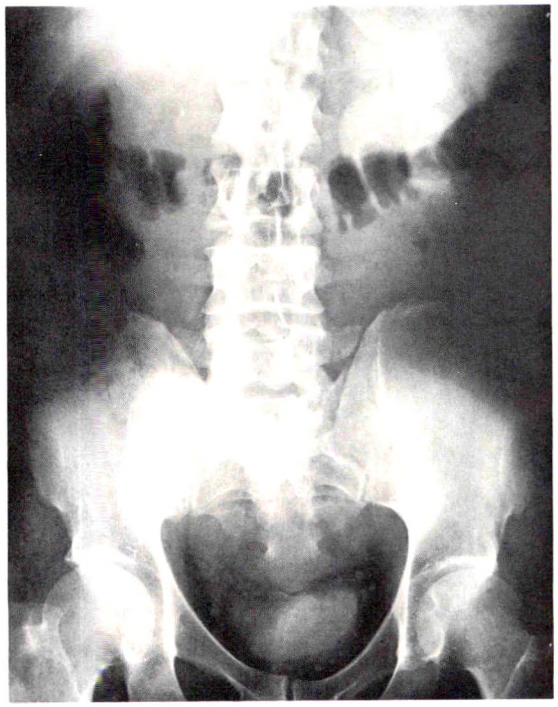


FIG. 1. Case 1. Intravenous pyelogram reveals a nonfunctioning right kidney and faint visualization of a partially obstructed left kidney.

Renal arteriogram showed a normal left renal artery. Some increased size of the upper pole of the left kidney was noted. The right kidney was small. He had a blood pressure of 164/96 mm. Hg, pyuria, blood urea nitrogen (BUN) 60 mg. per cent, and creatinine 7.0 mg. per cent.

The patient underwent a left kidney exploration at which time the left ureter was found to be surrounded by dense scar tissue starting below the uretero-pelvic junction for a distance of 4 inches. The ureter was successfully released from this tissue using blunt dissection and a nephrostomy tube was placed (Fig. 3).

Biopsy showed a process of hyalinizing fibrosis with sparse mononuclear inflammatory cells involving the fibroadipose tissue, but not the ureteral wall proper (Fig. 4). Biopsy of the left kidney revealed tubular hydronephrosis and acute pyelonephritis.

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The patient's clinical status and renal function improved steadily and he is asymptomatic at present, $2\frac{1}{2}$ years after surgery (Fig. 5).

CASE II. A 32 year old white male had a history of intermittent jaundice, occasional right upper quadrant pain, and slowly progressive obstruction type of liver disease for the past 7 years. An exploratory laparotomy performed early in the course of his illness revealed a white-pink firm mass, $5 \times 3 \times 2$ cm., sharply delineated, obscuring the common bile duct. This mass was incised and the exposed common bile duct was found to be rigid and thick-walled. No other pathology was found in the remainder

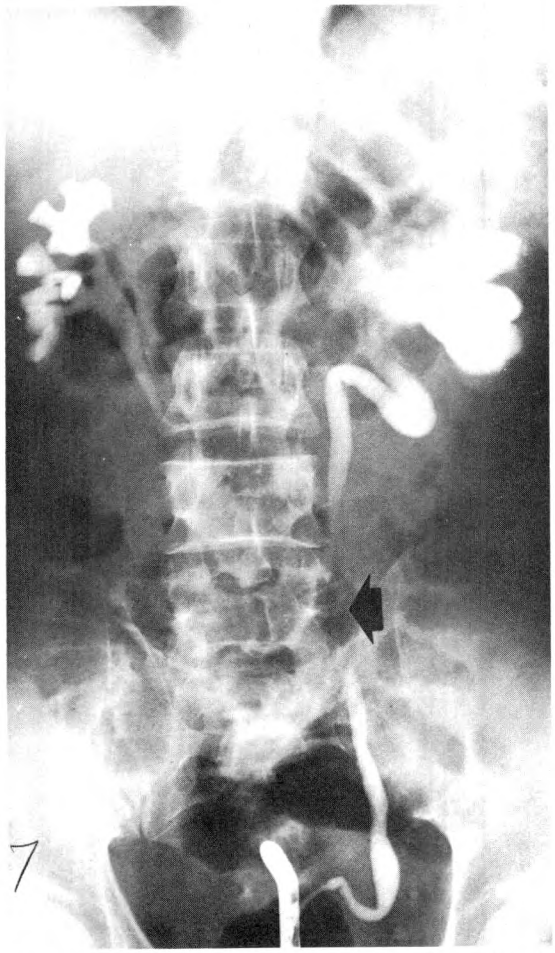


FIG. 2. Case 1. Retrograde pyelogram, immediately following intravenous pyelography, shows a small right kidney and a dilated partially obstructed left kidney with a narrowed mid third of the left ureter retracted medially (arrow).

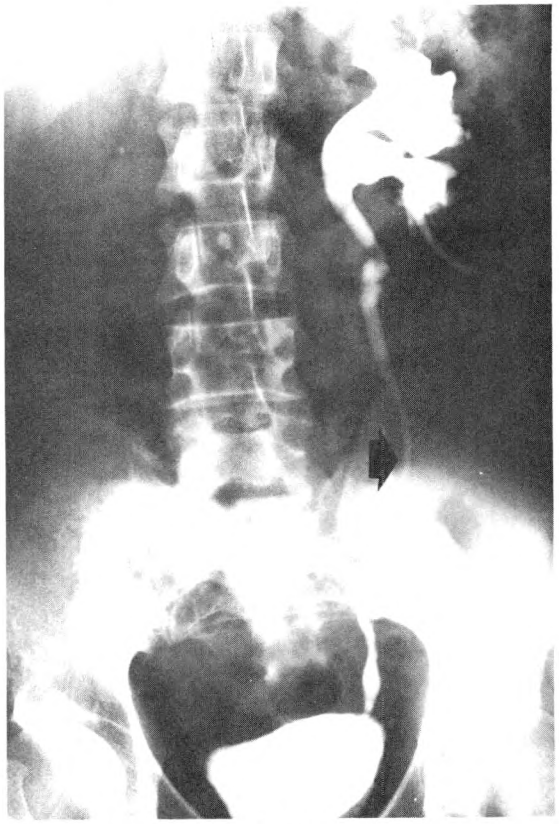


FIG. 3. Case 1. At operation, a nephrostomy tube was inserted in the lower pole dilated left collecting system. The left ureter was released from its encasing fibrosis (arrow).



FIG. 4. Case 1. Typical appearance of sclerosing fibrosis, involving retroperitoneal tissues surrounding the ureter. Notice diffuse pattern of ribbon-like areas of hyalinization with a scattering of inflammatory cells, mostly lymphocytes (H & E, 100×).

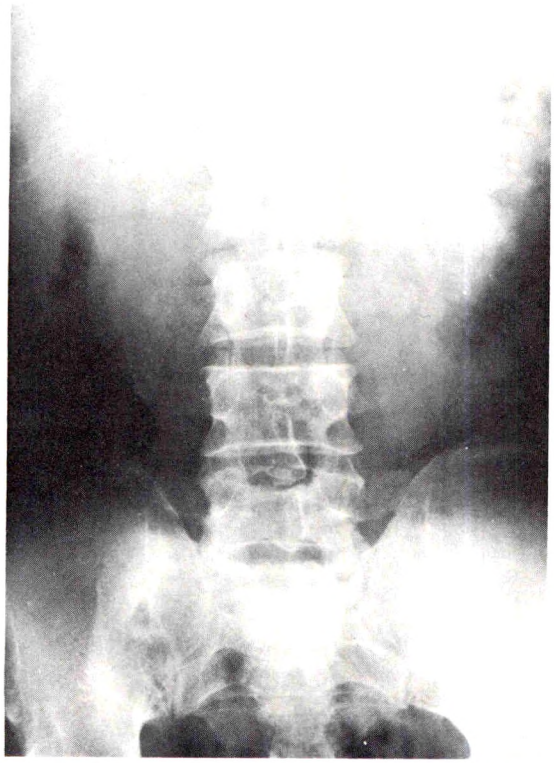


FIG. 5. Case I. Two and a half years after the initial intravenous pyelography better function and less obstruction were seen in the kidney.

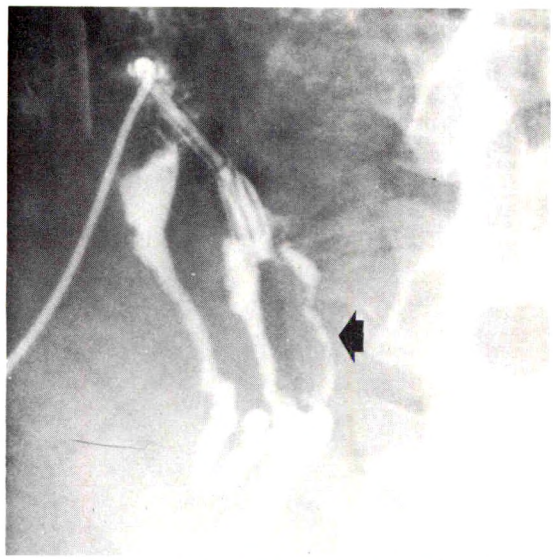


FIG. 6. Case II. The common duct (arrow) is very narrow, seen by filling from a tube placed in the proximal common duct at operation.

of the biliary system, and there were no gallstones.

Cholangiography during the procedure and postoperatively through a T-tube, revealed marked narrowing of the segments of the common duct and almost complete involvement of the intrahepatic biliary tree (Fig. 6; and 7). After placement of a T-tube, there was marked decrease in jaundice and obstructive liver chemistries.

On a subsequent admission the same year a cholecystojejunostomy was performed because of recurrent biliary obstruction. Percutaneous liver biopsies spanning the interval from 1966 to October 1973, showed a steadily progressive picture of cholangitis, pericholangitis and bile stasis, resulting in frank biliary cirrhosis, consistent with extrahepatic biliary obstruction. Biopsy of the common bile duct in 1966 demonstrated a process of dense transmural fibrosis with chronic inflammatory reaction (Fig. 8).

The patient did reasonably well during the intervening 6 years with occasional episodes of

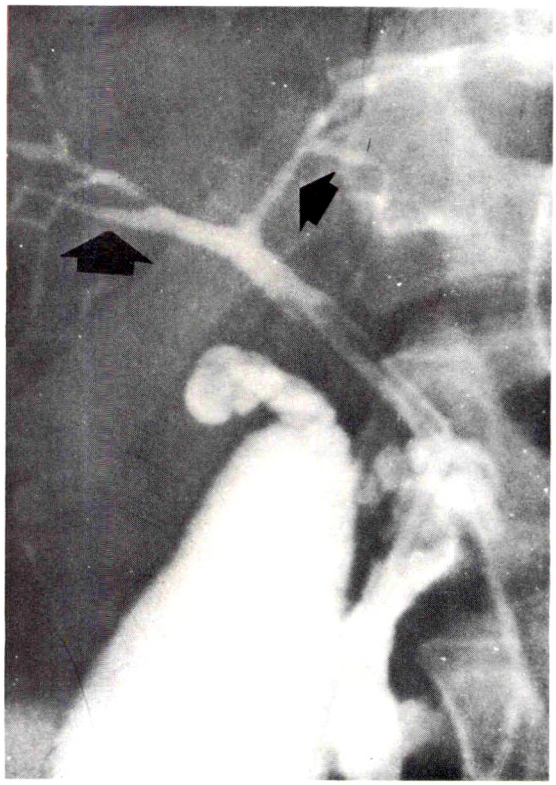


FIG. 7. Case II. T-tube cholangiogram shows very marked narrowing of the right and left hepatic ducts and intrahepatic radicals (arrows).

jaundice but generally in good health. For the last 3 years he has been maintained on small doses of Prednisone. In the past year the patient developed symptoms consistent with mild ulcerative colitis for which he is being treated supportively.

Case III. This 40 year old Black woman was admitted to St. Luke's Hospital Center in 1965 for hemoptysis. She gave a history of recurrent episodes of epistaxis and hemoptysis for 8 years, initiated by bouts of respiratory infections. Her past medical history up to that point had been entirely negative.

On several previous admissions, numerous laboratory tests were negative for any systemic or infectious disease. Cardiopulmonary studies disclosed no bronchial or pulmonary parenchymal disease, but demonstrated mild elevation of the pulmonary arterial pressure. Pulmonary arteriogram revealed a normal pulmonary arterial system; however, the venous phase of the pulmonary arteriography suggested large pulmonary veins dilated just before their entrance into the left atrium (Fig. 9; and 10).

In 1964 she underwent exploratory right thoracotomy, but no significant pathology was discovered.

After repeated subsequent bouts of hemoptysis she developed an upper respiratory infection associated with massive hemoptysis which precipitated her death.

At autopsy there was an ill-defined rubbery to hard white fibrous mass, 4 cm. in maximum dimensions, surrounding the hilar region of each lung compressing and invading the pulmonary

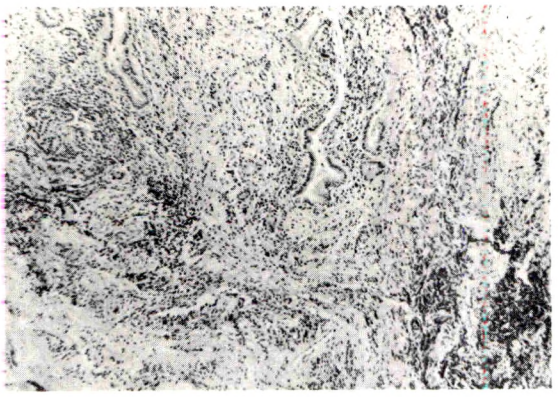


Fig. 8. Case II. Section of the common bile duct showing dense fibrosis and chronic inflammatory reaction involving the entire thickness of the wall (H & E, 100×).

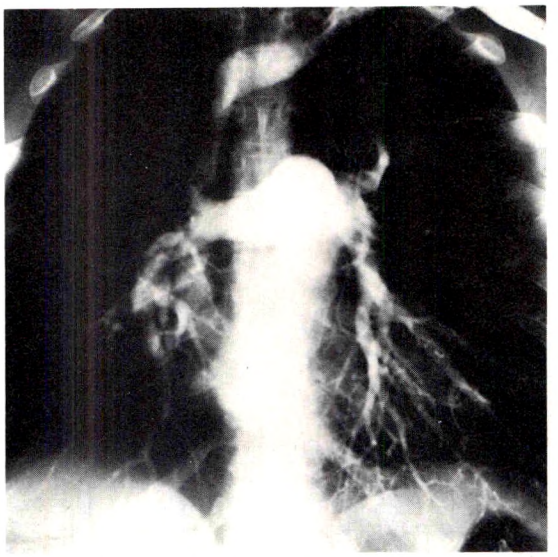


Fig. 9. Case III. Pulmonary arteriogram reveals a normal pulmonary arterial system.

veins and markedly reducing their lumina, particularly on the right side (Fig. 11). The bronchi and the primary branches of the pulmonary arteries were also surrounded by, and densely adherent to this tissue, but their lumina, although fixed, did not appear constricted. The entire right lower lobe was

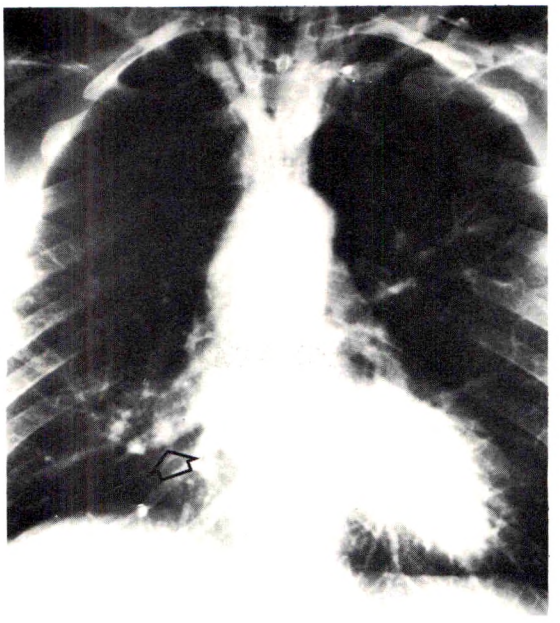


Fig. 10. Case III. Venous phase of the pulmonary arteriography suggests large pulmonary veins at their entrance into the left atrium (arrow).



Fig. 11. Case III. Fibrotic mass in hilum of right lung extending into the pulmonary vein (black arrows). Hilar lymph nodes (clear arrow) are surrounded, but not involved by the sclerotic process.

hemorrhagic and necrotic and the bronchial tree contained viscid hemorrhagic secretions with a cast of what histologically proved to be infarcted lung parenchyma in the lumen of the right lower lobe bronchus.

Histologically, the hilar masses consisted of dense fibro-collagenous tissue with scattered infiltrates of leukocytes, both polymorphonuclear and mononuclear types. This process surrounded and infiltrated the wall of the major pulmonary veins (Fig. 12) and to a lesser extent, the bronchi and pulmonary arteries. It extended into the pulmonary parenchyma in the perihilar region and out into the mediastinal space, abutting on the esophageal wall. The hilar lymph nodes were spared and appeared unremarkable.

The intrinsic pulmonary vasculature showed marked venous dilatation and fibrosis as well as moderate arteriolar sclerosis, characteristic of prolonged severe obstruction to the pulmonary outflow. The remainder of the postmortem examination was unremarkable.

Case IV. This 60 year old patient came to the hospital with severe respiratory distress. Three months prior to this, the patient had had a thyroidectomy in another hospital. Following the operation the patient had been in an oxygen tent for 10 days with severe respiratory difficulty and stridor. After some improvement she was discharged, but continued to have respiratory difficulty and stridor.

History and physical examination were relatively unremarkable. Direct laryngoscopy and bronchoscopy of the larynx were normal. No paralysis, edema or tumor was noted. In the mid-tracheal region about the fifth ring there was a concentric area of fibrotic tissue causing marked stricture. The lumen was diminished approximately 75 per cent. No record of intratracheal intubation was obtained.

Tomographic studies of the trachea revealed a smooth tapered narrowing of the tracheal air column at the level of the second and third thoracic vertebrae. Surrounding the trachea at this level was a poorly defined soft tissue density extending bilaterally to the substernal area (Fig. 13).

The patient was taken to the operating room for removal of the stricture of the lower trachea. At operation, the strap muscles and surrounding tissues were bound down by considerable scar tissue. The fifth tracheal ring had undergone complete collapse and had disappeared and was replaced by a segment of scar tissue causing marked narrowing of the trachea at this point.

Biopsies revealed residual thyroid gland infiltrated by a relatively acellular collagenous stroma disrupting greatly atrophic follicles and forming wide interstitial septa (Fig. 14). Immediately surrounding the follicles there was a dense infiltrate of small lymphocytes without formation of germinal centers, which differentiated it from Hashimoto's thyroiditis. The fibrotic process extended beyond the thyroid gland and infiltrated the adjacent skeletal muscle (Fig. 15).

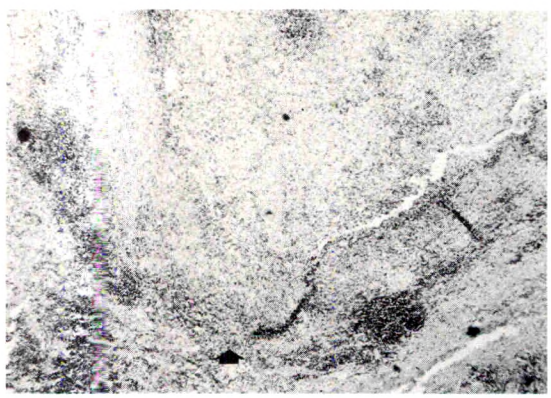


Fig. 12. Case III. Fibrosis with inflammatory cells is destroying the wall of the pulmonary vein (arrow), and is protruding into the lumen (H & E, 100×).

Biopsy of the constricted trachea at the level of the fifth ring showed infiltration of the wall by a similar process extending up to and causing ulceration of the mucosa (Fig. 16).

Case v. A 34 year old Black woman was admitted to St. Luke's Hospital Center for work-up of recurrent swelling of the right arm and face of 2 months' duration.

She was first seen by a physician for swelling of her right arm, which improved after discontinuing oral contraceptives, which the patient had been taking for the past 10 years. However, the swelling soon recurred, accompanied by cyanosis and she was admitted to another hospital where a diagnosis of right subclavian vein thrombosis was made by venography, and the patient was treated with anticoagulants.

Within the following month, she noted painful swelling of her neck and the right side of her face and in the week before her admission to St. Luke's Hospital Center she developed dyspnea, dysphagia, and abdominal cramps.

Her past history revealed that she had been taking amphetamines (desoxyn) for weight control for 10 years, but was otherwise negative for any illness.

Physical examination revealed mild right facial and arm swelling, marked bilateral cervical swelling with hard flat to nodular masses in the lateral cervical and supraclavicular region, originally interpreted as matted lymph nodes. In addition, there was a hard fixed mass over the right iliac area.

Roentgenogram of the chest showed a widening of the superior mediastinum from the clavi-

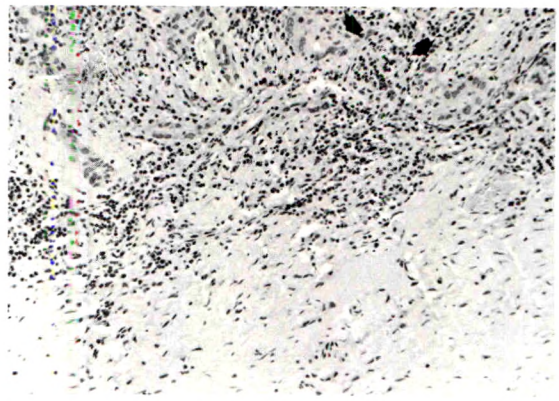


Fig. 14. Case iv. Sclerotic fibrous tissue and small lymphocytes extensively replace the thyroid parenchyma, leaving a few atrophic follicles present (H & E, 160×).

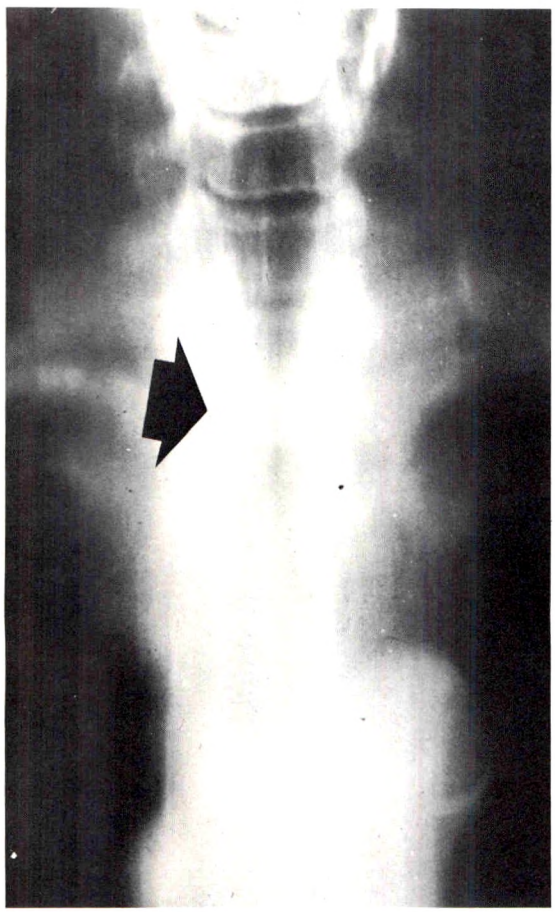


Fig. 13. Case iv. Tomogram of the trachea reveals a smooth tapered narrowing of the tracheal air column at the level of the mass (arrow).

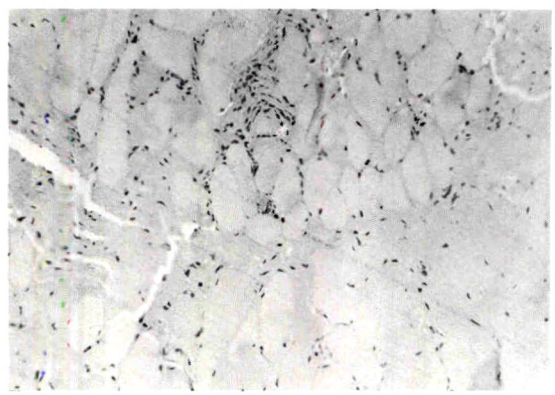


Fig. 15. Case iv. Skeletal muscle, adjacent to thyroid gland, dissected by hyalinized, relatively acellular fibrous tissue (H & E, 100×).

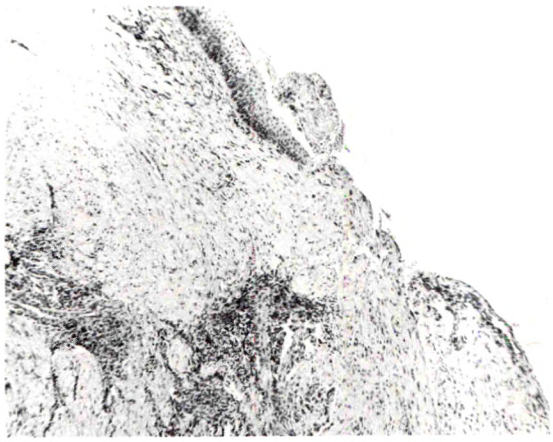


Fig. 16. Case iv. The process of fibrosis has permeated the wall of the trachea and caused ulceration of the overlying mucosa (H & E, 40×).

cle to the level of the hilum (Fig. 17). IVP was normal except for a 4×4 cm. mass in the right lumbar area, displacing the ureter medially (Fig. 18). All other laboratory tests were within normal limits.

Biopsies of the cervical and lower abdominal masses revealed identical histology, consisting of active fibroblastic cell growth, interspersed with acute and chronic inflammatory cells and with varying degrees of collagen fiber deposition (Fig. 19; and 20).

The patient was started on Prednisone, 60 mg. O.D.; within the next 10 days her cervical and abdominal masses receded dramatically and her mediastinal shadow disappeared on the chest roentgenogram. She was discharged symptom-free on steroid maintenance and has been followed since by a private physician. He reports that, although he was able to gradually taper her dose of Prednisone to 5 mg. without recurrence, when he stopped medications, there was prompt reappearance of the symptoms, necessitating re-institution of steroid therapy up to the present, 15 months after diagnosis.

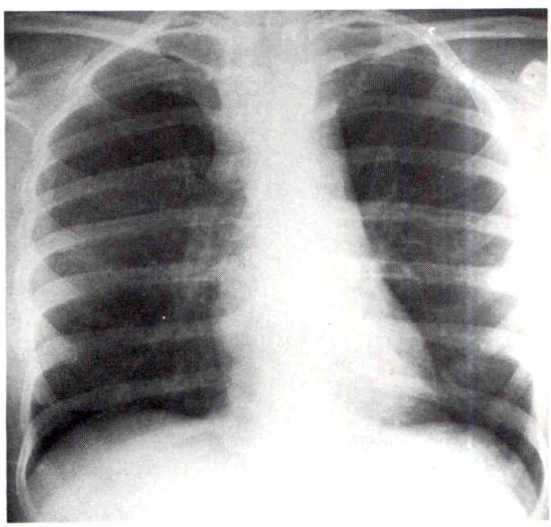


Fig. 17. Case v. Chest roentgenogram shows a right mediastinal mass from the clavicle to the level of the hilum.

DISCUSSION

RADIOLOGY

The radiologic presentation of multifocal fibrosclerosis depends upon the involvement of the organ systems present in the area and less often the effect of pressure or displacement by a new growth. Perhaps the most dramatic and potentially the most catastrophic syndrome commonly seen is

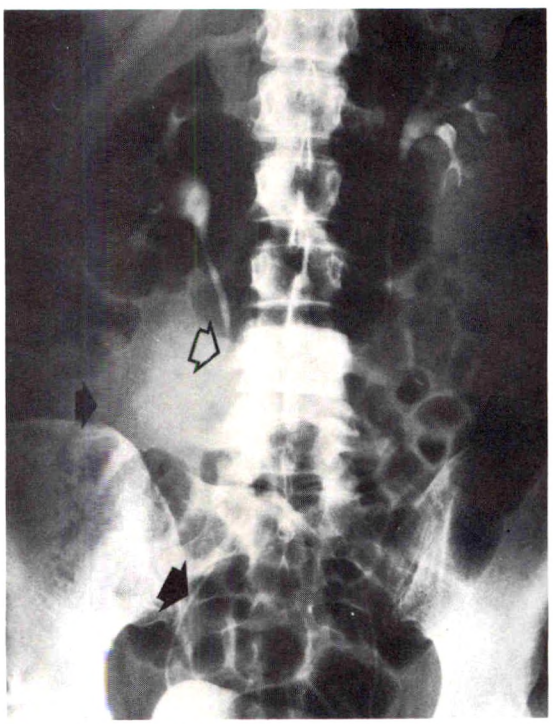


Fig. 18. Case v. Intravenous pyelogram shows a firm fixed mass in the right iliac area (upper solid arrow) with the mid third of the ureter displaced toward the mid line (open arrow). The lower one third of the right ureter is also slightly abnormal in its position, being more medial than normal as it reaches the level of the bladder (lower solid arrow).

the superior vena caval obstruction. The superior vena cava, because of its thin wall and low intraluminal pressure is most vulnerable to compression, distortion, and finally obstruction by fibrous tissue. It is interesting to note that obstruction of the superior vena cava first called attention to fibrous mediastinitis.[1,14,15] In both of our cases (Cases III and V) of mediastinal fibrosis, the superior vena caval obstruction was not prominent, although it is considered by some to be the characteristic finding of this entity. In Case III the major pulmonary veins were primarily and severely affected and in Case V extension into the subclavicular and lateral cervical regions with obstruction of the subclavian and jugular veins was dramatic. Chest roentgenogram commonly shows either hilar masses or mediastinal widening with occasional displacement or narrowing of the trachea.

When angiography is performed, occlusion of the superior vena cava shows marked collateralization in an effort to drain the upper extremities and jugular veins. The azygos system becomes prominent, if occlusion of the superior vena cava is above the entrance of the azygos system. If occlusion is below the entrance of the azygos system, then the hemiazygos system drains into the inferior vena cava.

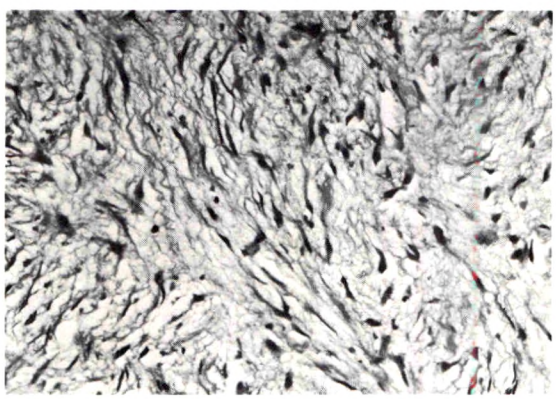


FIG. 19. Case V. Active spindle shaped and stellate fibroblasts in a loosely-textured collagenous stroma are the predominating elements in this patient's rapidly growing masses (H & E, 400×).

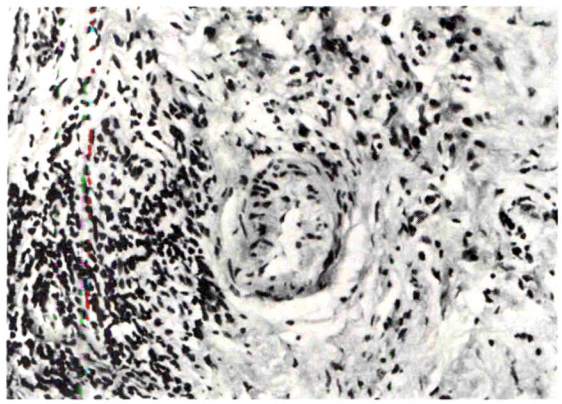


FIG. 20. Case V. Area of inflammatory cell infiltration, and an arteriole, which shows no evidence of vasculitis (H & E, 400×).

Marked collateralization is noted throughout. The radiologic picture takes on the classical appearance of malignancy in the chest, either primary or secondary. The proper diagnosis in this setting is extremely important because of the potential rapid response to appropriate therapy.

Stenosis of the pulmonary veins can be associated with idiopathic fibrosing mediastinitis.[3,10] Chest roentgenograms may reveal increase in pulmonary vascular markings often greater in the upper lobes. The hilar areas become prominent. Sudden episodes of hemoptysis associated with extreme dyspnea and cyanosis are characteristic. Hemoptysis often occurs during diagnostic procedures such as bronchoscopy or even angiography (Case III). In most patients reported, dyspnea and hemoptysis were striking manifestations. Death resulted in all cases, as in our case. The pathologic physiology of pulmonary vein obstruction is similar to that described in pure mitral stenosis. Angiography may reveal some irregular narrowing of the pulmonary arteries, but perhaps more striking is the dilatation of pulmonary veins at or just before their entrance into the left atrium.

When the retroperitoneal space is affected, the most striking radiologic finding is extrinsic ureter compression.[4,8,11,12,16,18–20]

Preliminary roentgenogram of the abdomen may show disappearance of the nor-

mal fat lines which outline the various retroperitoneal structures. This absence of fat planes may assist in identifying a mass of fibrous tissue. The radiologic diagnosis is strongly suggested by excretory urography. The ureters deviate medially often close to the uretero-pelvic junction and often pass straight downward close to the lateral margins of the lumbar vertebrae. When fibrosis extends into the pelvis, the ureters lose their usual lateral curvature. As the disease progresses, obstruction of the ureters occurs. The most common site of obstruction is at the level of the fourth and fifth lumbar vertebrae. The fibrosis can cover the aorta and vena cava down into the pelvis and often extends laterally to involve the bladder and rectum. It may extend to involve the common bile duct and the root of the mesentery, cause duodenal obstruction, and extend through the crura of the diaphragm as fibrous mediastinitis.

Pain is a common early symptom. As the disease progresses, involvement of the ureters and other structures such as bowel induce specific symptomatology. Kidney infection secondary to obstruction is common. Extension to involve the biliary system is referred to as sclerosing cholangitis and most often presents with jaundice due to diffuse thickening of duct walls producing a cord-like texture on palpation. The diffuse sclerosis may involve the entire duct system or may be limited to the common hepatic and common bile duct.[6,22]

Visualization of the ductal system is either accomplished preoperatively by percutaneous transhepatic cholangiography or T-tube cholangiography during surgery or postoperatively. The intrahepatic ducts may be smoothly narrowed or irregularly narrowed with intervening normal segments. The common duct may show diffuse narrowing of the lumen over short or longer segments with the remainder of the biliary tree relatively normal in contour and luminal diameter.

The clinical picture can be very similar to carcinoma of the pancreas, bile duct, ampullae or gallbladder. Differentiation from tumor in this area is difficult, but extremely important, since therapy is now available in sclerosing fibrosis. Involvement of specific localized areas or organs can be more confusing. Enlargement of the thyroid by fibrosis can mimic thyroiditis or carcinoma. Secondary involvement of the tracheal supporting cartilage may cause eventual necrosis and collapse with severe respiratory distress. Again this mimics the invasive destructive quality of malignant tumor.

As one reviews the radiologic and clinical picture of multifocal sclerosing fibrosis, it becomes apparent that the invasive, obstructive character of this process always suggests tumor. Because of the highly effective use of steroids in this condition, proper and early identification becomes crucial. Biopsy and/or exploration of the involved area are necessary for diagnosis.

PATHOLOGY

The gross appearance of sclerosing fibrosis is one of pale, scar-like tissue encasing and constricting the underlying hollow structures it involves, depending on its geographic location. Thus the trachea, the mediastinal large veins, the bronchi, and pulmonary veins, the esophagus, the common and hepatic bile ducts, the ureters and pelvic veins have most commonly been involved. The fibrosis may take the form of flat, plaque-like tissue, or nodular, circumscribed, tumor-like masses. Its consistency has always been described as stony hard or "wooden." It tends to spare the regional lymph nodes.

The histologic criteria for the diagnosis of multifocal fibrosclerosis are extremely simple, consisting of an overgrowth of fibrocollagenous stroma with varying degrees of fibroblastic activity and hyalinization, resulting occasionally in broad, keloid-like collagen bands. The fibrosis is virtually always connected with an infiltrate of inflammatory cells of all types, but predominantly lymphocytic. Blood vessels are relatively sparse and usually do not exhibit an inflammatory involvement. The relatively

bland picture of this lesion belies its infiltrative capacity which differentiates it from the mere constrictive effect of ordinary scar tissue.

The clinical picture of obstructive symptoms combined with a surgeon's operative report of ill-defined hard tissue mass or "frozen pelvis" is strongly suggestive of true malignancy from which this condition must be differentiated. The pathologist must be sure to search for the presence of malignant cells in the desmoplastic stroma and thus rule out a carcinoma, primary or metastatic, Hodgkin's disease and, depending on the location, a well differentiated fibrosarcoma. Finally, the condition should not be confused with fibromatosis, which presents a noninflammatory growth of fibroblastic and collagenous nature, and affects chiefly tendons and fasciae of the soft tissue.

ETIOLOGY

It must be stressed that the etiology and pathogenesis of this disease remain obscure.

Several reports have linked cases with infectious agents, notably *Histoplasma capsulatum*, particularly in the mediastinal location.[9,15]

Drug sensitivity appeared to be a plausible cause in a group of cases with a history of Sansert (methysergide maleate) ingestion for migraine headaches, reported by Graham[13] in 1966 and in subsequent sporadic reports.[21] Various autoimmune or collagen diseases, such as lupus erythematosus, periarteritis nodosa, rheumatoid arthritis, and Reynaud's phenomena have been the underlying diseases in some cases, —an association suggestive of a hypersensitivity or immunity disorder as a possible etiologic factor.[18,20] Finally, genetic factors have been implicated in a case report of multifocal fibrosclerosis occurring in the offspring of a consanguineous union.[5]

In one of our cases (Case II), the patient developed a mild ulcerative colitis 7 years after the onset of sclerosing cholangitis. In a series of 8 cases of sclerosing cholangitis,[6] 3 cases had ulcerative colitis—an incidence that indicates a possible pathogenetic relationship. The long history of amphetamine ingestion in 1 of our cases (Case V) was interesting, but it is considered unlikely to represent the cause of such a fulminating and rapidly progressing process as hers was. A search through the literature revealed no other such reports. There remain, however, a majority of reported cases in which no underlying inflammatory or other conditions exist.

SUMMARY

Five cases of sclerosing fibrosis, each affecting a different organ or system or a combination of systems, are described to illustrate the concept that these are different manifestations of a common process having perhaps a systemic etiologic basis.

We feel that the terms "sclerosing fibrosis" and "multifocal fibrosclerosis" are more appropriate designations than the various terms adopted in the past, which have very limited topographic application, *i.e.* "sclerosing mediastinitis," etc.

The characteristic pertinent radiologic and pathologic features are discussed.

Differentiation from malignant tumors and the importance of early diagnosis are stressed because of the effective therapeutic response to the use of steroids in this condition.

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REFERENCES

1. Barrett, N. P. Idiopathic mediastinal fibrosis. *Brit. J. Surg.*, 1958, *46*, 207–218.
2. Bartholomew, L. G., Cain, J. C., Woolner, L. B., Utz, D. C., and Ferris, D. P. Sclerosing cholangitis. *New England J. Med.*, 1963, *269*, 8–12.
3. Bindeglass, I. L., and Trubowitz, S. Pulmonary vein obstruction: uncommon sequel to chronic fibrous mediastinitis. *Ann. Int. Med.*, 1958, *48*, 876–891.
4. Buckberg, G. D. Protean manifestations of sclerosing fibrosis. *Surg., Gynec. & Obst.*, 1966, *123*, 729–736

5. Comings, D. E., Skubi, K. B., Van Eyes, J., and Motulski, A. G. Familial multifocal fibrosclerosis. *Ann. Int. Med.*, 1967, *66*, 884–892.
6. Cutler, B., and Donaldson, G. A. Primary sclerosing cholangitis and obliterative cholangitis. *Am. J. Surg.*, 1969, *117*, 502–511.
7. Dalinka, M. K., and McGee, J. W. Variable manifestations of sclerosing fibrosis. *J. Canad. A. Radiologists*, 1970, *21*, 280–286.
8. Dineen, J., Asch, T., and Pearce, J. M. Retroperitoneal fibrosis: anatomic radiologic review with report of three new cases and explanation of pathogenesis. *Radiology*, 1960, *75*, 380–390.
9. Dozois, R. R., Bernatz, P. E., Wollner, L. B., and Andersen, H. A. Sclerosing mediastinitis involving major bronchi. *Mayo Clin. Proc. Staff Meet.*, 1968, *43*, 557–569.
10. Edwards, J. E., and Burchell, H. E. Multilobar pulmonary venous obstruction with pulmonary hypertension. *Arch. Int. Med.*, 1951, *87*, 372–378.
11. Emmett, J. L., and Witten, D. M. Clinical Urography. Volume III. W. B. Saunders Company, Philadelphia, 1971, p. 1849.
12. Gleeson, M. H., Taylor, S., and Dowling, R. H. Multifocal fibrosclerosis. *Proc. Roy. Soc. Med.*, 1970, *63*, 1309–1311.
13. Graham, J. R. Fibrotic disorders associated with methysergide therapy for headache. *New England J. Med.*, 1966, *274*, 359–368.
14. Hache, L., Woolner, L. B., and Bernatz, P. B. Idiopathic fibrous mediastinitis. *J. Dis. Chest*, 1962, *41*, 9–25.
15. Kunkel, W. M., Clagett, O. T., and MacDonald, J. R. Mediastinal granulomas. *J. Thoracic & Cardiovasc. Surg.*, 1954, *27*, 565–574.
16. Millard, D. G., and Wyman, S. M. Periureteric fibrosis: radiographic diagnosis. *Radiology*, 1959, *72*, 191–196.
17. Mitchison, M. J. Pathology of retroperitoneal fibrosis. *J. Clin. Path.*, 1970, *23*, 681–689.
18. Morandi, L. P., and Grob, P. J. Retroperitoneal fibrosis. *A.M.A. Arch. Int. Med.*, 1971, *128*, 295–298.
19. Ormond, J. K. Bilateral ureteral obstruction due to involvement and compression by inflammatory retroperitoneal process. *J. Urol.*, 1948, *59*, 1072–1079.
20. Que, G. S., and Mandema, E. Case of idiopathic retroperitoneal fibrosis presenting as systemic collagen disease. *Am. J. Med.*, 1964, *36*, 320–329.
21. Utz, D. C., Rooke, E. D., Spithell, J. A., and Bartholomew, L. G. Retroperitoneal fibrosis in patient taking methysergide. *J.A.M.A.*, 1965, *191*, 983–985.
22. Wenger, J., Gingrich, G. W., and Mendeloff, J. Sclerosing cholangitis: manifestation of systemic disease. *A.M.A. Arch. Int. Med.*, 1965, *116*, 509–514.

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INFUSION TOMOGRAPHY OF THE FEMALE PELVIS*

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SINCE the original description of total body opacification in infants by O'Connor and Neuhauser,[8] this phenomenon has been utilized diagnostically in ovarian cysts in infants,[3] uterine fibroids,[2] gallbladder disease,[9] adrenal cysts,[6] and other conditions as well.

Martin *et al.*[7] describe the radiologic phenomenon of total body opacification following intravenous injection as a condition in which contrast medium opacifies the structures of the body in proportion to their blood supply. Cattell[4] has shown that this occurs because a peak plasma level is obtained almost immediately; it then rapidly declines representing the initial mixing of the contrast medium in the vascular compartment. A further fall in plasma level occurs which is dependent upon equilibration of the contrast medium in its body fluid space, and concomitant excretion of the compound via glomerular filtration.

Intravenous urography is often used prior to gynecologic surgery, in order to determine the presence of renal anomaly or disease. Simultaneous roentgenographic study of the pelvic inlet offers additional information about an ovarian or uterine mass. Before involution and necrosis, uterine fibroids consist of a mass of proliferating arteries with few, if any, veins within the tumor. The capillary network and interstitial space are profuse with drainage *via* veins around the periphery.[5] Birnholz[2] suggests that passive interstitial accumulation of the contrast material accounts for the opacification of leiomyomata. Visualization of cyst walls within ovarian lesions most likely represents vascular pooling of contrast medium within the wall itself. The avascularity of the cyst content accounts for its radiolucency. The use of large amounts of contrast medium by infusion technique combined with tomography of the pelvic inlet can in many instances offer significant information relative to the nature of a pelvic tumor.

TECHNIQUE

We have found the infusion technique to be superior to a bolus intravenous injection as it prolongs the vascular phase of the metabolism of the contrast medium, thereby delaying the period of declining plasma levels and allowing greater time for effective tomographic study.

The suggested technique is:

1. Preliminary anteroposterior roentgenograms of the abdomen (14×17 inch size).
2. Intravenous infusion of 300 cc. (adult) sterile aqueous solution of 30 per cent meglumine diatrizoate which contains 14 per cent (42.3 grams per 300 cc.) bound iodine.* Contrast material is delivered in approximately 7–10 minutes.
3. After completion of infusion, the following are obtained:
   (a) Abdomen, anteroposterior 14×17 inch roentgenograms to evaluate kidneys and pelvic inlet.
   (b) Midline pelvis zonogram, reviewed by radiologist who requests additional cuts at 1 cm. intervals to evaluate density in question.

Contraindications to this procedure are a

* Eeno-M-Dip (TM) Squibb.

* Presented as a Scientific Exhibit at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
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history of serious reaction to contrast medium, or known pregnancy.

RADIATION EXPOSURE

Radiation exposure is a definite consideration in tomography of the female pelvis. Table 1 illustrates the absorbed gonadal dose per tomogram at the specified conditions.

The average acceptable gonadal absorbed dose for radiation workers per year is 5,000 millirems.[1] Any patient may, of course, exceed these recommendations if the need exists.

MATERIAL

Figures 1 to 9 illustrate 9 cases representing examples of this technique.

SUMMARY

A method of simultaneous study of the urinary system and female pelvic tumors by the infusion technique and tomography of the pelvic inlet is presented.

Examples of uterine fibroids, ovarian cysts and tumors, and sacral meningocele, as well as an unexpected demonstration of pregnancy, are shown.

Table 1
RADIATION CONSIDERATIONS

	66 kv.	70 kv.
Absorbed gonadal dose with ovaries 11.5 cm. deep	293 millirems	352 millirems
Absorbed gonadal dose with ovaries 10 cm. deep	388 millirems	465 millirems

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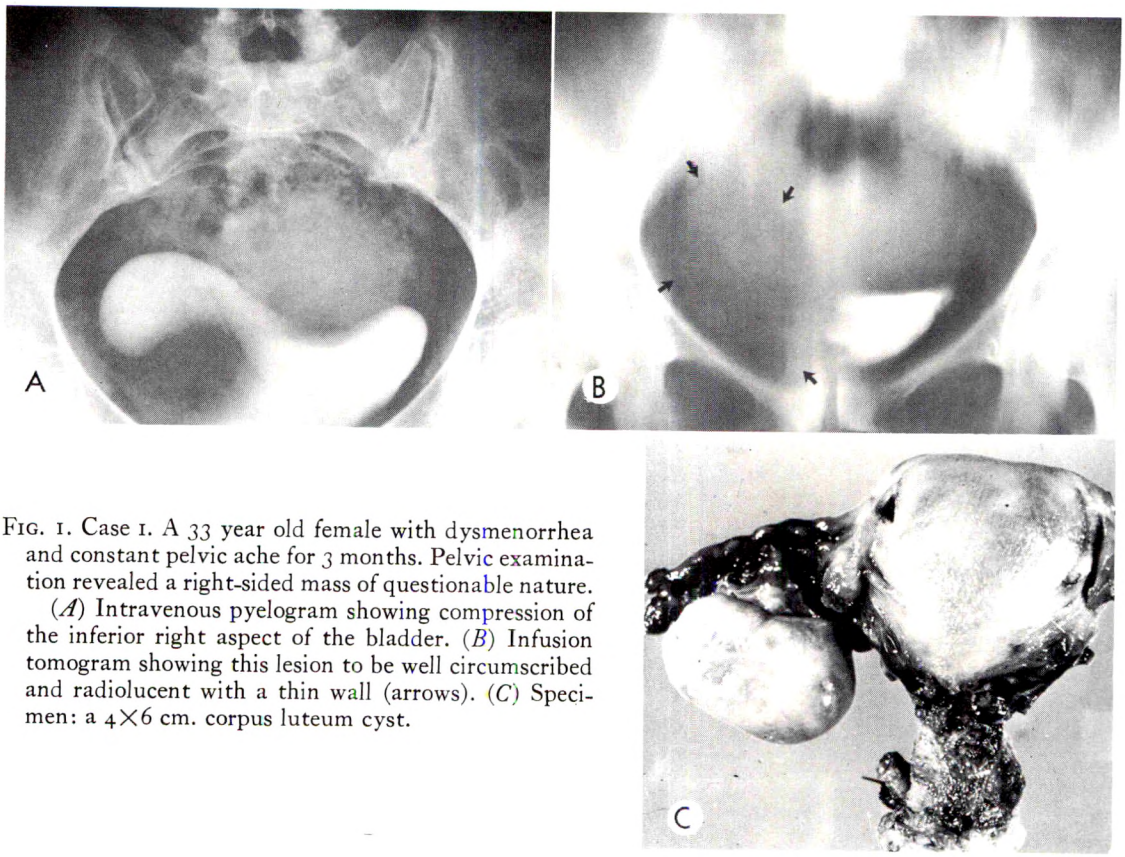


Fig. 1. Case 1. A 33 year old female with dysmenorrhea and constant pelvic ache for 3 months. Pelvic examination revealed a right-sided mass of questionable nature. (*A*) Intravenous pyelogram showing compression of the inferior right aspect of the bladder. (*B*) Infusion tomogram showing this lesion to be well circumscribed and radiolucent with a thin wall (arrows). (*C*) Specimen: a 4×6 cm. corpus luteum cyst.

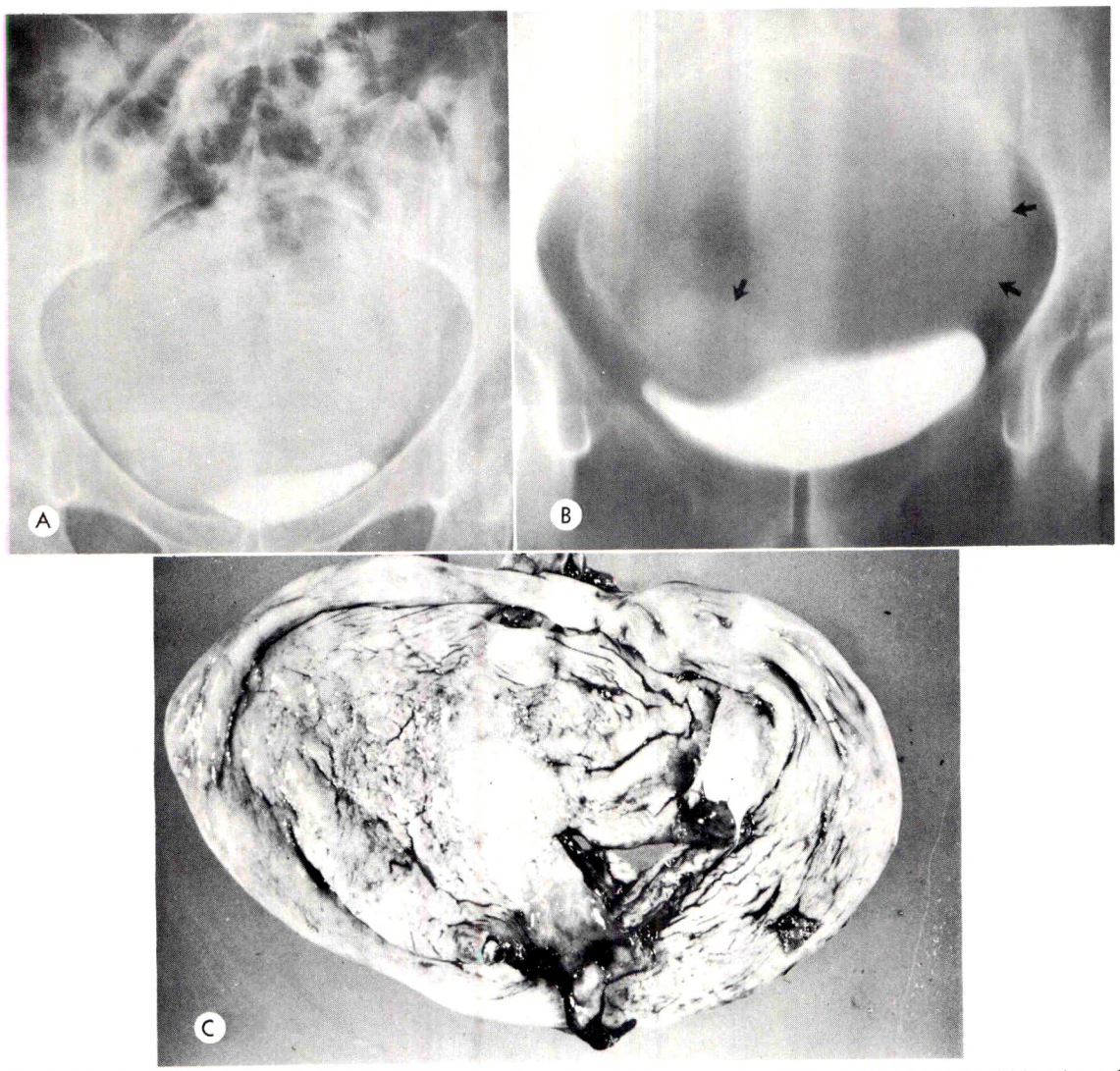


Fig. 2. Case 11. A 54 year old female who was 5 years postmenopausal and presented with vaginal bleeding of 6 weeks' duration.

(*A*) Intravenous pyelogram reveals a solid appearing mass which compresses the superior aspect of the bladder. (*B*) Infusion tomogram demonstrates a smooth walled cystic mass on the left (double arrows) compressing the uterus to the right (single arrow). (*C*) Specimen: a 580 gram ovarian cyst measuring $12 \times 8\frac{1}{2} \times 8\frac{1}{2}$ cm. The ovary is unilocular with a smooth inner lining and contains 500 cc. of clear yellow fluid.

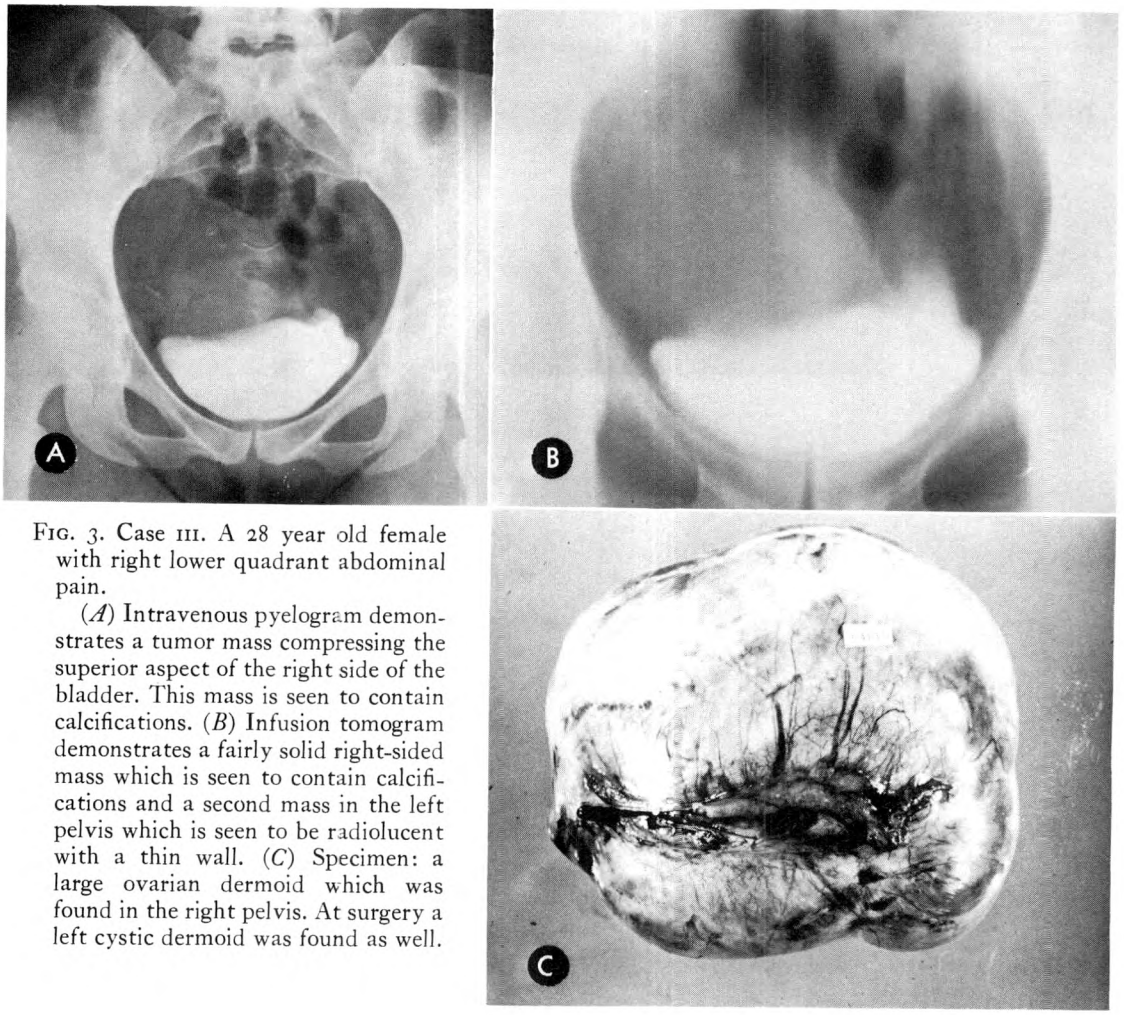


Fig. 3. Case III. A 28 year old female with right lower quadrant abdominal pain.

(*A*) Intravenous pyelogram demonstrates a tumor mass compressing the superior aspect of the right side of the bladder. This mass is seen to contain calcifications. (*B*) Infusion tomogram demonstrates a fairly solid right-sided mass which is seen to contain calcifications and a second mass in the left pelvis which is seen to be radiolucent with a thin wall. (*C*) Specimen: a large ovarian dermoid which was found in the right pelvis. At surgery a left cystic dermoid was found as well.

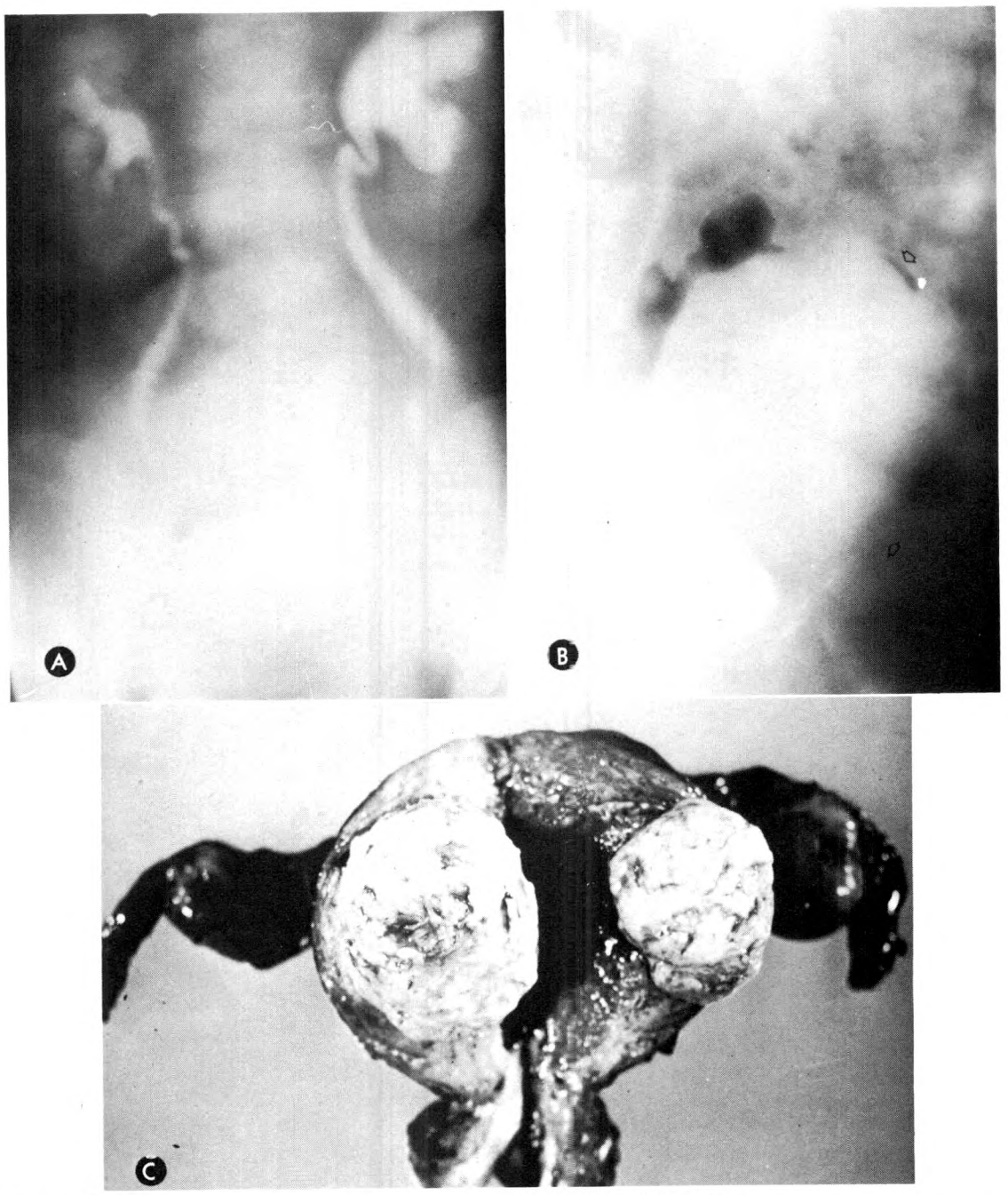


Fig. 4. Case IV. A 34 year old female with a gradually enlarging pelvic mass.
(*A* and *B*) Infusion tomograms demonstrate a solid midline mass arising from the pelvis. (*C*) Specimen: an 8 weeks' gestation size uterus containing multiple fibroids.

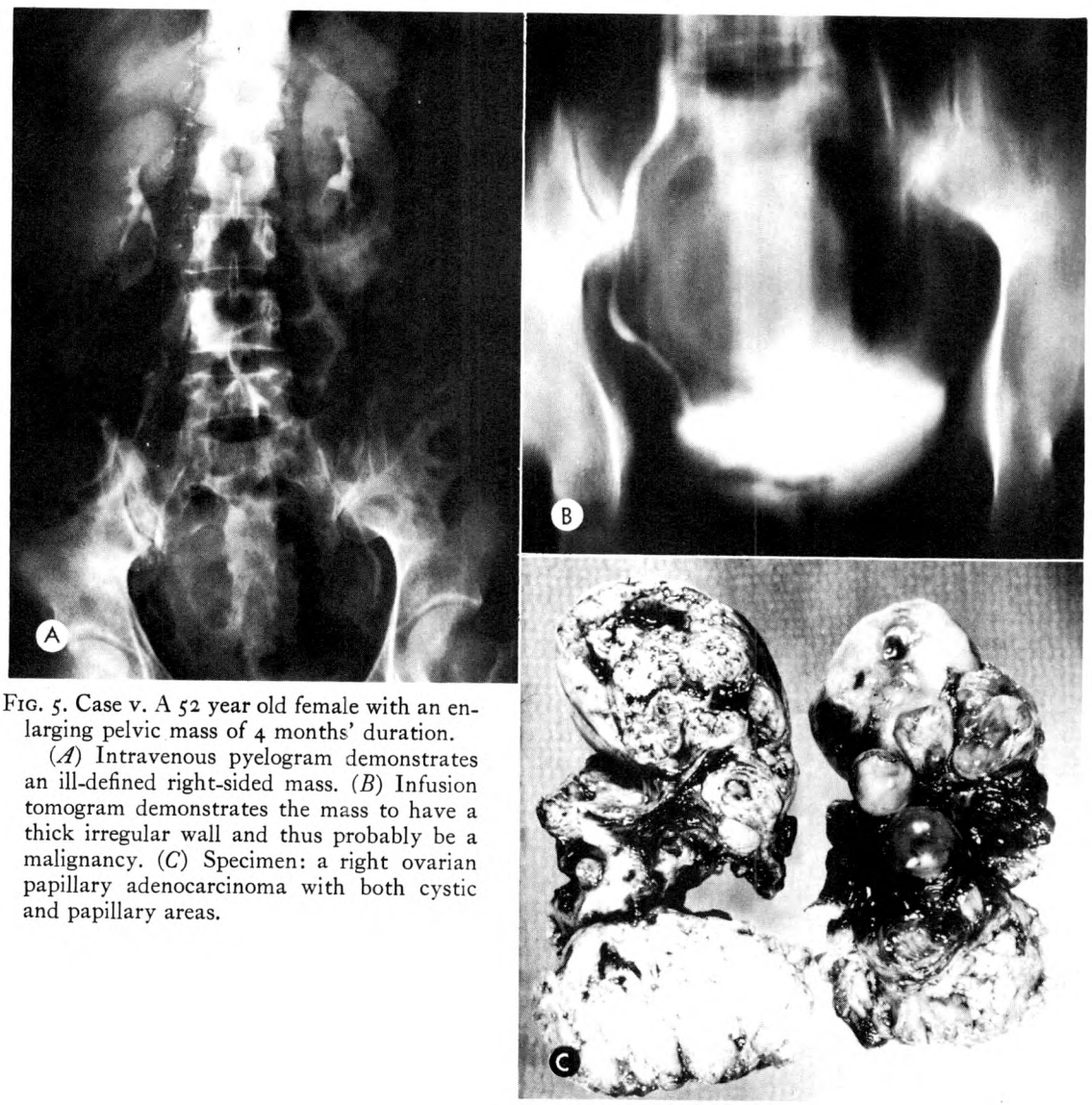


Fig. 5. Case v. A 52 year old female with an enlarging pelvic mass of 4 months' duration.

(*A*) Intravenous pyelogram demonstrates an ill-defined right-sided mass. (*B*) Infusion tomogram demonstrates the mass to have a thick irregular wall and thus probably be a malignancy. (*C*) Specimen: a right ovarian papillary adenocarcinoma with both cystic and papillary areas.

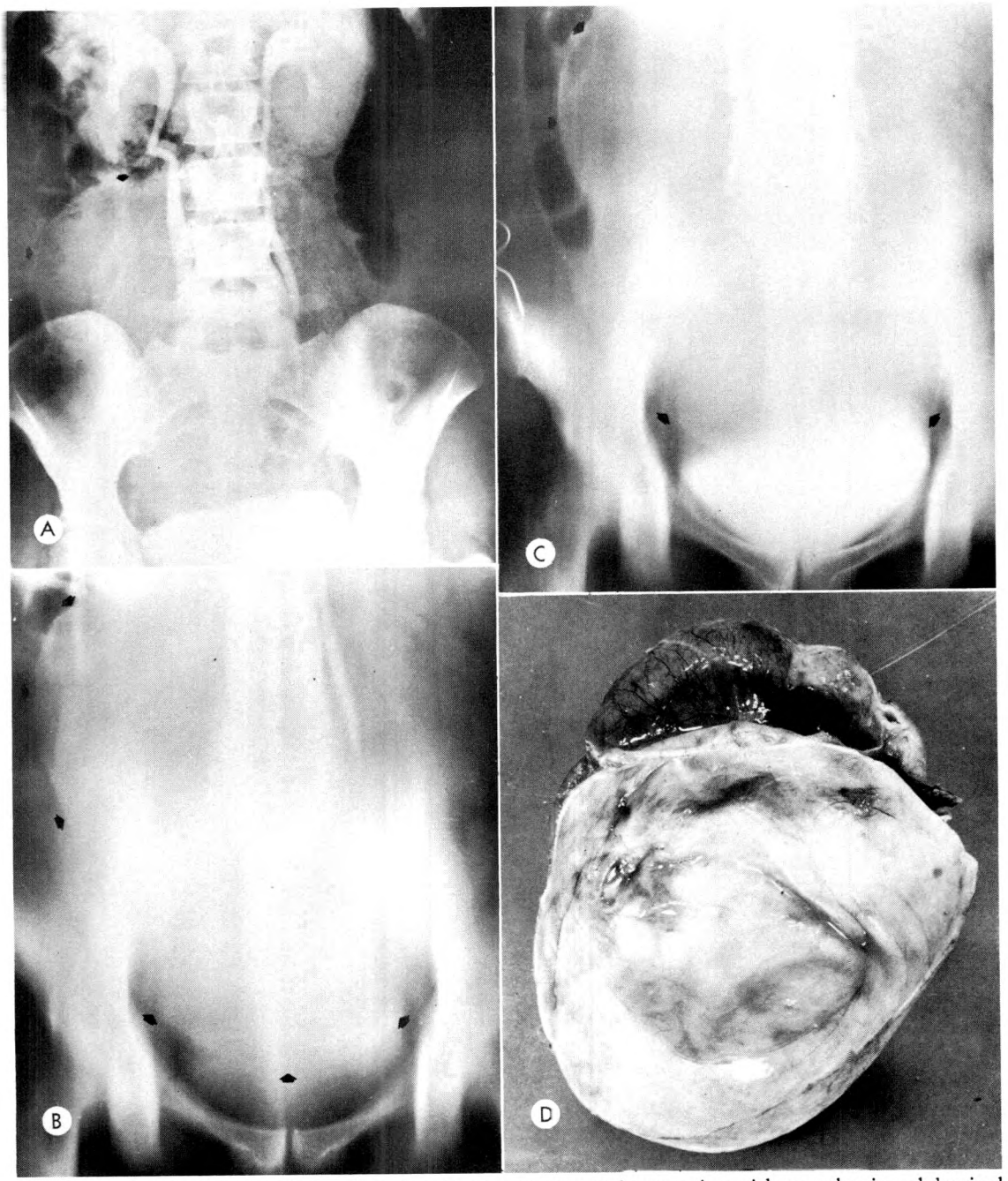


Fig. 6. Case vi. A 26 year old female 2 months post partum and presenting with an enlarging abdominal mass: suspect pregnancy.

(*A*) Intravenous pyelogram demonstrates a right pelvic mass to the level of L-3 (arrowheads). (*B* and *C*) Infusion tomograms demonstrate a large thin walled cyst with multiple sections revealing alternating areas of increased radiolucency (arrowheads). (*D*) Specimen: a right ovarian cystadenocarcinoma measuring 20×11×10 cm. with multiple papillary excrescences growing within the cyst cavity.

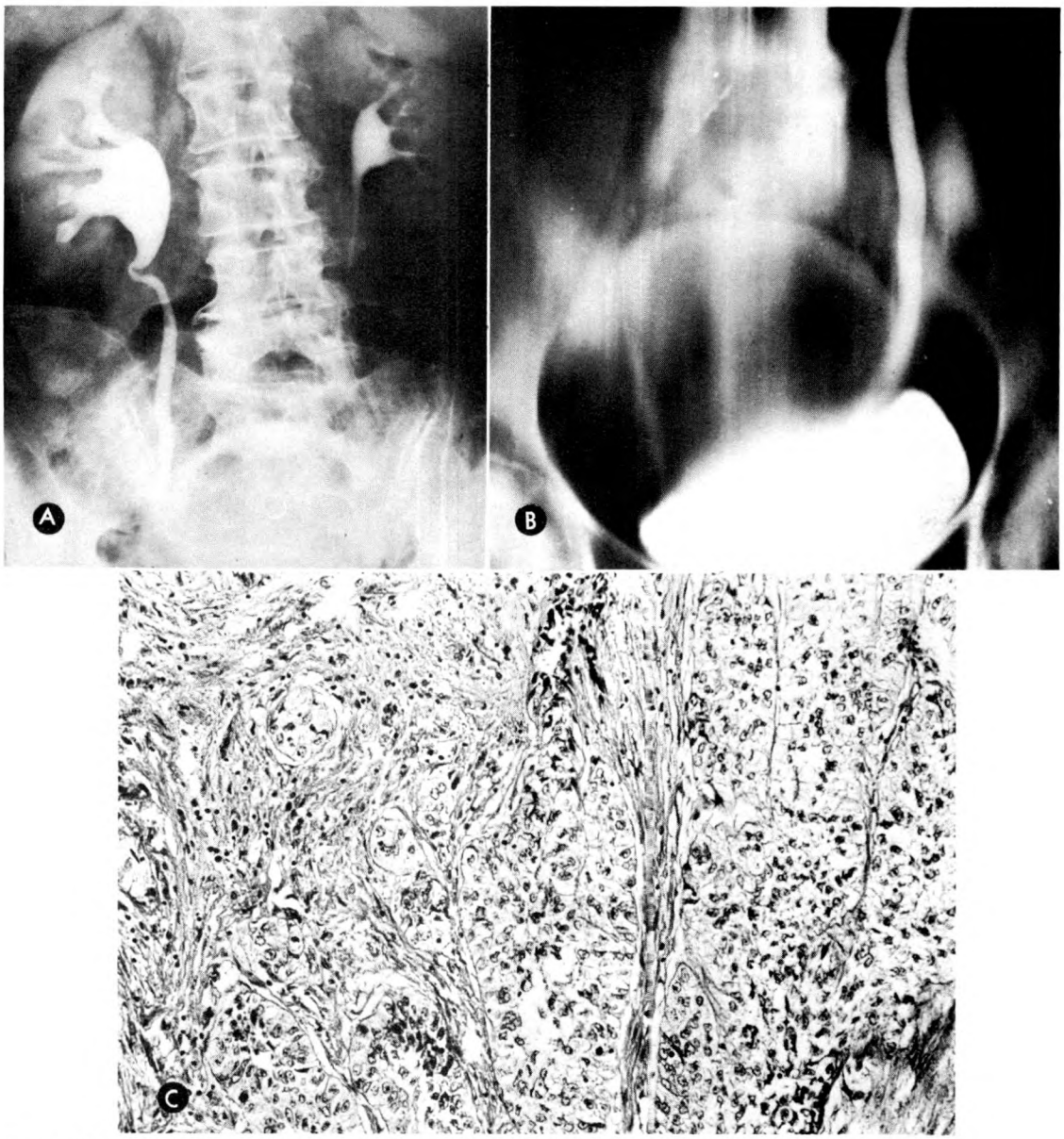


Fig. 7. Case VII. A 78 year old female with vaginal bleeding of 2 months' duration.

(*A*) Intravenous pyelogram demonstrating compression of the superior aspect of the bladder by a pelvic mass. (*B*) Infusion tomogram demonstrates a thick walled cystic appearing mass compressing the right side of the bladder. The thickness of the wall of this mass suggests malignancy. (*C*) Microscopic diagnosis is adenocarcinoma of the endometrium. The pathology report states "the intrauterine lumen is irregular with a grayish discoloration throughout the necrotic content." The myometrium is irregular measuring $2\frac{1}{2}$ cm. at its greatest thickness.

REFERENCES

1. Basic Radiation Protection Criteria. National Council on Radiation Protection and Measurements, Report No. 39. NCRP Publication, Washington, D.C., 1971.
2. Birnholz, J. C. Uterine opacification during excretory urography: definition of previously unreported sign. *Radiology*, 1972, *105*, 303–307.
3. Carlson, D. H., and Griscom, N. T. Ovarian cysts in newborn. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 664–672.
4. Cattell, W. R. Excretory pathways for contrast media. *Invest. Radiol.*, 1970, *5*, 473–486.

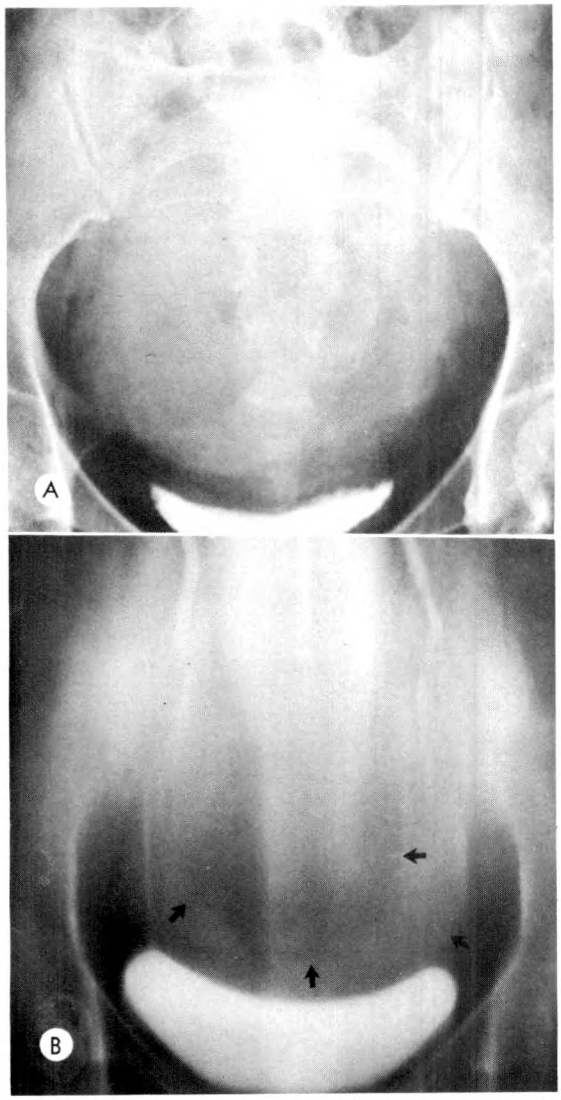


Fig. 8. Case viii. A 33 year old unmarried female presented with a melon sized pelvic mass and 2 negative pregnancy tests.

(*A*) Intravenous pyelogram demonstrates a solid appearing pelvic mass with a questionable radiolucent center. (*B*) Infusion tomogram shows the mass to have a smooth, thick wall and a definite radiolucent center which represents amniotic fluid (arrows). Patient denied sexual contact; however, when confronted with the evidence the story unfolded.

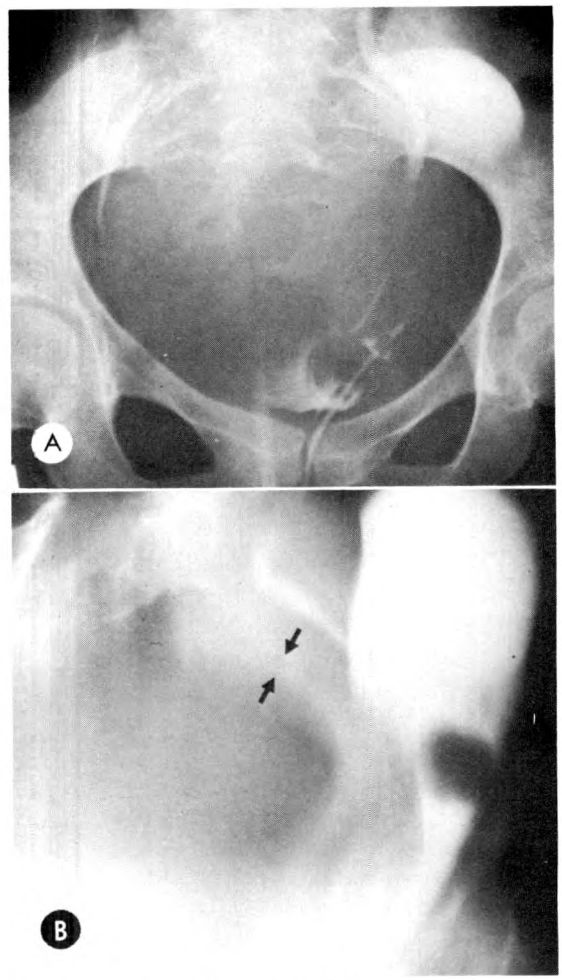


Fig. 9. Case ix. A 22 year old female with a pelvic mass and increasing constipation of 3 months' duration.

(*A*) Intravenous pyelogram demonstrates the bladder to be displaced anteriorly and to the left. Note "C" shaped defect in the distal sacrum. (*B*) Infusion tomogram demonstrates the thin, smooth rim of a radiolucent meningocele (arrows). This was proven at surgery.

5. Faulkner, R. L. Blood vessels of myomatous uterus. *Am. J. Obst. & Gynec.*, 1944, *47*, 185–197.

6. Kurti, D. J., and Patel, S. K. Diagnosis of adrenal cysts utilizing principle of total body opacification. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 576–579.

7. Martin, D. J., Griscom, N. T., and Neuhauser, E. B. D. Further look at total body opacification effect. *Brit. J. Radiol.*, 1972, *45*, 185–192.

8. O'Connor, J. F., and Neuhauser, E. B. D. Total body opacification in conventional and high dose intravenous urography in infancy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 63–71.

9. Rabushka, S. E., Love, L., and Moncada, R. Infusion tomography of gallbladder. *Radiology*, 1973, *109*, 549–552.

ULTRASONICALLY GUIDED PERCUTANEOUS ASPIRATION OF ABSCESSES*

By EDWARD H. SMITH, M.D., *and* ROYAL J. BARTRUM, JR., M.D.
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PULSED ultrasound has proven to be a valuable aid in evaluating patients with possible intra-abdominal or retroperitoneal abscesses.[2,7] When the abscess reaches 3 cm. in size, it can be identified in the majority of cases.[4] Occasionally some diagnostic difficulty will arise, however, when the abscess contains solid elements or when the clinical situation points to other explanations for a cystic abdominal mass.

We have used ultrasonically guided aspiration of the suspected abscess in such cases and consistently have been able to confirm the diagnosis.

REPORT OF CASES

CASE I. A 66 year old male was admitted with acute fulminant pancreatitis. His early course was stormy with extensive abdominal and retroperitoneal hemorrhage and tissue destruction. Seventeen days after admission, a large right retroperitoneal and scrotal abscess was drained. The postoperative course was satisfactory until the twenty-third hospital day, when the patient experienced increasing fever, abdominal pain and a slight elevation in amylase. Ultrasound examination of the abdomen revealed a large right retroperitoneal fluid collection in addition to a subphrenic fluid collection (Fig. 1, *A* and *B*). The surgical drain was ineffective since the fluid collection was deep to the drainage site. Because of the difficulty in clinically distinguishing between an infected pseudocyst or a sterile collection in a patient with chronic pancreatitis, the fluid collection was aspirated under ultrasonic guidance, purulent material obtained, and the diagnosis of abscess confirmed. A second surgical drainage was performed and the patient continues to do well.

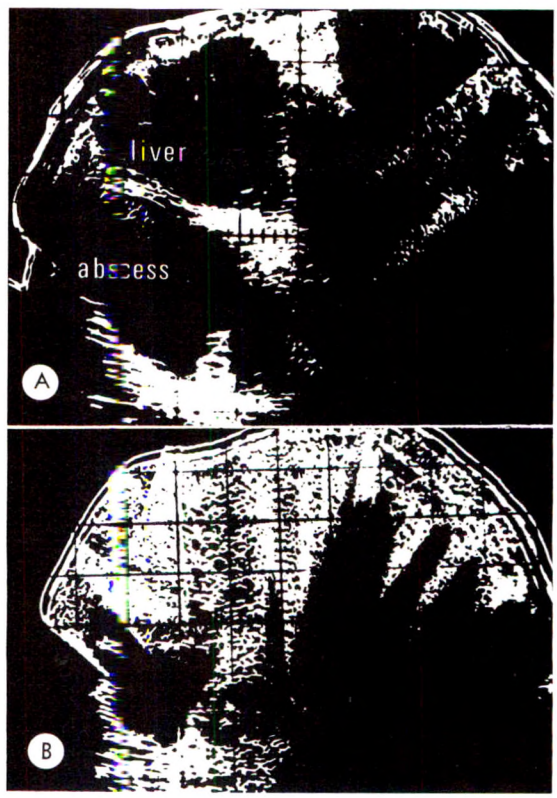


FIG. 1. CASE I. (*A* and *B*) Transverse ultrasonograms through the liver in a patient with a large subhepatic abscess.

(*A*) At low sensitivity both the liver and abscess are seen. (*B*) At high sensitivity the liver has filled in with echoes, while the abscess remains anechoic.

CASE II. A 75 year old male was admitted for abdominal pain and vomiting. His white blood cell count was 16,500 per mm.^{3} and his temperature was 101° F. without spiking. Intravenous pyelography showed a mass in the lower pole of the left kidney. Arteriography revealed a poorly vascular mass compatible with either abscess or tumor (Fig. 2, *A* and *B*). An ultrasound examination showed a mass consistent with abscess, but a necrotic tumor could not be excluded (Fig. 3, *A* and *B*). Aspiration of the mass revealed pus. The patient subsequently

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underwent surgical drainage of a large renal carbuncle. He did well postoperatively.

CASE III. A 24 year old juvenile diabetic received a cadaver renal transplant in March 1973. Her native kidneys were left intact. The transplant functioned well, but persistent urinary tract infections necessitated a right native nephrectomy and drainage of a right perinephric abscess in December 1973. The initial postnephrectomy course was good. Drainage from the wound ceased; the patient was afebrile and ready for discharge. On the 27th postoperative day, spiking fever, abdominal tenderness and an elevated white blood cell count again suggested an infectious process. The old wound was opened, but no new collection of pus was found. Urography and multiple plain roentgenograms were unremarkable. After much debate as to whether her course was due to rejection, an infected lymphocele, or an occult infection unrelated to her urinary tract, an ultrasound examination was performed. Her transplant kidney was shown to be normal, but an area suspicious for abscess was visible at the approximate site of the previous right nephrectomy (Fig. 4, *A* and *B*). Since this area was difficult to evaluate because of previous surgery and infection, percutaneous aspiration was

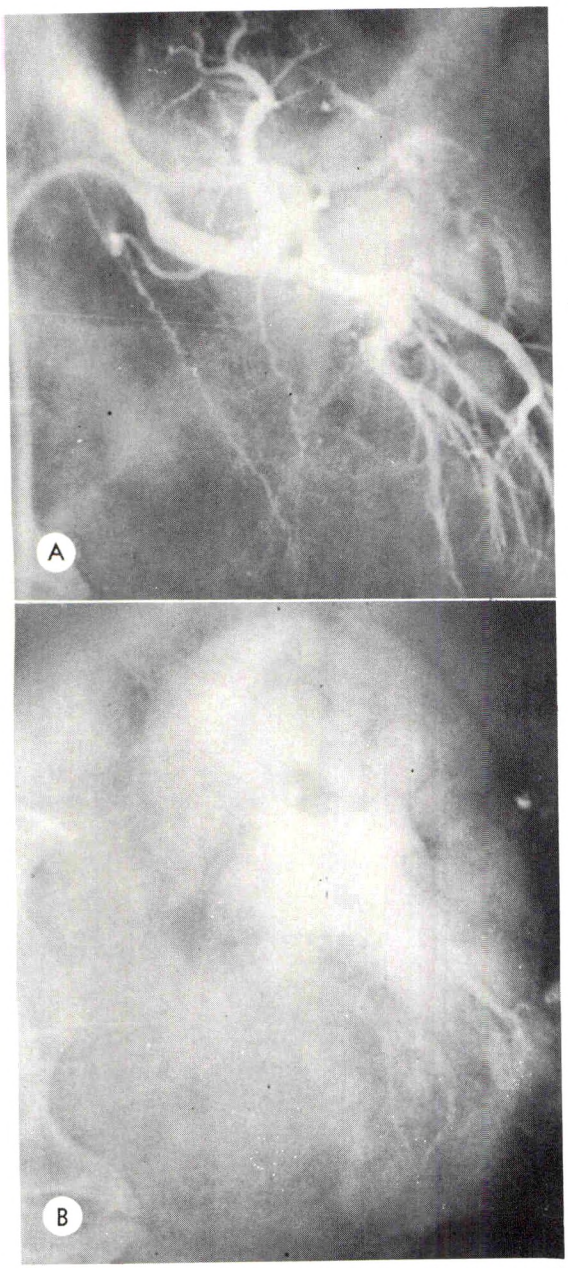


FIG. 2. Case II. (*A* and *B*) Left selective renal arteriograms in a patient with a large renal abscess. A tumor cannot be excluded.
(*A*) Arterial phase. (*B*) Capillary phase.

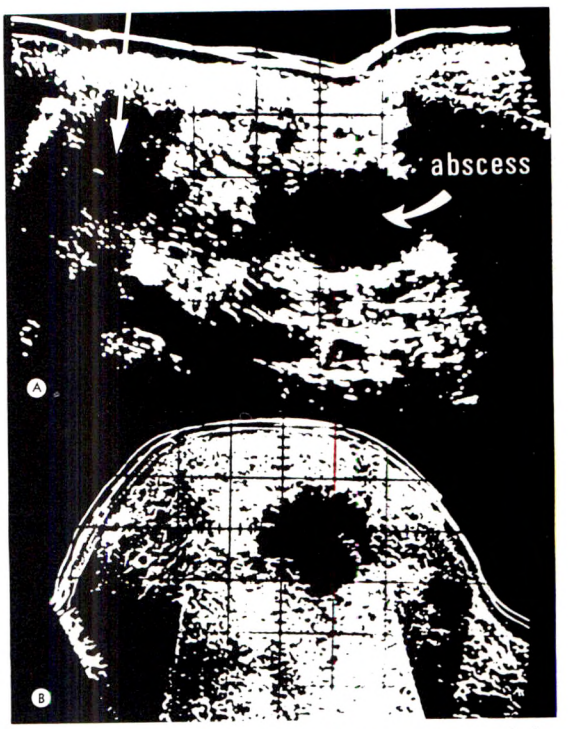


FIG. 3. Case II. (*A* and *B*) Ultrasonograms of the same patient as in Figure 2.
(*A*) Longitudinal prone scan. The long arrow points to the upper pole of the kidney. The short, curved arrow shows the abscess in the lower pole. (*B*) Transverse scan through the abscess showing it to be anechoic.

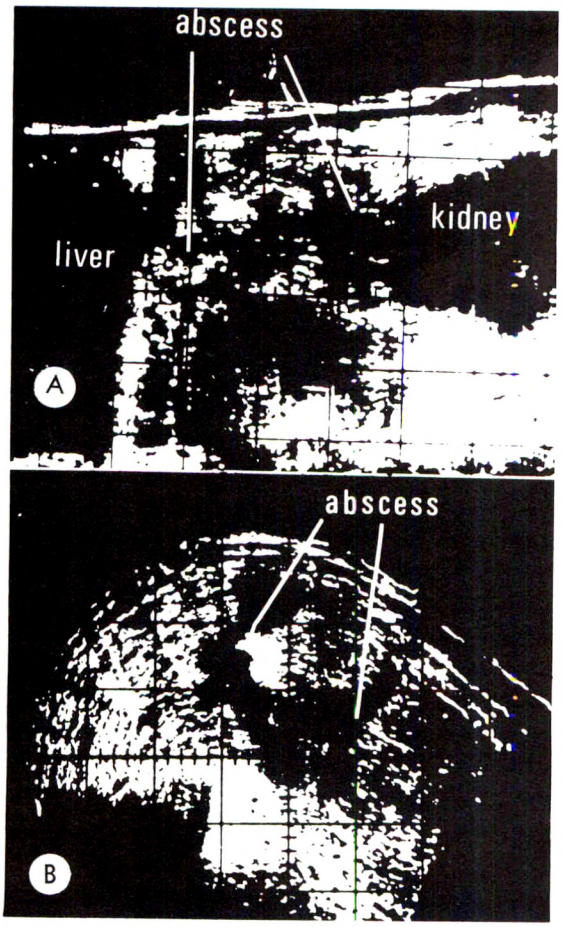


Fig. 4. Case III. (*A* and *B*) Retroperitoneal abscess in a patient with a renal transplant.

(*A*) Longitudinal supine ultrasonogram showing multiple anechoic areas in the right flank. The transplanted kidney is in the right iliac fossa. (*B*) Transverse section through the abscess with patient lying on left side.

performed under ultrasonic guidance and pus was obtained. At surgery, a large retroperitoneal abscess was found to extend to the subphrenic area.

Case IV. A 19 year old male developed acute renal allograft rejection 2 months after transplantation which necessitated nephrectomy. Shortly after the allograft was removed, a mycotic aneurysm developed in the internal iliac artery involved in the transplant. This was excised and the patient did well postoperatively and was discharged. Five months later, he was readmitted with pain at the site of the transplant. Temperature was 99° F., white blood cell count 10,300 per mm.^{3}. A plain abdominal roentgenogram, barium enema examination and arteriography were unrevealing. An ultrasound scan showed echo-free areas in the transplant bed (Fig. 5, *A* and *B*).

Because of confusing clinical findings and a history of previous surgical procedures in the area, it was felt that the diagnosis of abscess should be confirmed by aspiration with ultrasonic guidance. Pus was obtained and a surgical incision and drainage subsequently performed.

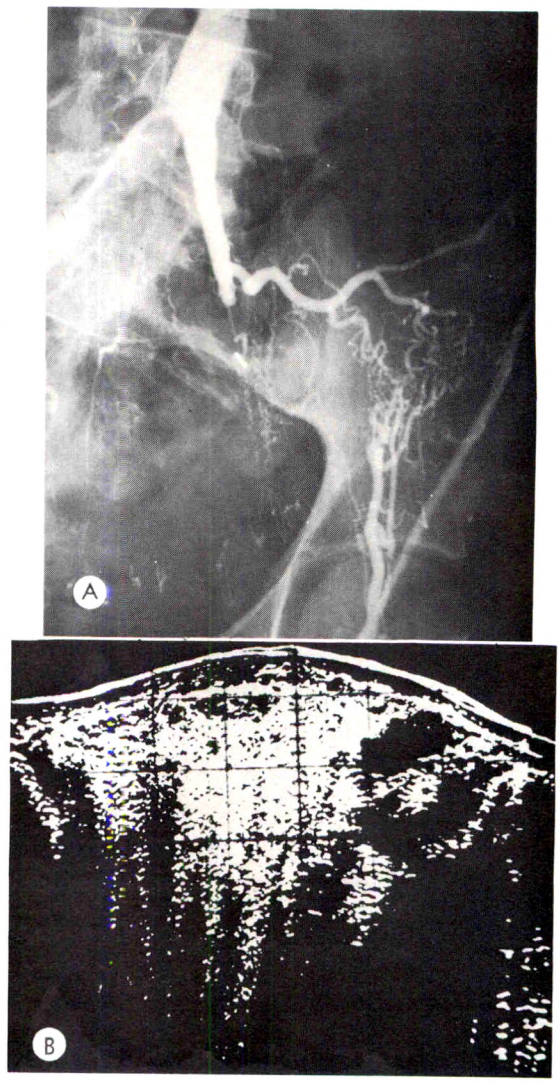


Fig. 5. Case IV. (*A* and *B*) Arteriogram and ultrasonogram of patient with an abscess in the bed of previous renal transplant.

(*A*) Arteriogram reveals only collateral vessels and postsurgical changes. (*B*) Transverse supine ultrasonogram shows anechoic areas in left pelvis highly suspicious of an abscess.

DISCUSSION

Percutaneous aspiration has been profitably used to diagnose many types of abdominal pathology. A limitation of the technique has been the difficulty in knowing the exact location of the needle point. The development of special ultrasound transducers which permit the accurate placement of the biopsy needle has greatly expanded the usefulness of aspiration biopsy.[1,3] The ultrasonically guided puncture technique has been described in detail by Holm *et al.*[5] and consists of locating the suspicious lesion using standard B-mode ultrasound. After the lesion has been identified, the regular transducer is replaced with the biopsy transducer which has a central canal through which the needle is passed (Fig. 6). The direction of the needle is assured by the transducer and the desired depth of puncture can accurately be determined from the A-mode display.

Using this technique, Kristensen *et al.*[6] have had a 90 per cent success rate in diagnosing renal mass lesions. Rasmussen *et al.*[9] compared the technique with standard blind Menghini biopsy in liver lesions. They reported an 85 per cent success rate in aspirating liver lesions with ultrasound as compared with 23 per cent for the blind Menghini biopsy in the same institution.

Despite the apparent advantages of this technique in the evaluation of possible intra-abdominal abscess, there has been only 1 report of its use in this condition.[8] We do not use aspiration in all patients with suspected abscess because the clinical setting and ultrasound examination are usually adequate for making the diagnosis. The cases reported show how the judicious use of guided percutaneous aspiration can clarify the diagnosis when confusion arises. This is especially important when the patient's clinical condition is precarious and the surgeon requires a confirmed diagnosis. When surgery is not possible or necessary, the organism may be cultured and the appropriate antibiotic chosen.

Morbidity appears to be insignificant but the possibility of extension of the infection must be considered. If prompt surgical treatment is to follow, this hazard appears to be minimal.

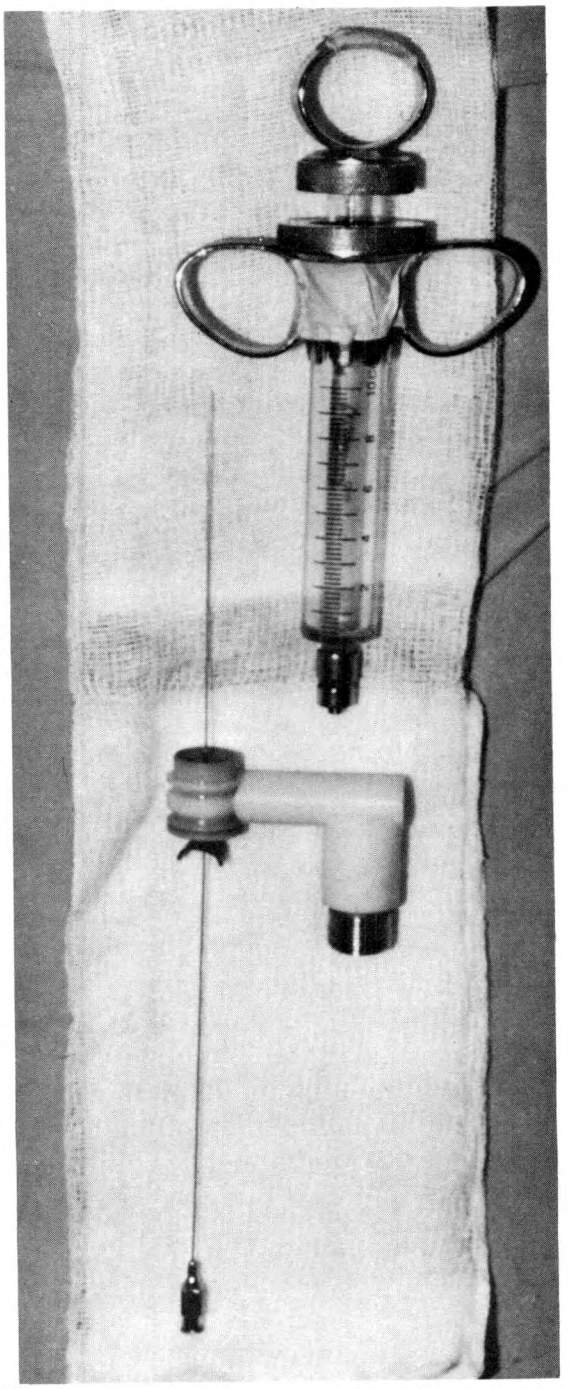


FIG. 6. Ultrasonic biopsy transducer with aspiration needle and syringe.

SUMMARY

Ultrasonic examination can reliably diagnose intra-abdominal or retroperitoneal abscesses in the majority of patients. Some cases in which the diagnosis is unsure or a precarious clinical condition contraindicates exploratory surgery can be definitively diagnosed by an ultrasonically guided aspiration of the suspected abscess.

The discomfort and risk are minimal.

Four cases are reported to demonstrate the value of such aspiration in selected patients.

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REFERENCES

1. Goldberg, B. B., and Pollack, H. M. Ultrasonic aspiration transducer. *Radiology*, 1972, *102*, 187–189.
2. Holm, H. H. Ultrasonic scanning in diagnosis of space occupying lesions of upper abdomen. *Brit. J. Radiol.*, 1971, *44*, 24–36.
3. Holm, H. H., Kristensen, J. K., Rasmussen, S. N., Northeved, A., and Barlebo, H. Ultrasound as guide in percutaneous puncture technique. *Ultrasonics*, 1972, *10*, 83–86.
4. Holm, H. H., Kristensen, J. K., Rasmussen, S. N., and Pedersen, J. F. Indications for abdominal ultrasonic scanning. Presented at Second World Congress on Ultrasonics in Medicine, Rotterdam, June 4–8, 1973.
5. Holm, H. H., Rasmussen, S. N., and Kristensen, J. K. Ultrasonically guided percutaneous puncture technique. *J. Clin. Ultrasound*, 1973, *1*, 27–31.
6. Kristensen, J. K., Holm, H. H., Rasmussen, S. N., and Barlebo, H. Ultrasonically guided percutaneous puncture of renal masses. *Scandinav. J. Urol., Nephrol.*, 1972, *15*, Suppl. 6, 49–50.
7. Leopold, G. R., and Asher, W. M. Diagnosis of extra-organ retroperitoneal space lesions by B-scan ultrasonography. *Radiology*, 1972, *103*, 130–133.
8. Pedersen, J. F., Hancke, S., and Kristensen, J. K. Renal carbuncle: antibiotic therapy governed by ultrasonically guided aspiration. *J. Urol.*, 1973, *109*, 777–778.
9. Rasmussen, S. N., Holm, H. H., and Kristensen, J. K. Ultrasonically guided liver biopsy. *Brit. M. J.*, 1972, *2*, 500–502.

Vol. 122, No. 2

THE SIGNIFICANCE OF A PALPABLE LIVER*

A CORRELATION OF CLINICAL AND RADIOISOTOPE STUDIES

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HEPATIC palpation is routinely performed and widely used to diagnose liver enlargement. A palpable liver edge first draws attention to possible liver enlargement, but most texts add the proviso that percussion is necessary to define the upper border of the liver.[1,3,4,8,12,13] However, their emphasis is placed on exclusion of major downward displacement of the diaphragm and liver rather than on the use of percussion and palpation together to obtain an exact liver span. In a limited series, Peternel *et al.*[7] found that the upper border of the liver as determined by percussion was frequently lower than that determined by hepatic scintiscan (up to 5.5 cm.). In addition, pathology in the lower right pleural cavity such as consolidation may make liver dullness seem higher than it actually is.[1] The limitations of hepatic percussion are well recognized and it appears that in practice palpation alone is widely accepted as adequate for the detection of hepatomegaly. This is evidenced by our review of hospital charts which showed that percussion was frequently neglected even when the liver edge was palpated. Furthermore, many patients, solely on the basis of palpable edge, have been referred for liver scanning to evaluate "hepatomegaly."

A comparison of physical examination with autopsy findings[9] in a series of patients showed that palpability was not a good index of hepatomegaly, but that study was limited by variable time interval between the physical examination and death and by the possibility of immediate premortem changes such as acute hepatic congestion. Another study[6] demonstrated that the liver scan can give a measure of liver size that correlates well with autopsy findings. Methods to evaluate liver size on radioisotope scan using multiple levels of background erase,[14] planimetry,[6] and area[5] and volume calculations[10] have all been advocated. We have recently shown that a single vertical measurement made at a point halfway between the xiphoid and right liver margin has excellent correlation with hepatic disease.[11]

Since many clinicians still accept the philosophy of F. M. Hanger, Jr. that "one good feel of the liver is worth any two liver function tests,"[2] the present study, using our method, not only compares palpation with the radioisotope scan as an index of hepatomegaly, but also correlates both those modalities with laboratory and tissue diagnosis to determine their relative reliability as criteria for hepatic pathology. In addition, the projection of the liver below the right costal margin on scan is compared with palpability.

MATERIAL AND METHOD

One hundred in-patient liver scans were chosen randomly from scannings performed between 1967 and 1972 (age range 24–85 years). These consisted of approximately equal numbers of studies done with colloidal Au^{198} and Tc^{99m} sulfur colloid. The scans were made on a 5 inch dual head rectilinear scanner with use of image diffusion (data blending), and similar technical factors were used in all cases. The xiphoid and the costal margin were indicated on the scan. At a point half-way between the xiphoid and the right liver margin, the vertical dimension of the liver was

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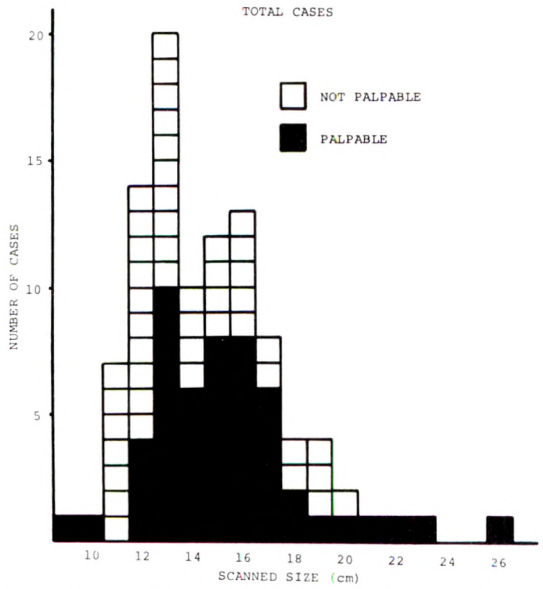


FIG. 1. Palpability and scan size of liver in all cases of the series. Each column represents the scan size ±0.5 cm. of the indicated size.

measured. Compared to other lengths, we have shown this measurement to be the most reliable indicator of hepatic enlargement and pathology.[11] The projection of the liver below the costal margin was also measured. Each patient's hospital chart was independently reviewed. All patients had determinations of SGOT and alkaline phosphatase and most also had leucine amino peptidase (LAP) determinations. The highest value of these enzymes recorded during the admission in which the scanning was performed was noted for data analysis. Autopsy or biopsy results, if done within 6 months of the scan, were noted. The palpability of the liver as indicated on the intern's discharge summary was used for comparison with the scan size. To exclude ambiguous cases, for the purpose of this paper only, those palpable 1 cm. or more below the right costal margin were regarded as palpable (*i.e.* those recorded as palpable just at the costal margin were regarded as nonpalpable).

RESULTS

All cases were charted according to scan size as in Figure 1 with an indication of palpability. The scan size (mean ± s.d.) of the palpable livers was 15.5 ± 3.3 cm. and of the nonpalpable livers 14.0 ± 2.5 cm. As can be seen from these data and the Figure, palpability was not a good index of enlargement of the liver. For further analysis the cases were divided into those with normal and abnormal livers. A third group was defined as those livers found enlarged by scan.

Normal. Thirty-three of the 100 patients were normal by all the following criteria:

1. Normal SGOT, bilirubin, and LAP (or alkaline phosphatase where the LAP was not done) during the entire hospital admission.
2. No tissue abnormality, if a biopsy or postmortem examination was performed.
3. No focal abnormalities or heterogeneous uptake on the scan.

This group is charted in Figure 2. The scan size was 12.7 ± 1.4 cm. with a maximum size of 15.4 cm. There were 12 negative biopsies or autopsies and no positive ones. Of this group 15 of the 33 livers were palpable with a scan size of 12.9 ± 1.7 cm. The 18 nonpalpable livers have a similar size distribution with a mean of 12.5 ± 0.6 cm.

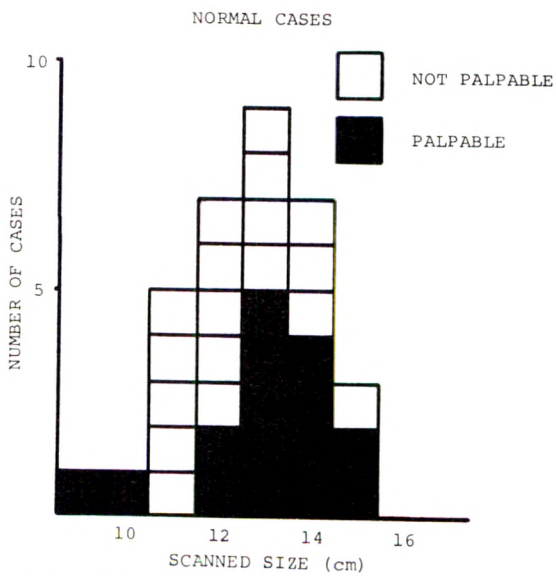


FIG. 2. Palpability and scan size of liver in cases with no evidence of hepatic disease.

Abnormal. All the remaining 67 cases were considered abnormal. Of these, 35 had only abnormal SGOT, bilirubin, and/or LAP, 3 had a positive tissue diagnosis of hepatic disease but normal enzyme studies, and 29 had both abnormal laboratory values and a positive tissue diagnosis. The tissue diagnoses consisted of 23 cases of metastatic carcinoma, 3 of "fatty change," 2 of cirrhosis, 2 of chronic hepatitis, 1 of bile stasis due to common duct obstruction and 1 sclerosing angiohamartoma. As can be seen from Figure 3, the palpable livers were distributed throughout this group with a scan size of 16.5 ± 3.2 cm., compared to the nonpalpable livers with a scan size of 15.0 ± 2.7 cm. The 4 largest livers were all palpable. No significant difference was noted in scan size or frequency of palpability between the 36 cases which were abnormal by enzymes alone and the 31 biopsy proven cases.

Enlarged by scan. Based on the analysis of the normal group,[11] a size of greater than 15.5 cm. on the scan was defined as liver enlargement. All livers enlarged by this criterion were in the abnormal group discussed above. As can be seen from Figure 1, of the 37 cases in this group only 24 were palpable. The projection of the liver below the costal margin on the scan was 5.3 ± 3.0 cm. for the palpable livers of this group and 5.6 ± 3.0 cm. for the nonpalpable livers. The clinically noted extension below the costal margin of the palpable livers which were enlarged by scan (Table 1: Group I) was 6.7 ± 3.4 cm. For comparison, the clinical extension below the costal margin of palpable livers which proved not to be enlarged by scan (Table 1: Group III) was 5.0 ± 3.0 cm.

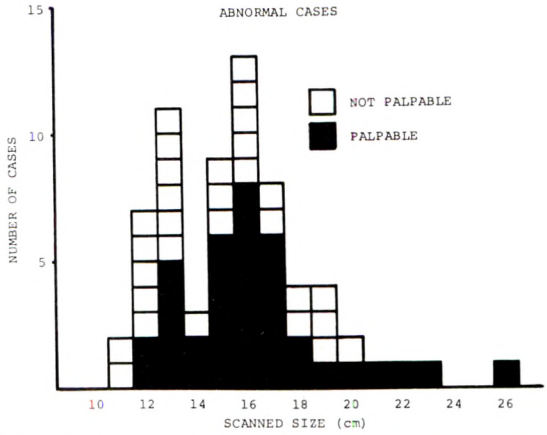


FIG. 3. Palpability and scan size of liver in cases with some indication of hepatic disease.

DISCUSSION

In only 59 per cent of our cases was there agreement between the presence or absence of hepatic enlargement as defined by the scan and the clinicians' findings by palpation (Table 1: Groups I and IV). The question then arises which of these diagnostic maneuvers is the more accurate or useful. Based on the analysis of our normal group we have been able to define an upper limit of liver size as measured on the scan[11]; *i.e.*, every case in our series with a liver "height" of 15.5 cm. or greater, measured vertically at a point half-way between the midsternal line and the right lateral liver

TABLE I

CORRELATION OF PALPABILITY, ENLARGEMENT BY SCAN, AND DOWNWARD DISPLACEMENT OF LIVER

Group	No. of Cases	Enlarged Scan	Palpable	Scan Size (cm. ± S.D.)	Distance Below Costal Margin	
					by scan (cm. ± S.D.)	by palpation (cm. ± S.D.)
I	24	+	+	18.0 ± 3.0	5.3 ± 3.0	6.7 ± 3.4
II	13	+	−	17.6 ± 1.4	5.6 ± 3.0	0
III	28	−	+	13.3 ± 1.4	3.4 ± 2.7	5.0 ± 3.0
IV	35	−	−	12.7 ± 1.3	2.0 ± 2.3	0

border, had some evidence of hepatic disease. Such a correlation with disease could not be derived from the palpability of the liver edge. It should be pointed out that the value for the upper limit of normal of scan size that we have adopted may be different for different techniques of liver scanning. Since the liver edge as seen on an isotope scan is never an absolutely sharp line, its actual placement will vary somewhat with the contrast properties of the final scan image. However, for any given scanning technique the normal liver "height" can be determined and regardless of whether or not this measurement can be directly converted to liver weight, which is the ultimate measure of hepatomegaly, it serves as an index of liver size which is better related to pathology than palpation. A normal liver size by scan obviously does not rule out disease.

One of the possible explanations for the frequent palpability of livers which are not enlarged by scan might be that some livers are displaced downward by low lying diaphragms. This is undoubtedly true in some cases and indeed in our series there was a significant difference ($p<.05$) between the mean displacement below the costal margin of the normal size palpable and nonpalpable groups (Table I: Groups III and IV). However, that cannot be a general explanation, since 14 of the palpable livers extended a shorter distance below the costal margin than 10 of the nonpalpable livers.

Another possible explanation is that those livers which are palpated but not actually enlarged are diseased and therefore have a firmer consistency. In our series this was not the case since only 12 of the 28 livers which were palpable but not enlarged by scan (Table I: Group III) had any evidence of abnormality. There were 4 positive autopsies in this group and 6 negative ones; 3 biopsies were negative and 1 positive. By contrast in Group II (Table I) all had an abnormal LAP and 6 had a positive tissue diagnosis (no tissue was reported as negative), yet none of these 13 livers were palpable. It is not clear why such livers should have been missed by palpation. Ascites might interfere with palpation but this could have explained only one of these cases. Obesity or other variations of body build could well play a role, but such factors were not specifically evaluated.

A lack of experience on the part of the physician might result in an inaccurate physical examination and contribute to the lack of correlation between palpability and scan size of the liver. In our study the statement of palpability was taken from the discharge summary and any differences between the intern and resident or visiting physician would presumably have been resolved in favor of the most reliable examination. As a further check of this point, we analyzed our results on the basis of trimonthly time periods and found that the correlation between scan size and palpability did not improve between the first and last quarter of the internship year, as it might have if the house officers' clinical skills were a major variable.

CONCLUSION

In conclusion, the chance of a palpable liver being abnormal in our series is only 37/67 (57 per cent) and similarly the chance of a palpable liver being enlarged by scan is 24/52 (46 per cent).

Palpability alone is not a useful indicator of hepatic size or disease.

On the other hand, hepatomegaly can be defined by a simple measurement on the radioisotope liver scan which if exceeded is an accurate indication of hepatic disease. In our series 33/33 livers exceeding this limit were abnormal.

The liver scan is necessary for reliable assessment of size and may prevent or limit unnecessary investigations for "hepatomegaly."

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REFERENCES

1. ADAMS, F. D. Physical Diagnosis. Williams & Wilkens Company, Baltimore, 1958.
2. HANGER, F. M. Quoted in Schiff, L. Diseases of the Liver. W. B. Saunders Company, Philadelphia, 1971.
3. HOPKINS, H. U. Leopold's Principles and Methods of Physical Diagnosis. W. B. Saunders Company, Philadelphia, 1965.
4. KNEELAND, Y., JR., and LOEB, R. F. Martinis's Principles and Practice of Physical Diagnosis. J. B. Lippincott Company, Philadelphia, 1962.
5. MACAFFEE, J. G., AUSE, R. G., and WAGNER, H. N. Diagnostic value of scintillation scanning of liver. *A.M.A. Arch. Int. Med.*, 1965, *116*, 95–110.
6. NAFTALIS, J., and LEEVY, C. M. Clinical estimation of liver size. *Am. J. Digest. Dis.*, 1963, *8*, 236–243.
7. PETERNEL, W. W., SHAFFER, J. W., and SCHIFF, L. Clinical evaluation of liver size and hepatic scintiscan. *Am. J. Digest. Dis.*, 1966, *11*, 346–350.
8. PRIOR, J. A., and SILBERSTEIN, J. S. Physical Diagnosis. The C. V. Mosby Company, St. Louis, 1963.
9. RIEMENSCHNEIDER, P. A., and WHALES, J. P. Relative accuracy of estimation of increased liver and spleen by radiologic and clinical methods. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 462–468.
10. ROLLO, F. D., and DELAND, F. H. Determination of liver mass from radionuclide images. *Radiology*, 1968, *91*, 1191–1194.
11. ROSENFIELD, A. T., and SCHNEIDER, P. B. Rapid evaluation of hepatic size on radioisotope scan. *J. Nuclear Med.*, 1974, *15*, 237–240.
12. SHERLOCK, S. Diseases of the Liver and Biliary System. F. A. Davis Company, Philadelphia, 1968.
13. STERN, T. N. Clinical Examination. Year Book Medical Publishers, Inc., Chicago, 1964.
14. YAGAN, R., MACINTYRE, W. J., and CHRISTIE, J. H. Estimation of liver size by multiple cut-off scintillation scanning technique. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *88*, 289–295.

POSITIVE IMAGING OF LIVER TUMORS BY DELAYED HEPATIC SCINTISCANNING WITH ACIDIC IONIC INDIUM 113m CHLORIDE*

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LIVER scanning with radiocolloids renders enough information for the precise localization of space-occupying lesions of the liver, which are seen on the scan image as filling defects. Unfortunately, this information is nonspecific, as such defects may be due to a great variety of lesions and it is not sufficient to reach a differential diagnosis between tumors,[21] cysts[18] and abscesses.[4]

By comparing these images with the scans of the hepatic blood pool procured a few minutes after the intravenous administration of ionic In^{113m} chloride at pH 1.4 to 1.7, we have been able to differentiate the etiology of the "cold" areas in 72 per cent of primary carcinomata of the liver and in 70 per cent of hepatic metastases.[5,11,12] However, this combined procedure is unable to differentiate the "cold" areas produced by liver tumors with poor vascularity from those caused by hepatic cysts and abscesses.[5,11,12]

Based on experimental evidence that seems to show that acidic ionic In^{113m} chloride is concentrated by the liver as a function of time,[13] we decided to explore the diagnostic potential of delayed hepatic scanning 6 hours after the intravenous administration of this radiopharmaceutical.

MATERIAL AND METHOD

A total of 22 patients was included in this study, of whom 13 had hepatic primary carcinomata and 9 had metastatic tumors to the liver. The diagnosis was confirmed in each instance by selective hepatic arteriography, peritoneoscopy, needle biopsy or laparotomy (and by necropsy in fatal cases).

In^{113m} chloride was eluted from a sterile Sn^{113}-In^{113m} generator, using 0.5 *N* HCl.[3] In^{113m} Fe coprecipitates were prepared using the method described by Adatepe and associates,[1] and sterile ionic, carrier-free In^{113m} chloride as eluted from the generator (pH 1.4 to 1.7) was administered for scanning of the hepatic blood pool.[6,7,10,16,17]

A liver scan was first obtained 10 minutes after the intravenous administration of 2 millicuries of In^{113m} Fe microcoprecipitates by using a Picker Magnascanner 500 with a 12.7 cm. (5 inch) crystal detector and a color imaging device. One or more filling defects were revealed by these radiocolloid scans. A few hours later, or on the next day, a second liver scan was procured 10 minutes after the intravenous administration of 10 millicuries of ionic, carrier-free, acidic In^{113m} chloride in order to depict the spacial distribution of the hepatic blood pool. The scanner was calibrated in the hepatic region with the highest counting rate, disregarding the somewhat higher radioactivity in the region of the cardiac blood pool, which appears on the scan image as an area where the imaging device was saturated. Lastly, a third scan was obtained 6 hours after the administration of the radiopharmaceutical, calibrating the imaging device at the point of highest counting rate in the liver. For a better comparison of the resulting images, these were digitized according to 5 different levels of radioactivity: 0–25 per cent (–), 26–50 per cent (o), 51–75 per cent (w), 76–100 per cent (#), and more than 100 per cent (M) of the maximum count rate. Notation was

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made in every instance of the counting rate at the cardiac blood pool, in a "normal" hepatic region and at the site of the lesion. This information was corrected by the physical decay of In^{113m}, and the following indexes were calculated:

1. Heart $_{6\ hr.}$	/ Heart $_{10\ min.}$	(H_6/H_{10})
2. Liver $_{6\ hr.}$	/ Liver $_{10\ min.}$	(L_6/L_{10})
3. Tumor $_{6\ hr.}$	/ Tumor $_{10\ min.}$	(T_6/T_{10})
4. Liver $_{10\ min.}$	/ Heart $_{10\ min.}$	(L_{10}/H_{10})
5. Tumor $_{10\ min.}$	/ Heart $_{10\ min.}$	(T_{10}/H_{10})
6. Liver $_{6\ hr.}$	/ Heart $_{10\ min.}$	(L_6/H_{10})
7. Tumor $_{6\ hr.}$	/ Heart $_{10\ min.}$	(T_6/H_{10})
8. Tumor $_{10\ min.}$	/ Liver $_{10\ min.}$	(T_{10}/L_{10})
9. Tumor $_{6\ hr.}$	/ Liver $_{6\ hr.}$	(T_6/L_6)
10. Tumor $_{6\ hr.}$	/ Liver $_{6\ hr.}$	(T_6/L_6)
Tumor $_{10\ min.}$	/ Liver $_{10\ min.}$	(T_{10}/L_{10})

RESULTS

Six hours after the intravenous administration of acidic, ionic, carrier-free In^{113m} chloride, the cardiac blood pool still retained 24 per cent ±2 of the radioactivity detected 10 minutes after the administration of the radiopharmaceutical (H_6/H_{10}). This is in contrast with a significantly higher retention (36 per cent ±6) at the "normal" hepatic region (L_6/L_{10}), and with an even higher retention (54 per cent ±10) at the site where the concentration defect was found with the radiocolloid (T_6/T_{10}) (Fig. 1).

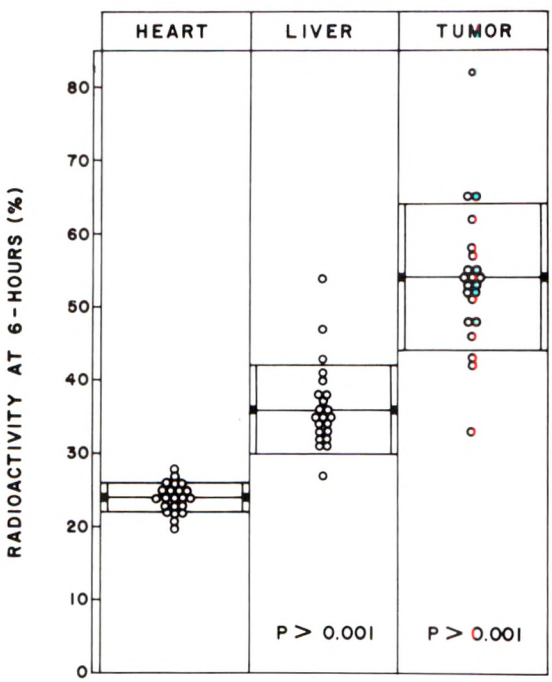


FIG. 1. Radioactive fraction remaining 6 hours after intravenous administration of In^{113m} chloride (expressed as a per cent of radioactivity found 10 minutes after its administration), at the cardiac blood pool, in a "normal" hepatic region and at the site of the tumor.

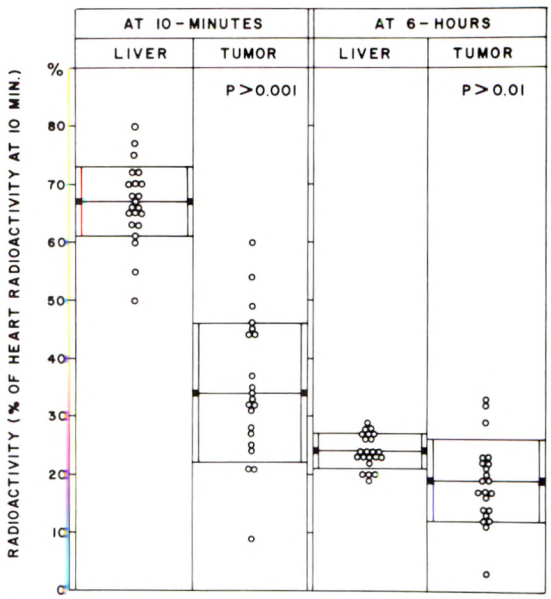


FIG. 2. Radioactivity in a "normal" hepatic region and at the site of the tumor, 10 minutes and 6 hours after the administration of In^{113m} chloride (expressed as a per cent of heart radioactivity at 10 minutes).

In 22 patients with primary and metastatic tumors of the liver, it was found that the radioactivity present in the "normal" hepatic region 10 minutes after the administration of acidic In^{113m} chloride was significantly higher than in the site of the tumor. At this time, the radioactive fraction in the "normal" hepatic region represents 67 per cent ±6 of the radioactivity found at the cardiac blood pool (L_{10}/H_{10}), while at the site of the tumor (T_{10}/H_{10}), this fraction stands for 34 per cent ±12 of the radioactivity found at the cardiac chamber (Fig. 2). The resulting scintigraphic contrast between these 2 regions still is obvious (Fig. 6–8).

Due to a faster disappearance rate of the

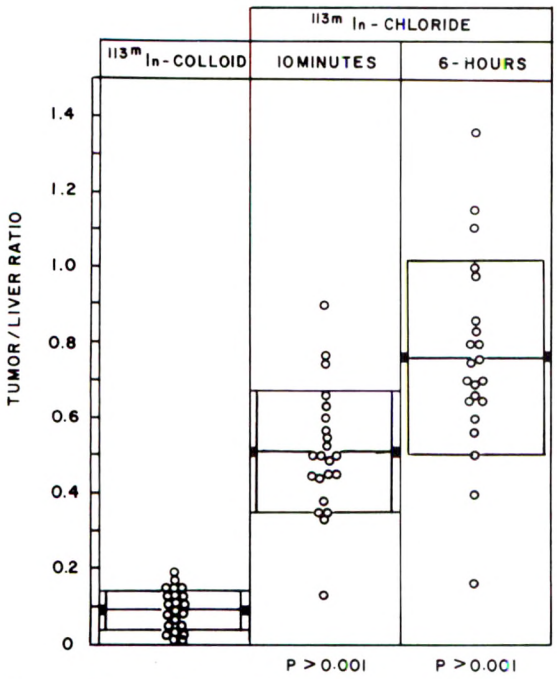


FIG. 3. Tumor/Liver ratio 10 minutes after the administration of In^{113m} Fe colloid, and 10 minutes and 6 hours after the administration of acidic ionic In^{113m} chloride.

radiopharmaceutical from the "normal" hepatic tissue than from the site of the tumor (Fig. 1), 6 hours later the differences of radioactivity between these 2 regions and the cardiac blood pool (L_6/H_{10}; T_6/H_{10}; H_6/H_{10}) are smaller (Fig. 2), considerably decreasing the scintigraphic contrast between the cardiac blood pool, the "normal" hepatic region and the site of the tumor (Fig. 6–8).

The ratio of the tumor radioactivity divided by the radioactivity found in the "normal" hepatic region on the study performed with radiocolloids is rather low (0.09 ± 0.05). This ratio increases to 0.51 ± 0.16, 10 minutes after the administration of acidic In^{113m} chloride (T_{10}/L_{10}), and to 0.76 ± 0.26, 6 hours later (T_6/L_6) (Fig. 3). A highly significant positive correlation was found between the ratio obtained at 10 minutes (T_{10}/L_{10}) and the ratio estimated 6 hours later (T_6/L_6) (Fig. 4).

A tumor concentration ratio was calculated by dividing the Tumor/Liver ratio at 6 hours (T_6/L_6) by the same ratio obtained 10 minutes after the administration of In^{113m} chloride (T_{10}/L_{10}). In every instance, this ratio resulted higher than 1.2 and lower than 1.6 (mean 1.37 ± 0.6) (Fig. 5). A highly significant positive correlation was observed between the Tumor/Liver ratio at 10 minutes (T_{10}/L_{10}) and this tumor concentration ratio (Fig. 5).

Table 1 shows that on the colloid scintiscans the Tumor/Liver ratio was lower than 0.25 in 100 per cent of cases. These lesions appeared on the color scan in white with some black dots, and on the digitized scan with the (–) sign. The scintigraphic contrast between these lesions and the normal concentrating hepatic tissue is obvious. On the scans obtained 10 minutes after the administration of acidic In^{113m} chloride, the images clearly demonstrated the presence of a blood pool in the interior of the tumor in 40.9 per cent of cases, which was depicted by a Tumor/Liver

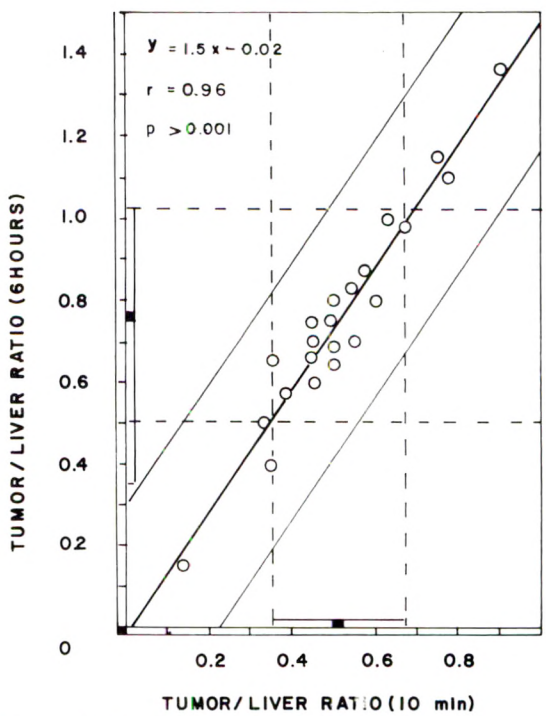


FIG. 4. Correlation between Tumor/Liver ratio at 10 minutes (Blood pool) and Tumor/Liver ratio at 6 hours (Tissue), after the administration of acidic ionic In^{113m} chloride.

ratio (T_{10}/L_{10}) higher than 0.50, by the (w) and (#) signs on the digitized scans and by green, yellow, orange and red dots on the color scan. In 54.5 per cent of cases, the existence of a vascular bed in the tumor was doubtful, and in just 1 case (4.5 per cent) there were no differences between the reticuloendothelial scan and the scintigraphic image of the hepatic blood pool. In this particular case, selective hepatic angiography demonstrated the presence of an avascular zone at the very site of the filling defect (Fig. 7, *A–D*).

On the delayed scans, obtained 6 hours after the intravenous administration of the radiopharmaceutical, the images clearly showed the presence of radioactivity in the interior of 86.4 per cent of hepatic tumors, including 3 cases (14.3 per cent) where the radioactive level at the tumoral area was higher than in the "normal" hepatic region (Fig. 8, *A–C*). In 2 cases (9.1 per

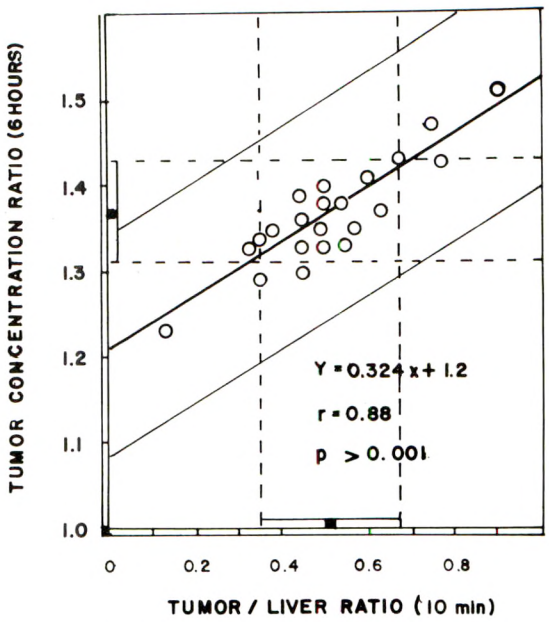


Fig. 5. Correlation between Tumor/Liver ratio at 10 minutes and the Tumor Concentration ratio $(T_6/L_6)/(T_{10}/L_{10})$ at 6 hours.

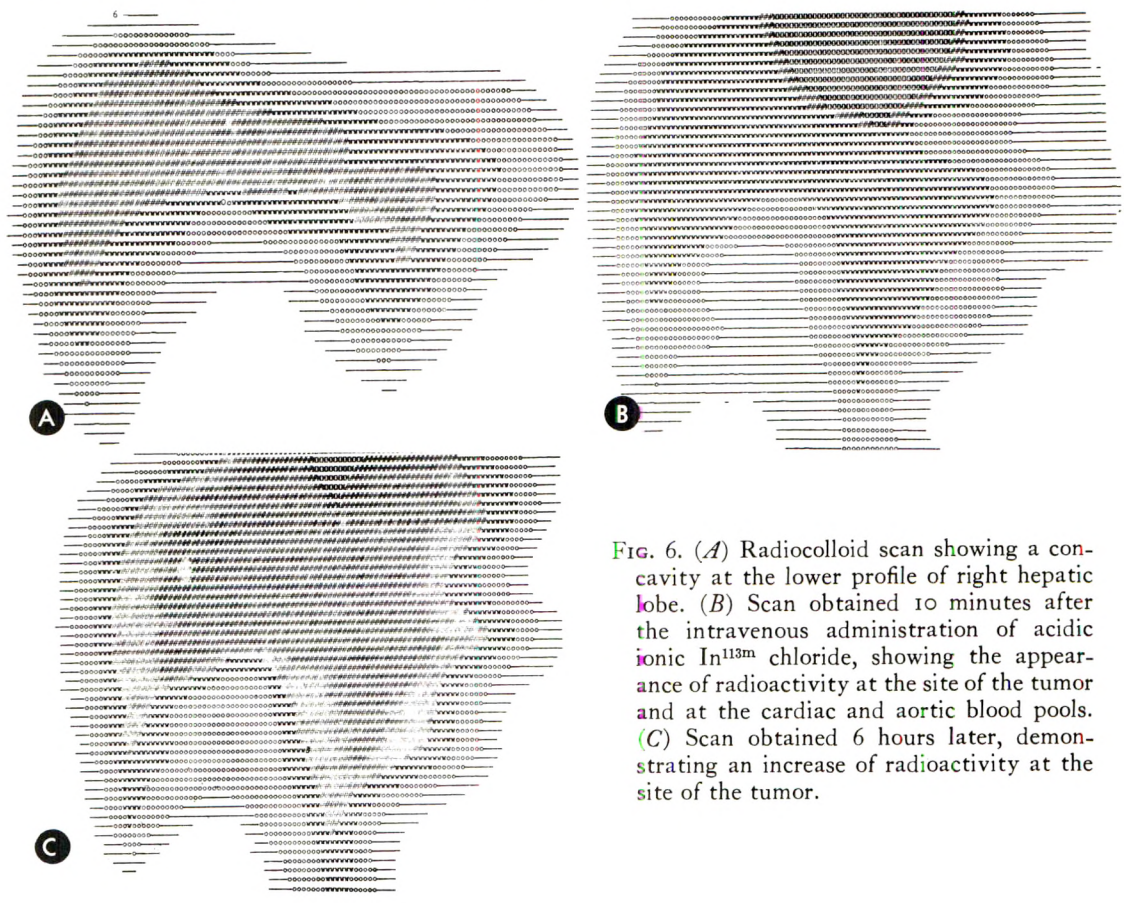


Fig. 6. (*A*) Radiocolloid scan showing a concavity at the lower profile of right hepatic lobe. (*B*) Scan obtained 10 minutes after the intravenous administration of acidic ionic In^{113m} chloride, showing the appearance of radioactivity at the site of the tumor and at the cardiac and aortic blood pools. (*C*) Scan obtained 6 hours later, demonstrating an increase of radioactivity at the site of the tumor.

TABLE I

TUMOR/LIVER RATIO, DIGITAL SIGN AND COLOR CODE AT THE SITES OF THE TUMOR IN 22 PATIENTS

Lesion/Liver	Digital Sign	Color Code	In^{113m} Fe Colloid	Acidic Ionic In^{113m} Chloride	
				10 Minutes	6 Hours
0–0.25	—	white, black	22 (100%)	1 (4.54%)	1 (4.54%)
0.26–0.50	o	violet, blue	0 (0%)	12 (54.54%)	2 (9.11%)
0.51–0.75	w	green, yellow	0 (0%)	7 (31.81%)	9 (40.20%)
0.76–1.00	#	orange, red	0 (0%)	2 (9.11%)	7 (31.81%)
1.00	M	saturated red	0 (0%)	0 (0%)	3 (14.34%)

cent), the results were doubtful, and just in 1 case (4.5 per cent) the concentration defect persisted in the left hepatic lobe (Fig. 7, *A–D*).

DISCUSSION

To date, liver scintigraphy with radiocolloids is considered as an excellent method for locating space-occupying le-

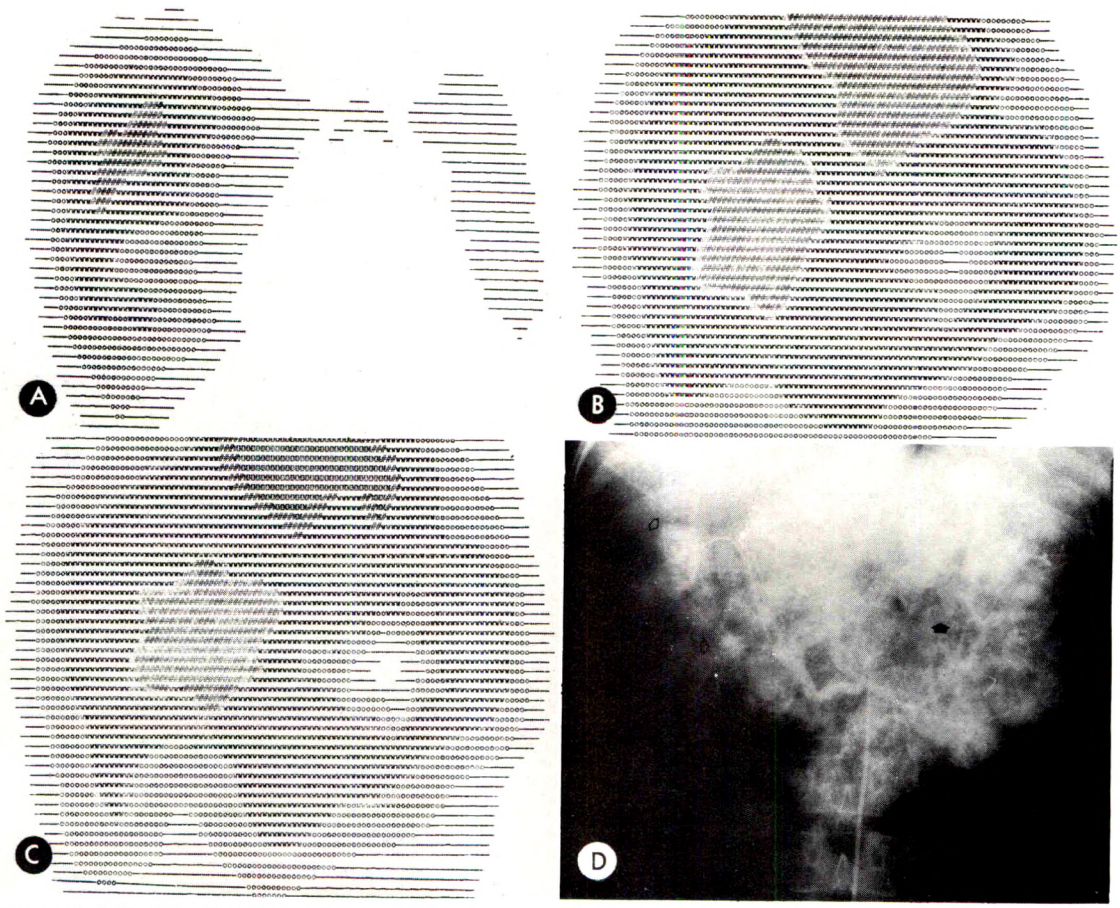


FIG. 7. (*A*) Radiocolloid scan showing multiple concentration defects at the right hepatic lobe and a great filling defect in the left hepatic lobe. (*B*) Scan obtained 10 minutes after the administration of In^{113m} chloride (Blood pool), showing the persistence of one small filling defect in the center of the left hepatic lobe. (*C*) Scan obtained 6 hours later, showing the persistence of the filling defect in the center of left hepatic lobe. (*D*) Selective angiogram showing an avascular region in the center of left hepatic lobe.

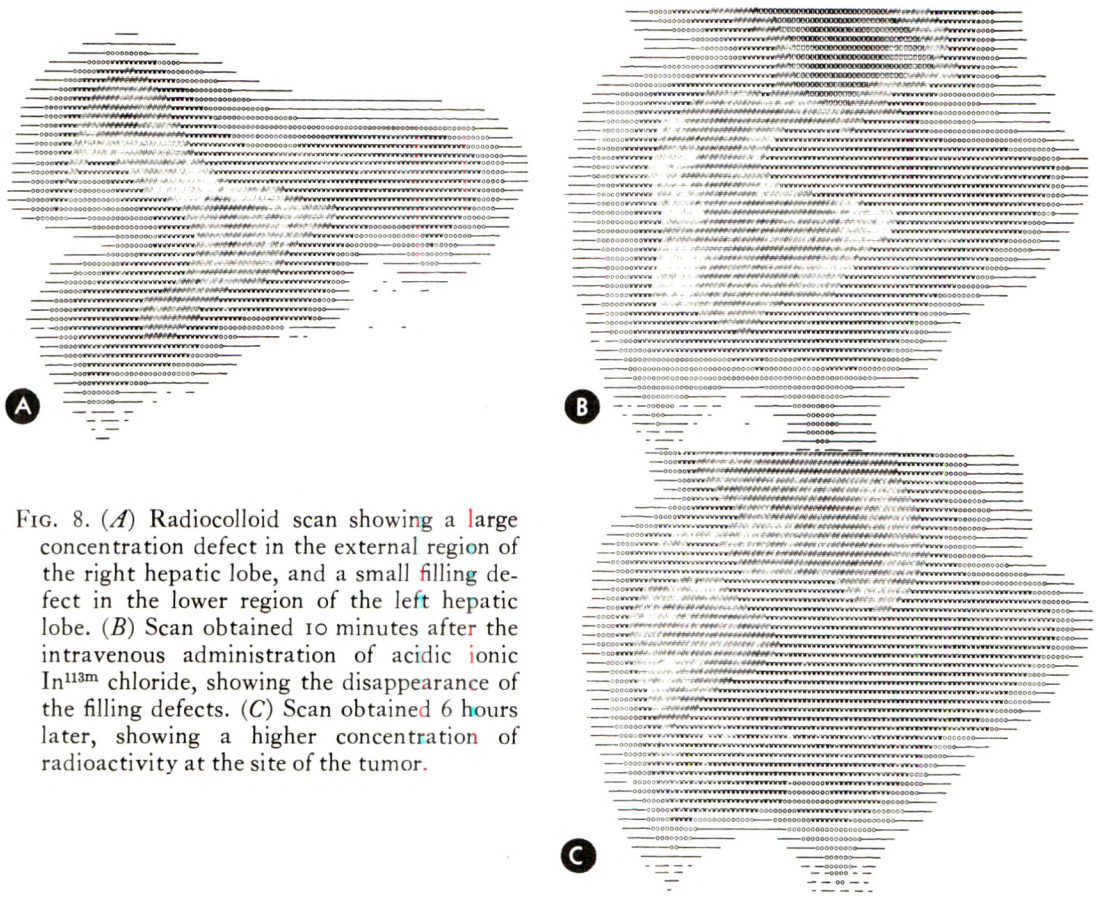


Fig. 8. (*A*) Radiocolloid scan showing a large concentration defect in the external region of the right hepatic lobe, and a small filling defect in the lower region of the left hepatic lobe. (*B*) Scan obtained 10 minutes after the intravenous administration of acidic ionic In^{113m} chloride, showing the disappearance of the filling defects. (*C*) Scan obtained 6 hours later, showing a higher concentration of radioactivity at the site of the tumor.

sions of the liver, but the information provided by this method is nonspecific, because the filling defects may be caused by an abscess, a cyst or a tumor.

Since 1969, Hosain and associates[14] have shown that carrier-free ionic In^{113m} chloride, at a pH lower than 4.0, binds as a cation to a beta globulin known as transferrin. When this condition is met, its half-life in blood is between 2 and 3.5 hours, without significant excretion in the urine. This radiopharmaceutical was used soon after its development as a label for scanning the placental blood pool,[10,16] and for imaging the cardiac blood pool for diagnosis of pericardial effusion.[14,19]

A series of encouraging studies on the use of this radiopharmaceutical as a means to differentiate liver abscesses from other space-occupying lesions of the liver have been reported by our group since 1969,[5,7,8,10–12,17] and in 1970, Lubin and associates[15] reported their results with this compound in the differential diagnosis between vascularized and nonvascularized intrahepatic lesions. More recently, Yeh *et al.*[22] wrote concerning their results by comparing radiocolloid scans with a series of sequential images that show the transit of a Tc^{99m} pertechnetate bolus through the hepatic vascular bed in 50 cases.

The use of ionic, acidic, carrier-free In^{113m} chloride is based on the fact that this radiopharmaceutical remains circulating in blood after it is bound to serum transferrin, and, thus, the hepatic vascular blood pool can be depicted by scintigraphic methods. Therefore, the degree of vascularity in a lesion can be determined when radioactivity is detected in the location of the filling defect observed in the previous scan with radiocolloids, and when this radio-

activity is compared with that in the normal surrounding tissue.[5,11,12]

Precise diagnosis of acute amebic abscess can be made with this combined method because there is not a blood pool in the interior of the abscess; therefore, the filling defect persists in the image obtained a few minutes after the administration of acidic In^{113m} chloride. This type of lesion can be differentiated from a cyst because in acute amebic abscess there is an increased radioactivity at the periphery of the lesion due to an increased vascularity of the hepatic tissue around the abscess,[2,5,11,12,20] while in cases of hepatic cysts, the radioactivity at the periphery of the lesion is rather low due to a poor vascularity around the cyst as a consequence of elongated and diminished blood vessels.[5,11,12] On the other hand, distinction between a cyst and a chronic abscess is rather difficult, because there is no increased vascularity at the periphery of the chronic abscess.[5,11,12]

In 72 per cent of primary carcinomata and in 70 per cent of metastatic tumors studied in our previous reports,[5,11,12] the increase of radioactivity in the interior of the filling defects generally was noteworthy, but did not reach the level of radioactivity in the normal surrounding hepatic tissue. This amount of radioactivity seems to be directly proportional to the degree of vascularity in the tumor, which explains why, in cases of tumors with a poor vascularity, there is no radioactivity detectable in the location of the lesions. There is an extraordinary increase of radioactivity in the interior of the lesion in highly vascularized masses, such as hemangiomata, and the amount of radioactivity is considerably greater than in cases of malignant tumors.[5,11,12]

Thus, this combination of radiocolloid scans with the exploration of the hepatic blood pool with early scanning after the administration of acidic In^{113m} chloride, proved to be more precise than conventional scanning with radiocolloids in the imaging of the actual size, shape, position and number of acute amebic abscesses, and helped to obtain a differential diagnosis of the etiology of the "cold" areas in all cases of cysts, in cases of hemangioma, in 96 per cent of acute amebic abscesses, in 72 per cent of primary hepatic carcinomata and in 70 per cent of liver metastases.[5,11,12] Nevertheless, there still remained the problem of differentiating 30 per cent of hepatic tumors, in which their poor vascularity rendered negative results. We therefore decided to use Ga^{67} citrate for a few months, but soon were discouraged by the fact that this radiopharmaceutical was also concentrated by an important fraction of the inflammatory lesions studied.[9]

In 1972, Hor and associates[13] reported experimental evidence of the active concentration of acidic, ionic, carrier-free In^{113m} chloride by the liver of pregnant and virgin rats. This information encouraged our group to study the possible concentration of this radiopharmaceutical by hepatic tumors, and its potential as an aid for the differential diagnosis of liver masses.

Our present results seem to suggest that the radiopharmaceutical is slowly liberated from its bonds to serum transferrin, and concentrated by normal and tumoral hepatic tissue. Six hours after the intravenous administration of In^{113m} chloride we found a higher retention of radioactivity in the "normal" areas of the liver than at the cardiac blood pool, and an even larger retention at the site of the tumor (Fig. 1). Ten minutes after the administration of the radioactive tracer, the radioactive level was higher in the "normal" hepatic region than in the tumor (Fig. 2; and 6–8), but 6 hours later the differences of the radioactivity between these 2 regions, and that at the cardiac blood pool, were smaller due to the faster disappearance rate of the radiopharmaceutical from the "normal" hepatic tissue than from the tumor.

On the radiocolloid scans, liver tumors appear as concentration defects due to the absence of functioning reticuloendothelial tissue able to phagocytize the colloidal particles. In these cases, the ratio obtained by dividing the tumor radioactivity by the

radioactivity found in the "normal" hepatic region is rather low (0.09 ± 0.05) (Fig. 3). This ratio is higher (0.51 ± 0.16) 10 minutes after the administration of the radioactive agent due to the presence of a blood pool in the interior of the tumor. This ratio seems to be related to the degree of vascularity and blood flow into the tumor. Six hours later, this ratio increases to 0.76 ± 0.26, which seems to be evidence of a more active concentration of the agent by the tumor than by the "normal" liver tissue (Fig. 3). It seems that the degree of increase of this ratio, as a function of time, depends directly on the degree of vascularity and blood flow into the tumor, as there exists a highly significant positive correlation between this ratio at 6 hours and the ratio obtained 10 minutes after the administration of the tracer, which is dependent on vascularity and blood flow (Fig. 4). This seems to be logical, as the radionuclide has to reach the tumor to be concentrated. These evidences are strengthened by the fact that the tumor concentration ratio $(T_6/L_6)/(T_{10}/L_{10})$ is also dependent on the Tumor/Liver ratio at 10 minutes, and thus, it is also dependent on the degree of vascularity and blood flow in the tumor (Fig. 5).

The concentration mechanism of In^{113m} chloride by normal hepatic tissue and by liver tumors is still unknown. It may be due to a rather slow but continuous replacement of In^{113m} chloride molecules by iron molecules at the serum transferrin binding sites, as this beta globulin may have a higher affinity for iron than for indium. The individual differences in the rate of hepatic and tumoral concentration of In^{113m} chloride, may be explained by individual differences in transferrin free binding capacity. Once the radiopharmaceutical is liberated from its bonds to serum transferrin, it is able to be concentrated, probably by the iron storage sites. Hor *et al.*[18] have shown that this radiopharmaceutical is slowly concentrated by liver, spleen and bone—the most important sites for iron storage.

With the scintigraphic images of the hepatic blood pool, obtained 10 minutes after the intravenous administration of acidic In^{113m} chloride, we were able to reach a differential diagnosis in 40.9 per cent of cases, and the results were negative in 4.5 per cent of cases (Table 1). These results are in contrast with those obtained 6 hours later With these delayed images, we were able to reach a precise diagnosis about the nature of the lesion in 86.4 per cent of cases, the results were in doubt in 0.1 per cent of cases, and were negative in 4.5 per cent of cases (Table 1).

The diagnostic efficiency of this procedure seems to be higher than the use of the so called "tumor seeking" agents such as Ga^{67} citrate. Even though, it needs the administration of 2 different radiopharmaceuticals: 2 millicuries of In^{113m} Fe microcoprecipitates, for scanning the reticuloendothelial tissue distribution, and 10 millicuries of ionic, acidic, carrier-free In^{113m} chloride for the scanning of the hepatic blood pool distribution and the positive imaging of the tumor. Furthermore, it requires the obtaining of a series of 3 different images, which can be tiresome for the patient. Nonetheless, we rather prefer to expose the patient to a higher radiation dose and reach a definite diagnosis, than to save the patient of this burden and leave the physician in doubt.

CONCLUSION

We may conclude that liver scintigraphy with radiocolloids can be considered a routine method for the localization of intrahepatic lesions, and that liver scanning of the distribution of hepatic blood pool with ionic, acidic, carrier-free In^{113m} chloride may be used optionally to obtain more precise information on the degree of vascularity and blood flow in the interior of the lesion, thereby improving the probability of reaching a differential diagnosis between tumors and other space-occupying lesions of the liver.

In case of remaining doubts, the scan may be repeated 6 hours later in order to

depict the positive concentration of the radiopharmaceutical at the site of the tumor.

Meanwhile, it is of importance to obtain more data about the behavior of this radiopharmaceutical in other types of space-occupying lesions of the liver, such as abscesses, cysts and cirrhotic regenerative nodules.

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REFERENCES

1. ADATEPE, M. H., WELCH, M., EVENS, R. G., and POTCHEN, J. Clinical application of broad spectrum scanning agent: indium 113m. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *112*, 701–706.
2. AGUIRRE, J. Peculiaridades histopatóligicas de la lesión amibiana. *Arch. Inv. Med.* (Méx.), 1970, *1*, Suppl. 1, 147–156.
3. COLOMBETTI, L. G., GOODWIN, D. A., and HINKLEY, R. L. Preparation and testing of sterile Sn^{113}-In^{113m} generator. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *106*, 745–749.
4. CUARÓN, A., and GORDON, F. Liver scanning: analysis of 2,500 cases of amebic hepatic abscesses. *J. Nuclear Med.*, 1970, *9*, 435–439.
5. CUARÓN, A., GORDON, F., MUÑOZ, J. R., and LANDA, L. Scanning of hepatic blood pool in liver amebiasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *117*, 373–379.
6. CUARÓN, A., GORDON, F., and MUÑOZ, J. R. ^{113m}Indium: most versatile radionuclide for liver scanning. First Scientific Assembly, World's Federation of Societies of Nuclear Medicine and Biology, Los Angeles, 1971.
7. CUARÓN, A., and GORDON, F. Evolución de la centelleografía hepática en el diagnóstico del absceso ambiano del hígado. *Arch. Inv. Med.* (Méx.), 1972, Suppl. 2, 403–409.
8. CUARÓN, A., TREVIÑO, H., MUÑOZ, J. R., SOTOMAYOR, J. M., and GORDON, F. Avances en la técnica centelleográfica. In: Memorias del I Congreso Americano de Medicina de la Seguridad Social. Instituto Mexicano del Seguro Social Ed., 1969, 327–328.
9. CUARÓN, A., and GORDON, F. Unpublished data.
10. GORDON, F., and CUARÓN, A. Avances en el estudio centelleográfico del absceso hepático amibiano. *Arch. Inv. Med.* (Méx.), 1971, *1*, Suppl. 2, 415–420.
11. GORDON, F., CUARÓN, A., MUÑOZ, J. R., and LANDA, L. Scanning of hepatic blood pool in differential diagnosis of space-occupying lesions of liver. *Ann. Int. Med.*, 1973, *78*, 247–250.
12. GORDON, F., CUARÓN, A., MUÑOZ, J. R., and LANDA, L. Comparison of colloidal and ionic ^{113m}In in study of liver tumors. *Radiology*, 1973, *108*, 359–361.
13. HOR, G., KRIEGEL, H., and SCHRAM, E. Biologisches Verhalten von Radioindium (^{113m}In, ^{114}Ir) bei virginellen und graviden Ratten. *Int. J. Appl. Rad. & Isotopes*, 1972, *23*, 595–602.
14. HOSAIN, F., MCINTYRE, P. A., POULOSE, K., STERN, H. S., and WAGNER, H. N., JR. Binding of tracer amounts of ionic indium-113m to plasma transferrin. *Cli. chim. acta*, 1969, *24*, 69–75.
15. LUBIN, E., LAOR, J., SHIMEONI, A., LEWITUS, Z., PICK, A. I., KADISH, M., DINTSMAN, M., and GALTI, I. Two-stage scintiscanning in differential diagnosis of vascularized and non-vascularized intrahepatic space-occupying lesions. *J. Nuclear Med.*, 1970, *9*, 17–24.
16. MUÑOZ, J. R., SOTOMAYOR, J. M., GORDON, F., and CUARÓN, A. Centelleografía de la placenta con ^{113m}Indio. In: Memorias de la III Reunión Anual de la Sociedad Mexicana de Medicina Nuclear. Sociedad Mexicana de Medicina Nuclear Ed., 1969, 167–173.
17. MUÑOZ, J. R., SOTOMAYOR, J. M., TREJO, G., GORDON, F., and CUARÓN, A. Versatilidad del ^{113m}In como radionúclido para la exploración centelleográfica del hígado. (Abstr.) *Rev. Biol. Med. Nuclear*, 1970, *2*, 186.
18. OSORIO, A., and TOUYA, J. J. La gammagrafía en la hidatidosis hepática del niño. *Arch. Pediat.* Uruguay, 1968, *39*, 189–193.
19. POTCHEN, F. J., ADATEPE, M. H., WELCH, M., ARCHER, E., and STUDER, R. Indium-113m for visualization of body organs: broad-spectrum scanning agent. *J.A.M.A.*, 1968, *205*, 208–212.
20. STOOPEN, M., FLORES IZQUIERDO, G., and LANDA, L. Angiografía selectiva del tronco celíaco en el estudio del absceso hepático. *Rev. Gastroenterol. Méx.*, 1969, *34*, 471–482.
21. WAGNER, H. N., JR., MCAFEE, J. G., and MOSLEY, J. M. Diagnosis of liver disease by radioisotope scanning. *A.M.A. Arch. Int. Med.*, 1961, *197*, 324–334.
22. YEH, S. H., SHIH, W. J., and LIANG, J. C. Intravenous radionuclide perfusion hepatography combined with ^{99m}Tc-S colloid liver scintiphotography in diagnosis of hepatic neoplasms and benign diseases. *J. Nuclear Med.*, 1972, *13*, 480–481.

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USE OF SELECTIVE, INTRAHEPATIC, PORTAL VENOGRAPHY AND *IN VIVO* COLORATION IN PLANNING SEGMENTAL HEPATIC RESECTION*

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SEGMENTAL resection of liver for small, solitary, hepatic tumors or abscesses has been described.[2] Because segmental anatomy of the human liver is not apparent from inspection of the surface, segmental resection would be facilitated by a technique that outlines the segment for the surgeon. We used selective, intrahepatic, portal venography to identify angiographically and color selectively, by instillation of a vital dye, a specific hepatic segment.[4,5] Selective staining of other tissues with vital dyes has been performed,[6,14] but vital staining of hepatic segments by way of the portal vein has not been reported.

MATERIAL AND METHOD

Experimental Animals. Selective, intrahepatic, portal venography, coloration, and segmental resection of the liver were performed in 1 newborn lamb and 5 adult dogs. The lamb liver, like human liver, is unlobulated. The newborn lamb has an umbilical vein, which allows access to the intrahepatic portal venous system. Because newborn lambs were difficult to obtain, however, most of our study was performed in dogs. The dog liver, unlike the human liver, is distinctly lobulated into 2 major portions, each containing 2 lobes.[9] The adult dog does not have an umbilical vein, but its pancreatic vein enters the extrahepatic portal vein, allowing access to the intrahepatic portal venous system for selective catheterization.

Technique of Portal Venography. In the lamb, the umbilical vein was cannulated extraperitoneally; in dogs, the pancreatic vein was cannulated through an abdominal incision. A clear, polyethylene catheter* was placed into the vein to act as a sheath through which a directional side-hole catheter† was placed. Under fluoroscopic control, the catheter tip was guided through the common portal vein and into a branch of the portal vein. Ten to 20 ml. of Renografin-76‡ was injected by hand as a single-push dose. A selective venogram was then obtained. Ten ml. of 1 per cent solution of sterile toluidine blue dye was then injected slowly (30 sec.) by hand through the catheter into the liver segment. In 2 dogs (Dogs 1 and 2) and the lamb, the abdomen was opened within 30 minutes after injection of the dye and the appropriate liver segment identified by noting the coloration on its surface. Glisson's capsule was incised anteriorly along the base of the colored segment and a finger-fracture technique (digitoclysis) was used to disrupt the liver parenchyma. The segment was then removed, omentum placed against the raw surface of the liver, and the abdomen closed. One dog (Dog 1) and the lamb were sacrificed immediately after the surgical procedure. Twenty-four hours after the surgical procedure, repeat portal venograms were obtained on the other dog (Dog 2) using the indwelling

* P.E. 240 (I.D. .066 inches, O.D. .095 inches), manufactured by Clay-Adams Division, Becton Dickinson, Parseppany, New Jersey 07054.

† V/5/65, 5 French (tip I.D. .029 inches), manufactured by Medi-tech Corp., Watertown, Massachusetts 02172.

‡ Meglumine diatrizoate (66 per cent) and sodium diatrizoate (10 per cent) injection, manufactured by E. R. Squibb and Sons, Princeton, New Jersey 08540.

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TABLE I

SERUM ENZYME VALUES IN 5 DOGS BEFORE AND AFTER INJECTION OF 10 ML. OF 1 PER CENT TOLUIDINE BLUE DYE INTO AN INTRAHEPATIC PORTAL VEIN

Dog	SGOT (IU/L)		SGPT (IU/L)		LDG (IU/L)	
	Before	After	Before	After	Before	After
1	40	225*	15	220*	135	885*
2	35	315*	30	210*	70	460*
3	12.5	15	10	15	175	200
4	75	25	55	30	155	100
5	40	40	20	25	320	320

* These values are elevated because resection of the colored segment was performed before blood was drawn.
SGOT = serum glutamic oxaloacetic transaminase;
SGPT = serum glutamic pyruvic transaminase;
LDH = lactate dehydrogenase.

sheath in the pancreatic vein; the dog was then sacrificed.

The remaining 3 of the 5 dogs (Dogs 3, 4, and 5) did not undergo the surgical excision: 1 (Dog 3) was sacrificed immediately after venography; 1 (Dog 4) was sacrificed 24 hours later; and another (Dog 5) was sacrificed 48 hours after portal venography and segmental coloration.

Histologic Studies and Liver Function Tests. Histologic studies of the liver were performed on all the animals to locate the dye and ascertain hepatocellular damage. Liver tissue from the segments to be selectively injected was obtained by means of needle biopsy before venography and toluidine blue dye injection. The resected liver segments of Dogs 1 and 2 and wedge biopsy specimens from the colored segments of Dogs 3, 4, and 5 were then examined by light and electron microscopy.

In addition to the histologic studies performed on the colored liver segments, other organs were to be microscopically examined to determine the possible presence of the dye outside the liver. We, therefore, again injected Dog 5 with toluidine blue dye 48 hours after the initial injection. The dog was then sacrificed and sections of parathyroid, thyroid, spleen, lung, liver, kidney, pancreas, and heart were immediately obtained.

Liver function tests were performed before and immediately after the injections of contrast material and toluidine blue dye in all the dogs (Tables 1 and 11). Central portal vein pressures had been measured before injection of contrast material and

TABLE II

LIVER FUNCTION TESTS IN 5 DOGS BEFORE AND AFTER INJECTION OF 10 ML. OF 1 PER CENT TOLUIDINE BLUE DYE INTO AN INTRAHEPATIC PORTAL VEIN

Dog	Alkaline Phosphatase (IU/L)		Protein (g./dl.)		Albumin to Globulin Ratio		Prothrombin Time (sec.)	
	Before	After	Before	After	Before	After	Before	After
1	53	57	6.1	5.2	2.5/3.7	2.0/3.2	7.2	9.4
2	38	44	5.8	5.6	2.4/3.4	2.3/3.3	7.0	7.2
3	25	19	6.2	5.7	2.0/4.2	1.9/3.8	6.9	6.9
4	61	61	5.3	5.6	2.1/3.2	2.4/3.2	7.3	7.1
5	32	25	7.8	6.8	1.4/6.4	1.2/5.6	7.5	7.0

Table III

PORTAL PRESSURE (MM. HG) IN 3 DOGS BEFORE AND AFTER INJECTION OF TOLUIDINE BLUE DYE INTO A BRANCH OF THE PORTAL VEIN

Dog	Before Injection	Immediately after Injection	12 Hours after Injection	24 Hours after Injection	48 Hours after Injection
3	12	15	—	—	—
4	16	15	12	7	—*
5	11	12	16	16	10

* Not measured.

toluidine blue dye and at the conclusion of the study (in Dogs 3, 4, and 5) (Table III).

Because cardiac arrhythmias have been reported to occur after injection of toluidine blue dye,[6,12] electrocardiograms were obtained before, during, and after the initial and the 48 hour dye injection in Dog 5.

RESULTS

Selective venography was easily accomplished in all the animals. With this technique we were able to opacify the single portal vein branch supplying its individual segment, as well as produce a parenchymogram of that segment (Fig. 1). In addition, the venous drainage of the hepatic segment was visualized (Fig. 2*A*).

The coloration of hepatic segments by toluidine blue dye clearly outlined the boundaries of that segment in each case (Fig. 2, *B* and *C*). In 2 dogs, however, in addition to the segmental coloration, small patches of blue coloration were noted on the surface of other segments of the liver. We found that, in these animals, the catheter had become displaced slightly from the segmental vein during injection; the patches of coloration were most likely due to leakage of the dye into the main portal venous system. Nevertheless, the patches did not interfere with the clear identification of the colored segment.

Surgical extirpation of the colored segment in the lamb and 2 dogs (Dogs 1 and 2) was performed without difficulty. Blood vessels and bile ducts were easily identified, divided, and ligated. In every instance, the colored segment was supplied by a single branch of the portal vein in the portal triad and drained by a single hepatic vein (Fig. 1; and 2*A*). The segmentectomy took less than 20 minutes. The postoperative portal venogram compared to the preoperative venogram showed absence of the single segmental portal vein (Fig. 3, *A* and *B*).

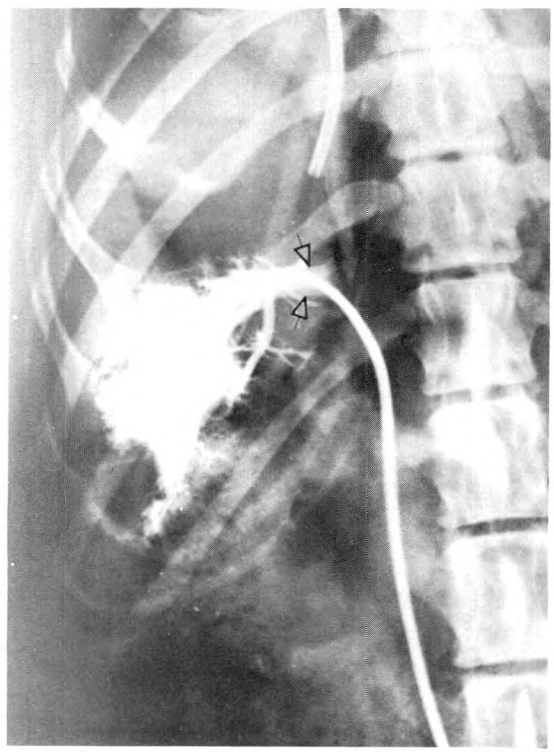


Fig. 1. Selective portal venogram in a dog demonstrates a portal vein branch (arrows) supplying a well-defined, hepatic segment.

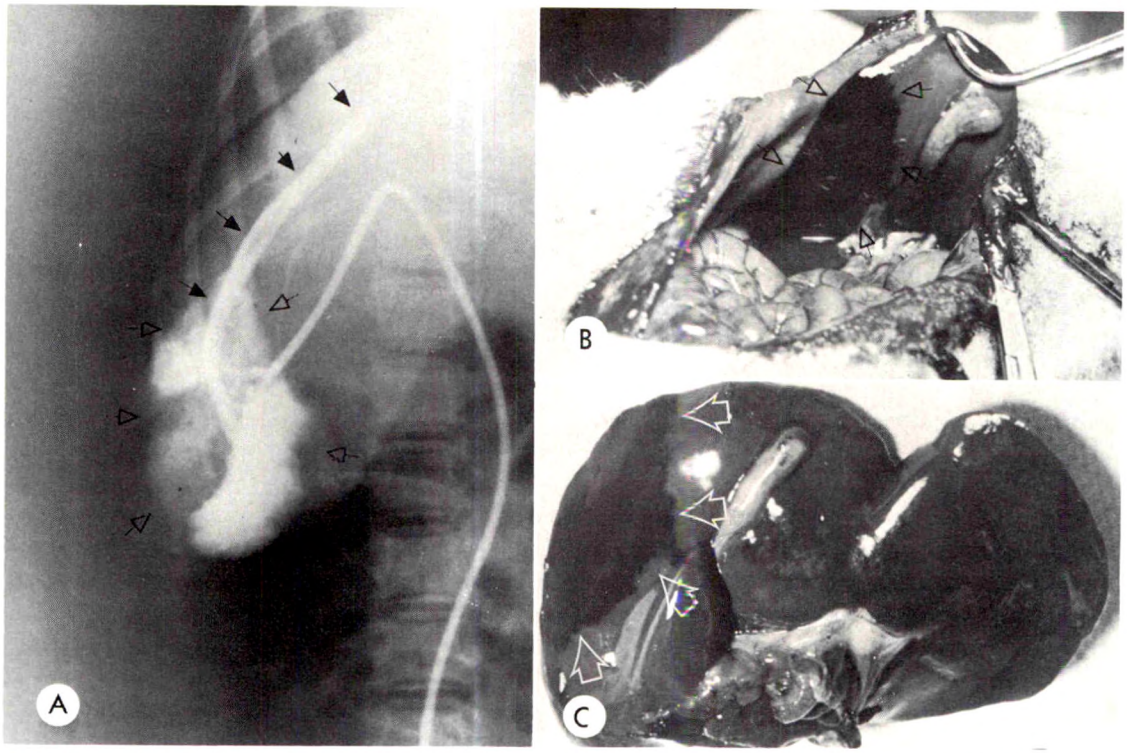


FIG. 2. (*A*) Selective umbilicoportal venogram in the lamb illustrates a single hepatic vein (closed arrows) draining an hepatic segment. The dense blush of contrast corresponds to sinusoidal and venule collections of contrast material in the segment. The segment outlined by open arrows does not have a homogeneous density because the catheter was wedged distal to the take-off of some small subsegmental branches. Enough venous flow was maintained, however, to permit homogeneous coloration of the entire segment by toluidine blue dye. (*B*) Selective coloration with toluidine blue dye of the right inferior hepatic segment in the lamb (arrows). (*C*) When the liver was removed, the full extent of hepatic segment coloration was easily recognized (arrows).

In all the animals, no change in liver function was noted immediately after the injection of contrast material and toluidine blue dye (Tables I and II). In the 2 dogs (Dogs 1 and 2) which underwent segmentectomy, the levels of serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), and lactate dehydrogenase (LDH) increased after the surgical procedure, as would be expected; results of the other liver function tests did not change (Table I). The 2 dogs (Dogs 4 and 5) followed for 12, 24, and 48 hours showed no change in liver function.

Portal venous pressure obtained immediately after injection of contrast medium and dye by means of the portal vein catheter showed minimal change, when compared to control values, in Dogs 3, 4, and 5. A slight rise in portal venous pressure of 4 mm. Hg was noted after 12 hours in 1 dog (Dog 5), but by 48 hours the portal venous pressure had returned to the control value (Table III).

Histologic examination of the segment specimens of all the animals demonstrated no abnormalities in hepatocytes or reticuloendothelial cells after the injection of toluidine blue dye. Liver architecture was generally normal. The cytoplasmic-nuclear ratio of hepatocytes was normal, as were the Kupffer cells. With normal hematoxylin-eosin staining, the blue dye could not be identified. Electron microscopic examination of the liver sections revealed no abnormalities in the nucleus, cytoplasm, or cell membrane.

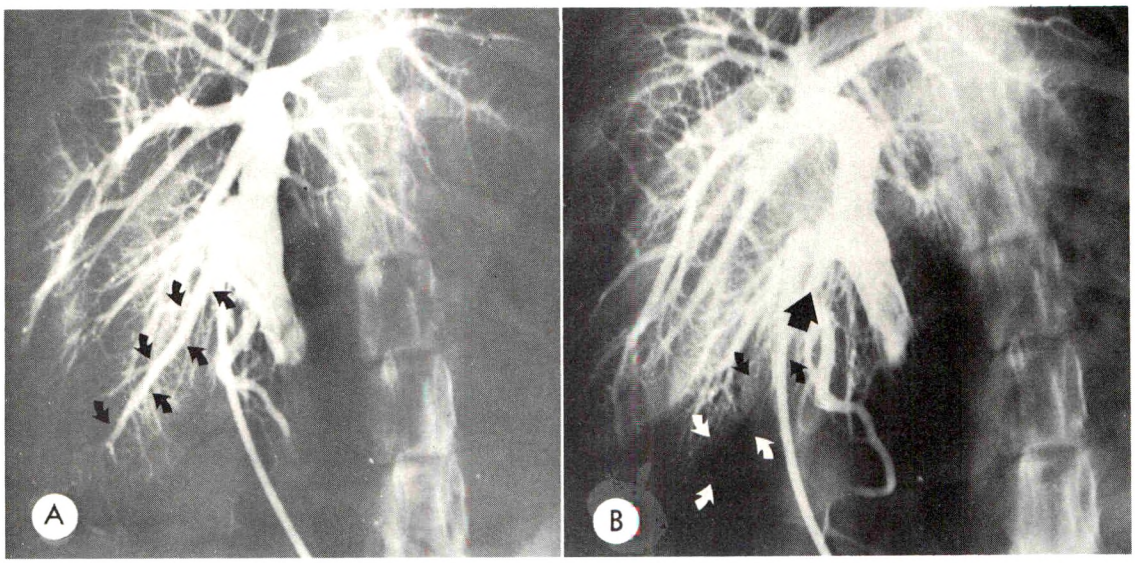


FIG. 3. (*A*) A preoperative, total portal venogram in a dog shows a single vein into which toluidine blue dye was selectively injected to obtain the boundaries of the segment (arrows). (*B*) The postoperative venogram demonstrates the stump of the segmental portal vein (large arrow). No leakage of contrast material into the liver capsule or abdomen was noted. Small arrows outline the space previously occupied by the removed segment.

Light microscopic study of hematoxylin-eosin stained sections of kidney, thyroid, and parathyroid, spleen, lung, pancreas, heart, and lymph nodes (Dog 5) showed no evidence of histologic abnormalities. Toluidine blue dye could not be identified in the cells or interstitium of these tissues.

The electrocardiograms obtained of Dog 5 before, during, and after the injection of dye showed no abnormal cardiac rhythm.

DISCUSSION

Portal venography with the use of umbilical vein catheterization has become an established method for studying portal venous circulation.[1,3,5,7,8,10,11] This method allows access to the portal venous circulation for measuring portal venous pressure, obtaining portal venous blood samples, and demonstrating portal venous anatomy.

Use of selective umbilicoportography permits evaluation of individual hepatic segments and of hepatic venous drainage from each of these segments. It seemed reasonable that this method would lend itself to segmental coloration to act as an anatomic guide for surgical segmentectomy. Ordinarily, the surgical treatment for primary hepatic neoplasms, single metastasis, or abscesses is lobar resection. If a lesion is confined to 1 segment, however, that segment alone could be removed.[2] Although hepatic arteriography and venography are used to locate lesions in the liver parenchyma, portal venography can also locate lesions, and it is the only method of defining the portal venous supply to a segment and hepatic venous drainage from a segment.

The use of vital dyes injected intravenously or interarterially to identify a specific lesion is not new. Previous investigators have utilized this approach in patients to locate pancreatic and parathyroid adenomas and gastric lesions.[6,12,13] We chose toluidine blue dye because it stains the liver segment a dense blue-black, which is easily discernible from surrounding hepatic tissue. Toluidine blue did not alter liver morphology or function in this study.

Yamakawa *et al.*[14] have noted cardiac arrhythmias in rats given a 2 per cent solution of toluidine blue dye at a dose of 20 mg./kg. These were mainly ventricular arrhythmias and were associated with visible cyanosis. When toluidine blue dye

was injected intravenously at 5 or 10 mg./kg. in a ½ to 1 per cent solution, no arrhythmias were noted. On the other hand, Keaveny and co-workers,[6] studying dogs and rats injected with 1 per cent toluidine blue dye infused at a dose of 10 mg./kg., found no toxic effects in any of their animals and, in particular, no evidence of cardiac arrhythmias.

It is important to note that we administered a 1 per cent solution of toluidine blue dye as a single intravenous bolus at a dose of approximately ½ mg./kg.—far below the levels used by Yamakawa *et al.*[14] in rats exhibiting arrhythmias. We encountered no cardiac arrhythmia. The rare complication of methemoglobinemia as a result of intravenous injection of toluidine blue dye has been reported in a patient with glucose 6-phosphate dehydrogenase deficiency.[13] If selective toluidine blue coloration of liver segments is to be used in humans, this possible complication must be kept in mind. Toluidine blue dye is excreted through the gastrointestinal tract[14] and the urinary tract; we noted a blue-green color to the urine beginning 2 hours after injection and lasting for approximately 48 hours. Histologic examination of the kidneys after 48 hours showed no evidence of cell damage or toluidine dye within the kidney cells or interstitium.

SUMMARY

Selective intrahepatic portal venography was performed in 5 dogs and 1 newborn lamb so that toluidine blue dye could be injected into an hepatic segment to permit visual identification of its boundary.

The venogram and coloration permitted successful segmental resection, because the vascular supply and drainage, as well as surface boundaries, were easily identified. Portal venography and instillation of the dye into a branch of the portal vein did not result in any histologic changes or change in liver function or portal pressure.

The technique of selective intrahepatic portal venography and segmental coloration by way of umbilical vein catheterization can be useful to the surgeon when performing a segmental hepatic resection.

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REFERENCES

1. CHIANDUSS, L. Umbilico-portal catheterization in diagnosis of liver disease. *Prog. Liver Dis.*, 1970, *3*, 466–480.
2. COUINAUD, C. Le Foie: Études Anatomiques et Chirurgicales. Masson et Cie, Paris, 1957, p. 530.
3. DELVICNE, J., JACQUET, N., and COPS, A. La portographie transombilicale: intérêt des injections sélectives et occlusives. *Ann. de radiol.*, 1971, *14*, 373–393.
4. GROSS, G., and RAUTUREAU, J. La segmentographie hépatique par voie ombilicale. *M.C.D.*, 1972, *1*, 15–20.
5. GROSS, G., ROUCAYROL, R., CADY, J., RAUTUREAU, J., and MERCADIER, M. La segmentographie hépatique par voie ombilicale. *Chirurgie*, 1971, *97*, 585–587.
6. KEAVENY, T. V., FITZGERALD, P. A., and MCMULLIN, J. P. Selective parathyroid and pancreatic staining. *Brit. J. Surg.*, 1969, *56*, 595–597.
7. KESSLER, R. E., TICE, D. A., and ZIMMON, D. S. Value, complications, and limitations of umbilical vein catheterization. *Surg., Gynec. & Obst.* 1973, *136*, 529–535.
8. LAVOIE, P., LEGARE, A., and VIALLET, A. Le cathétérisme portal trans-ombilical (une étude de 120 omphalo-portographies). *Rev. Internat. d'hépatol.*, 1967, *17*, 753–768.
9. MILLER, M. E., CHRISTENSEN, G. C., and EVANS, H. E. Anatomy of the Dog. W. B. Saunders Company, Philadelphia, 1964, p. 702.
10. MOORE, G. E., and BRIDENBAUGH, R. B. Portal venography. *Surgery*, 1950, *28*, 827–831.
11. NAHMAD, M. H., LANKAU, C. A., DEL GUERCIO, L. R. M., and MENG, C-H. Transumbilical cholangiography and selective venous portography in patients with obstructive jaundice. *S. Forum*, 1968, *19*, 338–339.
12. RÖHER, H. D., and TREDE, M. Intraoperative Epithelkörperchenlokalisation durch Vitalfärbung mittels Toluidinblau-O. *Chirurg*, 1972, *43*, 274–277.
13. TEUNIS, B. S., LEFTWICH, E. I., and PIERCE, L. E. Acute methemoglobinemia and hemolytic anemia due to toluidine blue. *A.M.A. Arch. Surg.*, 1970, *101*, 527–531.
14. YAMAKAWA, T., von HOFE, E., KAGAN, R., and MORGENSTERN, L. Use of tolonium in diagnosis of malignant gastric lesions. *A.M.A. Arch. Surg.*, 1972, *104*, 773–777.

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EFFECT OF CHOLESTYRAMINE ON TELEPAQUE ORAL CHOLECYSTOGRAPHY*

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DURING the evaluation of a 27 year old man with postgastrectomy syndrome, an oral cholecystogram with Telepaque was ordered, while the patient was receiving cholestyramine for endoscopically diagnosed "alkaline gastritis."[1] The appearance of the gallbladder was markedly abnormal (Fig. 1*A*). Because of the odd appearance of the gallbladder on the initial study, and since the effect of cholestyramine on Telepaque cholecystography was not known, the oral cholecystogram was repeated following withdrawal from cholestyramine for 1 week. This resulted in excellent visualization of a normal appearing gallbladder (Fig. 1*B*).

IN VITRO STUDIES

To evaluate the possible direct effect of cholestyramine on Telepaque (iopanoic acid), the following *in vitro* studies were performed. An isosmolar solution of iopanoate buffered with 50 mM. $NaCO_3$ at pH 9.5 was separated into three 30 ml. aliquots. The pH was adjusted in 2 of the aliquots by titrating with 1 *N* HCl, resulting in iopanoic acid suspensions of pH 7.5 and pH 6.8. Two ml. of each solution or suspension was filtered into small vials for iodine concentration measurements. A total of 1 gm. cholestyramine* was then added to the remaining 28 ml. of iopanoate solution or suspension. This mixture was intermittently stirred at room temperature for 15 minutes and then filtered for determination of iodine concentration in the filtrate. Iodine concentrations were determined as previously reported.[3]

Table 1 reveals the marked reduction in iodine concentration brought about by *in vitro* mixing of cholestyramine with iopanoic acid suspensions and an iopanoate solution. No significant pH change occurred following cholestyramine treatment. This indicates that cholestyramine has an apparent high affinity for Telepaque (iopanoic acid).

DISCUSSION

The collapsed appearance of the gallbladder in Figure 1*A* is puzzling. A decrease in bile flow with bile acid depletion caused by cholestyramine might be postulated, but increased bile acid synthesis should compensate for increased losses in most instances.[2]

Patients who receive cholestyramine for bile acid diarrhea also have an increased incidence of gallstones.[2] Abnormal oral cholecystography in such patients who are taking cholestyramine should be interpreted with caution. Although Ware and Combes[5] have indicated that *in vitro* cholestyramine binding of a drug does not necessarily decrease intestinal absorption, the experience in the patient described above indicates that cholestyramine binding of iopanoate may be of diagnostic significance. Recent investigation of simultaneous administration of cholestyramine and iopanoic acid to dogs reveals nonvisualization in dogs that visualized normally on iopanoic acid alone (personal communication, Dr. Robert N. Berk).

To avoid iatrogenic nonvisualization or poor visualization, oral cholecystography should be performed after cholestyramine has been discontinued long enough for complete evacuation from at least the small bowel.[4]

* Questran, 2 gm. Mead & Johnson.

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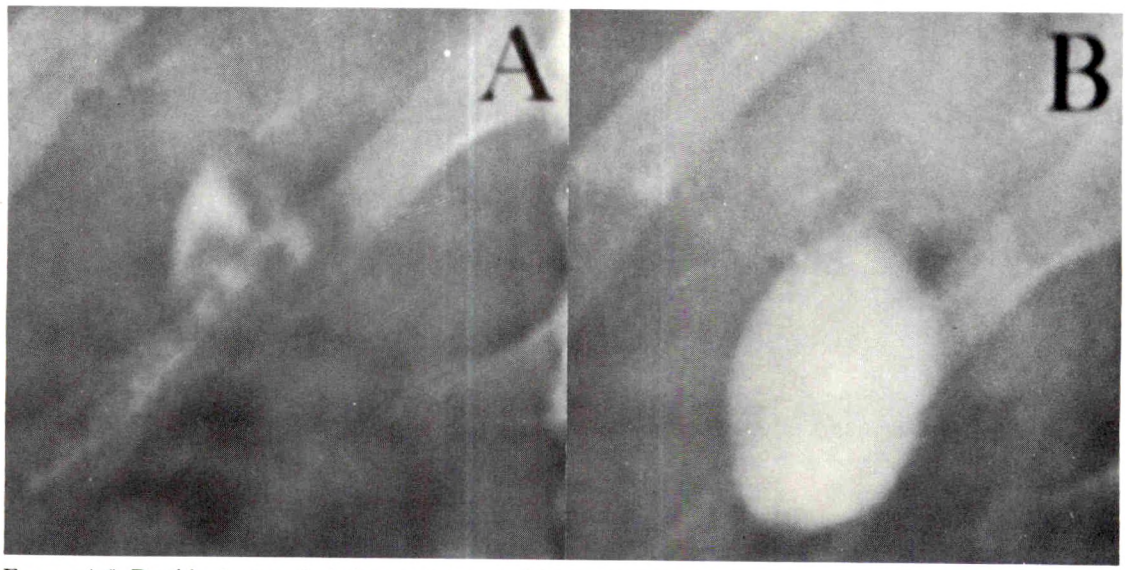


FIG. 1. (*A*) Double dose oral cholecystogram on May 17, 1973, while patient was taking cholestyramine (*B*) Single dose oral cholecystogram on June 12, 1973, following 1 week cessation of cholestyramine.

SUMMARY

A patient receiving cholestyramine underwent oral cholecystography with iopanoic acid (Telepaque) and showed evidence of an abnormal gallbladder.

When the oral cholecystography was repeated following discontinuation of cholestyramine, the study was normal.

In vitro studies demonstrate that cholestyramine has a strong affinity for iopanoate.

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TABLE I

EFFECT OF CHOLESTYRAMINE ON *In Vitro* IOPANOATE IODINE CONCENTRATION

	Control I mg./ml.	Post-cholestyramine I mg./ml.
Iopanoate solution pH 9.5	5.78	0.02
Iopanoate suspension (saturated supernatant) pH 7.5	3.37	0.02
Iopanoate suspension (saturated supernatant) pH 6.8	1.15	0.02

The author is grateful to Ms. Mittie Puckett for secretarial assistance.

REFERENCES

1. BLACK, R. B., RHODES, J., DAVIES, G. T., GRAVELLE, H., and SWEETNAM, P. Controlled clinical trail of cholestyramine in treatment of gastric ulcer. *Gastroenterology*, 1971, *61*, 821–825.
2. HOFMANN, A. F. Bile acid malabsorption caused by ileal resection. *A.M.A. Arch. Int. Med.*, 1972, *130*, 597–605.
3. KAUFMAN, L., NELSON, J., PRICE, D., SHAMES, D., and WILSON, C. J. Some applications of SI(LI) detectors to clinical problems. *IEEE Tr. Nuclear Sc.*, 1973, *20*, 402–410.
4. NELSON, J., MOSS, A. A., GOLDBERG, H. I., BENET, L. Z., and AMBERG, J. R. Gastrointestinal absorption of iopanoic acid. *Invest. Radiol.*, 1973, *8*, 1–8.
5. WARE, A. J., and COMBES, B. Influence of sodium taurocholate, cholestyramine, and mylanta on intestinal absorption of glucocorticoids in rat. *Gastroenterology*, 1973, *64*, 1150–1155.

ANGIOGRAPHY OF THE JAUNDICED PATIENT*

WITH EMPHASIS UPON THE ANGIOGRAPHIC APPEARANCE OF BILIARY DUCT DILATATION

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THE differential diagnosis of jaundice which requires surgical decompression from that in which surgery is contraindicated remains a difficult clinical problem. Selective visceral angiography may be of value in the jaundiced patient by demonstrating dilatation of the gallbladder and/or hepatic ducts when a distal obstruction is present. Lack of dilatation of these structures is a relatively strong point against surgically correctable jaundice. Specific arterial, capillary and venous abnormalities often suggest the etiology and extent of an obstructing lesion and may also indicate an intrahepatic abnormality if the jaundice is due to disease of the liver parenchyma.

In the present communication selective visceral angiograms of 61 patients who were studied for jaundice are reviewed, and the significance of positive and negative findings described. The angiographic appearance of dilated hepatic ducts with obstructive jaundice is emphasized, since the appearance and frequency of this finding have not been sufficiently stressed in the past, and because defects due to dilated hepatic ducts may be misinterpreted as hepatic metastases.

MATERIAL AND METHOD

The results of selective visceral arteriography in 61 jaundiced patients were reviewed. The specific entities and their frequency of occurrence are shown in Table I. The diagnosis in each case was confirmed by surgery, biopsy or autopsy.

Angiography in these patients usually consisted of sequential anteroposterior (AP) celiac and right posterior oblique (RPO) superior mesenteric arteriograms using 50 cc. of hypaque M 75 or renografin 76 per cent per injection with serial films obtained at a rate of 1 film per second for 6 seconds, followed by 1 film every 2 seconds for an additional 28 seconds. The examination was occasionally supplemented by oblique celiac and/or AP superior mesenteric arteriograms. Superselective hepatic arteriograms were also obtained on occasion, but were not a routine part of the examination.

The angiograms were evaluated for evidence of gallbladder and biliary duct dilatation, arterial and venous encasement, neovascularity, tumor blushes, liver metastases, cirrhosis and ascites. The gallbladder was measured in the arterial or capillary phase and the area obtained by tracing the outline to graph paper.

RESULTS

GALLBLADDER DILATATION

(Table II)

There were 40 patients with obstruction of the common bile duct who had not had cholecystectomies. Of these, gallbladder dilatation was seen in 36 (Fig. 1; 8; and 15). Gallbladders with an area greater than 35 cm.^{2} were considered distended.[37] Of the patients without cholecystectomies gallbladder distention was present in 26 of 29 patients with carcinoma of the head of the pancreas, 1 patient each with carcinoma of the papilla of Vater and lymphosarcoma involving the pancreatic head region, 3 of 4 patients with carcinoma of the common bile duct, 4 of 5 patients with choledocholithiasis, and the 2 patients with stricture of the common bile duct and impacted gallstone

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TABLE I

DIAGNOSIS AND ANGIOGRAPHIC FINDINGS IN 61 PATIENTS PRESENTING WITH JAUNDICE

	Encased Arteries						Encased Veins				New Vessels	Blush	Add. Diagnosis
	PD	GD	H	SM	S	Σ	SM	P	S	Σ			
Carcinoma of head of pancreas (30)	15	15	7	13	2	26/30	21	11	11	26/30	6	10	A=2
Carcinoma of papilla of Vater (1)	1												
Lymphosarcoma of head of pancreas (1)		1					1	1	1				
Carcinoma of gallbladder (5)	3	3	3			4/5	3	4	4	5/5	4	4	
Carcinoma of common hepatic duct (5)	1							2				1	P=1
Carcinoma of common bile duct (4)	2												P=1, Cr=1
Calculi of common bile duct (6)	1												P=1
Cirrhosis (3)													A=1, Cr=2, PH=1
Carcinoma of body of pancreas with liver metastases (1)					1				1				M

Arteriogram normal with common bile duct stricture (1), cystic duct calculus compressing common hepatic duct (1), sclerosing cholangitis (1), and hepatitis (2).

Add. Diagnosis=Additional angiographic findings; A=Ascites; Cr=Cirrhosis; P=Pancreatitis; PH=Portal hypertension; M=Liver metastases.

PD=pancreaticoduodenal; GD=gastroduodenal; H=hepatic; SM=superior mesenteric; S=splenic; P=pancreatic.

TABLE II

ANGIOGRAPHIC EVIDENCE OF DISTENTION OF GALLBLADDER AND BILIARY DUCTS IN PATIENTS WITH EXTRAHEPATIC BILIARY OBSTRUCTION

	Number	Gallbladder			Biliary Ducts	
		D	N	C	D	N
Carcinoma of head of pancreas	30	26	3	1	17	13
Carcinoma of papilla of Vater	1	1			1	
Lymphosarcoma of head of pancreas	1	1			1	
Carcinoma of gallbladder	5	2	2	1	3	2
Carcinoma of common hepatic duct	5		5		3	2
Carcinoma of common bile duct	4	3		1	3	1
Calculi of common bile duct	6	4	1	1		6
Cystic duct calculus, compression of common hepatic duct	1	1				1
Stricture of common bile duct	1			1	1	

D=Dilated; N=Normal, Small, or Not Visualized; C=Cholecystectomy.

in the cystic duct producing jaundice by compressing the common hepatic duct. Gallbladder distention was noted in 5 of 7 patients with coexistent carcinoma of the head of the pancreas and cholecystitis-cholelithiasis. In the 2 patients with carcinoma of the head of the pancreas and shrunken gallbladders due to chronic fibrotic cholecystitis, the gallbladders were not visualized on the angiograms. In 1 case in which the gallbladder was compressed by metastatic tumor, the gallbladder was not seen. When gallbladder dilatation was present no difference in intensity of staining of the wall was noted between patients with normal gallbladders and those with chronic cholecystitis.

Gallbladder distention was also present in 2 of 4 patients with carcinoma of the gallbladder, and in 1 patient with cirrhosis and gallstones, in whom no obstruction of the cystic or common bile ducts was present. The gallbladder was not distended in any of the other patients with jaundice due to liver disease or those with common hepatic duct tumors. In these patients the gallbladder was normal to small in size or not visualized.

BILIARY DUCT DILATATION

(Table II)

Dilated hepatic ducts were often present with obstruction. In the majority of cases with dilated ducts the dilatation was mild, but occasionally the distention was quite striking. Ductal dilatation was seen to involve the central and/or peripheral hepatic ducts.

Dilated ducts may appear as large circular central lucencies (Fig. 1) which in the venous phase are often seen in relationship to the bifurcation of the portal vein (Fig. 2*A*) or as branching central defects (Fig. 2, *B* and *C*; 4*B*; and 5*B*) which lie parallel to the portal vein and its branches (Fig. 2*C*; and 9*B*). Figure 3 demonstrates the appearance of dilated peripheral ducts.

When dilated, the right hepatic ducts are usually more clearly demonstrated than the left hepatic ducts. However, a dilated left hepatic duct may be seen on occasion if the course followed by the left hepatic artery is scrutinized (Fig. 5). On occasion dilatation of the common hepatic duct and even the common bile duct may be noted (Fig. 6). At times the portal vein appears elevated by a dilated common hepatic duct (Fig. 7) or neck of a distended gallbladder (Fig. 8). In 1 case a localized indentation of the inferior surface of the portal vein was considered to be related to direct pressure from a common hepatic duct tumor (Fig. 9).

The ductal defects are best demonstrated in the capillary and venous phases of the celiac arteriogram and on occasion are also noted on the venous phase of the superior mesenteric arteriogram. The defects are at times better seen on the AP, and at other times on the RPO celiac arteriogram. Occasionally after 1 or 2 previous contrast medium injections, dilated ducts are visible as defects in the liver due to prolonged hepatic parenchymal opacification.

The hepatic ducts as visualized on the angiograms were dilated in 29 of 54 patients with obstructive jaundice (53 per cent). No evidence of dilated ducts was noted in any of 7 patients with nonobstructive jaundice. Dilatation was present in 17 of 30 patients with carcinoma of the head of the pancreas, in the 2 patients with carcinoma of the papilla of Vater and lymphosarcoma of the head of the pancreas, in 3 of 5 patients with carcinoma of the gallbladder, in 3 of 5 patients with common hepatic duct tumors, and in 3 of 4 patients with common bile duct tumors. No angiographic evidence of dilated ducts was seen in any of the 6 patients with obstructing common bile duct calculi or in the patient with jaundice due to a cystic duct calculus compressing the common hepatic duct, but dilated ducts were evident in the solitary case of a common bile duct stricture (Fig. 3).

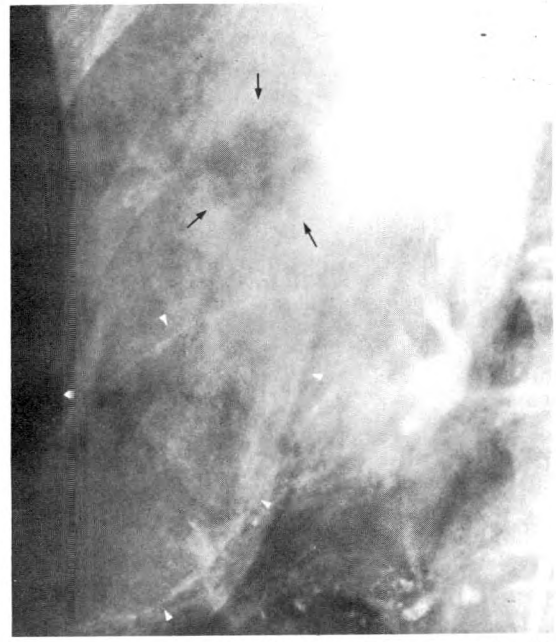


FIG. 1. Dilatation of gallbladder (white arrowheads) and biliary ducts in the liver hilus (black arrows) demonstrated during capillary phase of right posterior oblique (RPO) celiac arteriogram in patient with carcinoma of the head of the pancreas.

The gallbladder was dilated in 12 of 13 patients with carcinoma of the head of the pancreas who did not have dilated ducts. Indeed some of the largest gallbladders were noted in the patients without ductal dilatation.

The gallbladder and/or biliary ducts were distended proximal to the obstructing

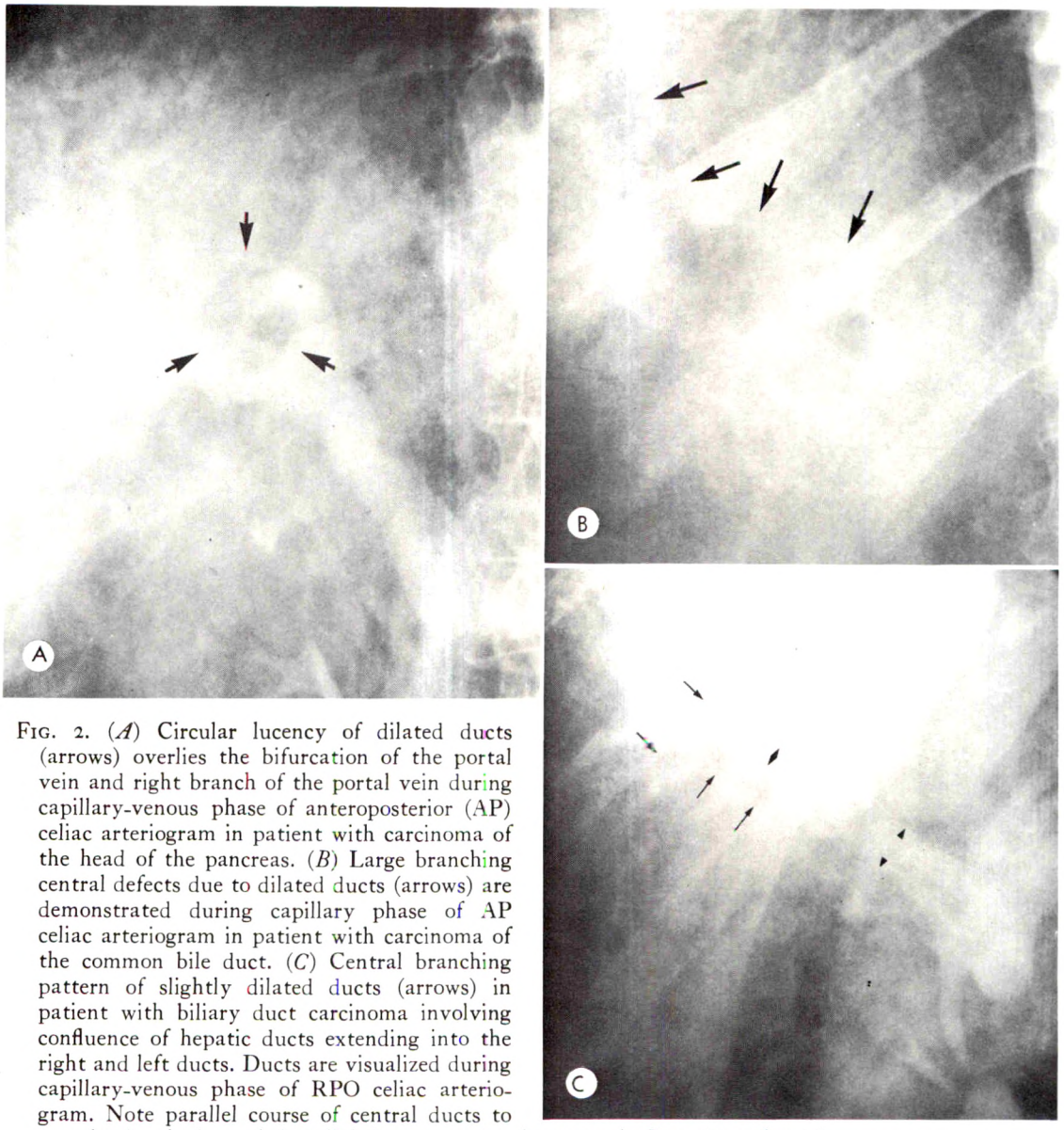


FIG. 2. (*A*) Circular lucency of dilated ducts (arrows) overlies the bifurcation of the portal vein and right branch of the portal vein during capillary-venous phase of anteroposterior (AP) celiac arteriogram in patient with carcinoma of the head of the pancreas. (*B*) Large branching central defects due to dilated ducts (arrows) are demonstrated during capillary phase of AP celiac arteriogram in patient with carcinoma of the common bile duct. (*C*) Central branching pattern of slightly dilated ducts (arrows) in patient with biliary duct carcinoma involving confluence of hepatic ducts extending into the right and left ducts. Ducts are visualized during capillary-venous phase of RPO celiac arteriogram. Note parallel course of central ducts to portal vein, the latter being slightly narrowed as it enters the liver (arrowheads).

lesion in 29 of 30 patients with carcinoma of the head of the pancreas, in the 2 patients with carcinoma of the papilla of Vater and lymphosarcoma of the head of the pancreas, in 3 of 5 patients with common hepatic duct tumors, in 3 of 5 patients with carcinoma of the gallbladder, in the 4 patients with common bile duct tumors, in 4 of 6 patients with obstructing common bile duct calculi, and in the 2 patients with stricture of the common bile duct and narrowing of the common hepatic duct due to a cystic duct calculus.

OTHER ANGIOGRAPHIC ABNORMALITIES

(Table 1)

Carcinoma of the head of the pancreas and papilla of Vater, and lymphosarcoma of the pancreas. Arterial encasement was present in 26 of 30 patients with carcinoma of the head of the pancreas. The gastroduodenal and pancreaticoduodenal arteries were each

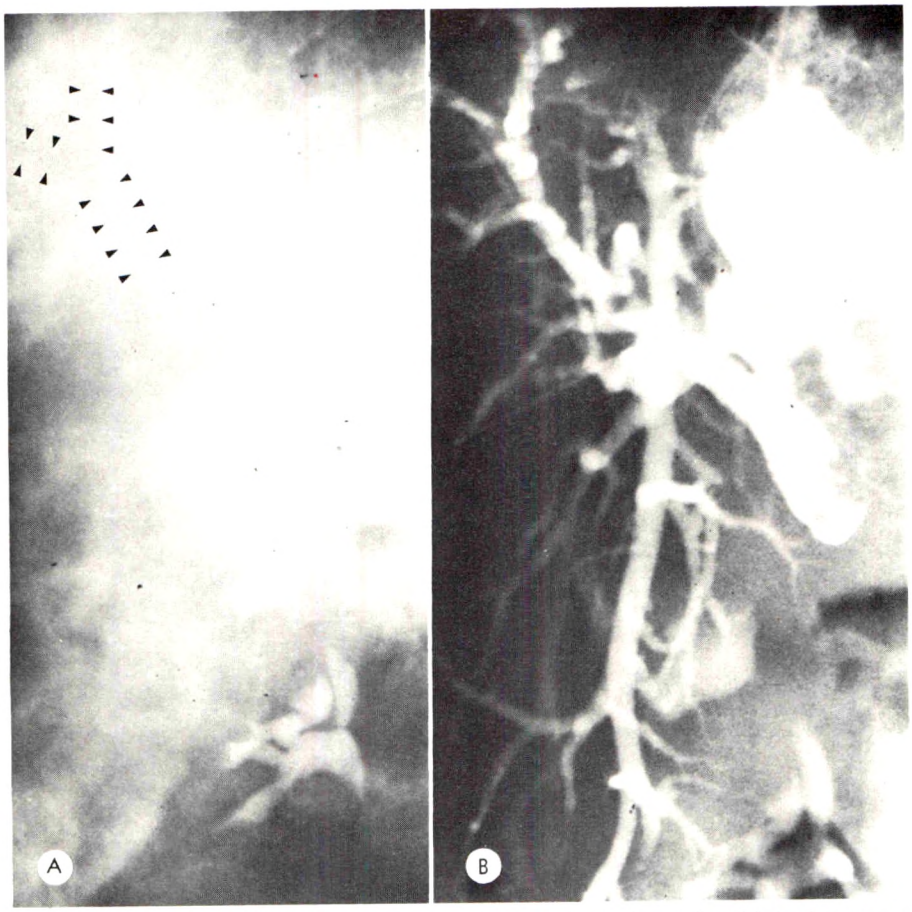


Fig. 3. (*A*) Branching pattern of slightly dilated peripheral ducts (arrowheads) is demonstrated during capillary phase of AP celiac arteriogram in patient with stricture of the common bile duct. (*B*) Percutaneous transhepatic cholangiogram confirms the existence of dilated peripheral ducts. Sludge is noted in the larger central ducts.

involved in 15 patients (Fig. 10), and the superior mesenteric artery and/or the origins of its proximal branches in 13 patients (Fig. 11). The hepatic artery was involved 7 times (Fig. 14) and the splenic artery twice. The angiograms of the patient with carcinoma of the papilla of Vater showed displacement with slight narrowing of the pancreaticoduodenal arcade (Fig. 12). The angiogram of the patient with lymphosarcoma showed stretching with slight displacement of the hepatic and gastroduodenal arteries and slight narrowing of the gastroduodenal artery (Fig. 13*A*).

Neovascularity was present in 6 cases of carcinoma of the head of the pancreas (Fig. 14). Tumor blushes were present in 5 of these 6 cases and in 5 other cases without neovascularity. Neither the patient with carcinoma of the papilla of Vater or the patient with lymphosarcoma had neovascularity or a tumor blush demonstrated.

The superior mesenteric, portal and/or splenic veins were narrowed or occluded in 26 of the 30 patients with carcinoma of the head of the pancreas. The superior mesenteric vein was involved in 21 patients (Fig. 11*B*), and the splenic and portal veins were each involved in 11 patients. All 3 veins were narrowed or occluded in the patient with lymphosarcoma (Fig. 13, *B* and *C*), while none of these veins was involved in the patient with carcinoma of the papilla of Vater.

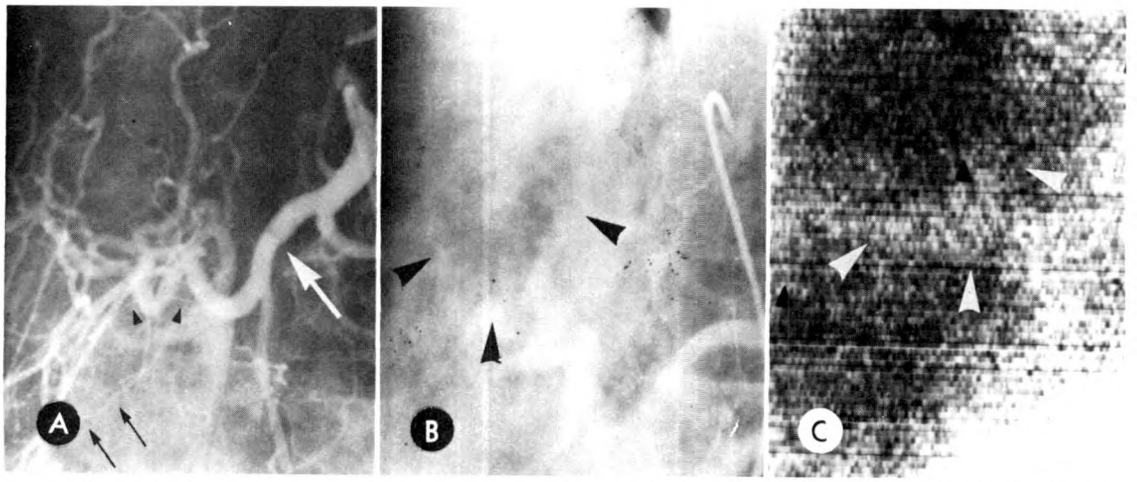


FIG. 4. (*A*) AP celiac arteriogram in patient with carcinoma of the gallbladder shows encasement of hepatic artery (large white arrow) and of cystic artery branches (small black arrows). Note displacement of arteries at bifurcation of cystic artery around an enlarged gallbladder (black arrowheads). Slight neovascularity in gallbladder region is also present. (*B*) Capillary phase shows large central branching defect due to dilated ducts (arrowheads). (*C*) Liver scan shows central defect due to dilated ducts (arrowheads).

Ascites was identified in 2 patients with carcinoma of the head of the pancreas. Liver metastases were not seen in this group of patients.

Carcinoma of the gallbladder. An enlarged cystic artery was noted in 2 of 5 patients with carcinoma of the gallbladder. Encasement of arteries was present in all 5 patients. In 1 case the cystic was the only encased artery. The main cystic artery was encased in 2 cases and its more peripheral branches in 2 other cases (Fig. 4*A*). The hepatic (Fig. 4*A*), gastroduodenal and pancreaticoduodenal arteries were each involved in 3 patients.

Neovascularity and a tumor blush were encountered in 4 patients (Fig. 16*A*).

All patients had narrowing or occlusion of the major veins. The portal vein was involved in 4 patients, the superior mesenteric vein in 3 and the splenic vein in 2. Early filling of hepatic veins and early

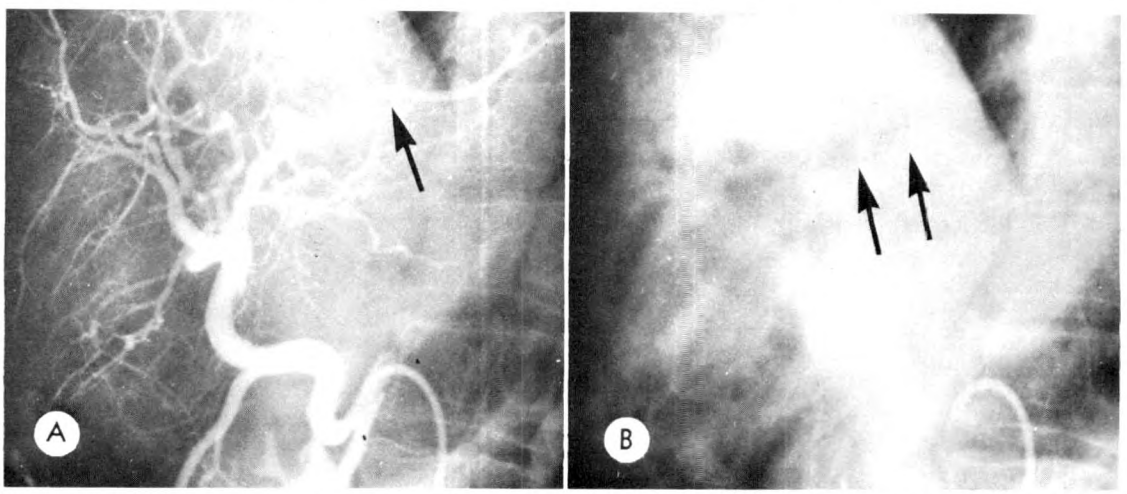


FIG. 5. (*A*) RPO arteriogram (common celiac-superior mesenteric trunk) in patient with carcinoma of the head of the pancreas. Arrow points to the left hepatic artery. (*B*) Capillary phase demonstrates dilated central and peripheral ducts. Arrows point to a dilated left hepatic duct.

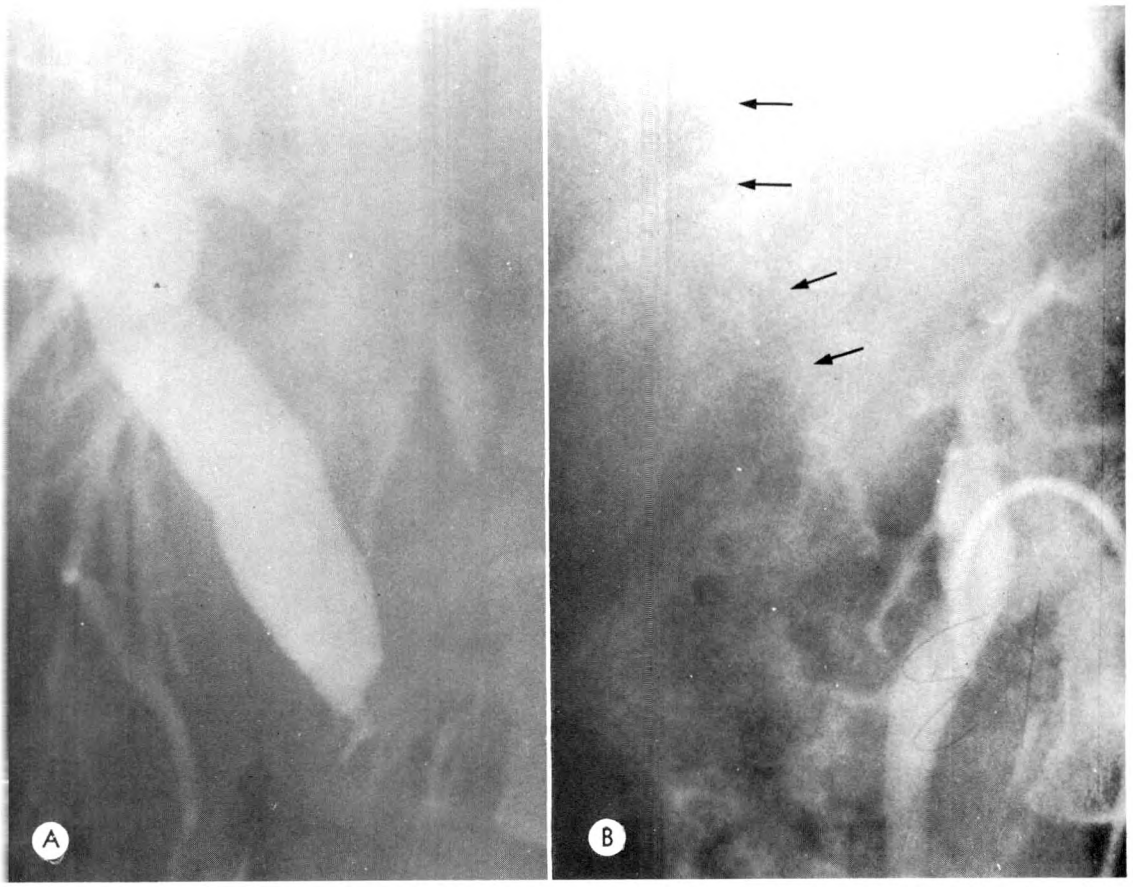


Fig. 6. (*A*) RPO view during percutaneous transhepatic cholangiography demonstrates dilatation of the common hepatic and common bile ducts in patient with carcinoma of the head of the pancreas. Note narrowing of the common bile duct by tumor in the pancreas. (*B*) Late capillary phase of RPO celiac arteriography shows defect due to dilated common hepatic and common bile ducts (arrows).

filling of enlarged cholecystic and pericholecystic veins were seen in 2 patients (Fig. 16*B*).

Carcinoma of the hepatic and common bile ducts. Of the 5 patients with common hepatic duct tumors and 4 patients with common bile duct tumors the only arterial abnormality related to the neoplasms was irregularity of the pancreaticoduodenal arteries in 2 patients with carcinoma of the common bile duct (Fig. 15). One other patient with carcinoma of the common hepatic duct had pancreaticoduodenal encasement due to pancreatitis.

Neovascularity was not seen in any of these patients. A tumor blush was seen in 1 patient with a common bile duct tumor (Fig. 15), but not in any other of these patients. The portal vein and its branches showed slight narrowing in 2 patients with hepatic duct tumors (Fig. 2*C*; and 9*B*), while the veins were normal in the 7 other patients.

Other patients. The only arterial abnormality noted on the angiograms of 6 patients with common bile duct calculi causing jaundice was pancreaticoduodenal narrowing in 1 patient who had pancreatitis. The capillary and venous phases were normal. The patient with a common bile duct stricture and the patient with a cystic duct calculus compressing the common hepatic duct had normal angiograms.

Two patients with hepatitis and 1 with sclerosing cholangitis had normal arteriograms. Two patients with cirrhosis had en-

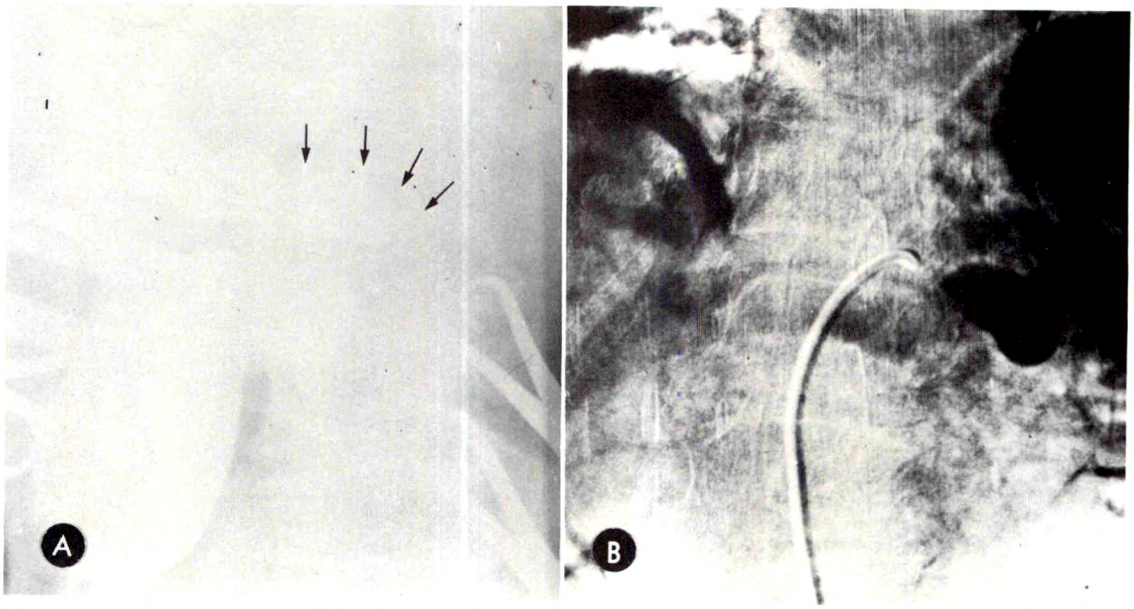


FIG. 7. (*A*) Operating room cholangiogram shows dilatation of the gallbladder and biliary ducts in patient with carcinoma of the head of the pancreas. Arrows point to the upper margin of the common hepatic duct. (*B*) Subtraction technique film of venous phase of AP celiac arteriogram shows slight upwards concavity of portal vein where the vein is in relationship to the common hepatic duct.

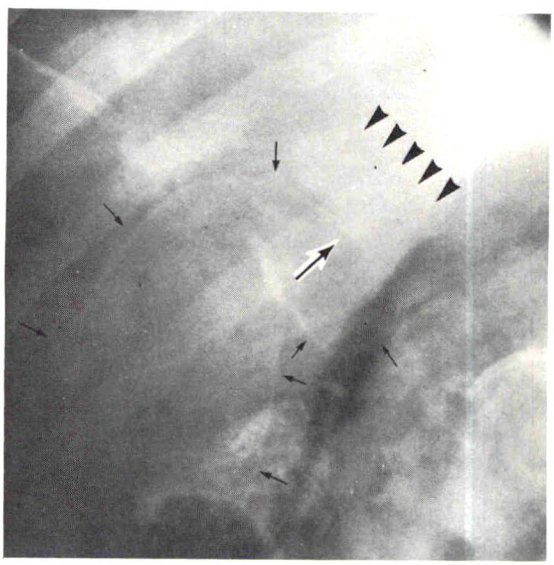


FIG. 8. Dilated neck of the gallbladder presses upon the portal vein (large black and white arrow) as seen in the capillary venous phase of RPO superior mesenteric arteriogram in patient with carcinoma of the head of the pancreas. The black arrowheads point to the upper medial aspect of the portal vein and the black arrows point to the distended gallbladder. The cystic artery arose from the superior mesenteric artery.

larged tortuous intrahepatic arteries. One of these patients had ascites which was first detected on the angiogram and the other had severe portal hypertension with no filling of the intrahepatic branches of the portal vein. One other patient with cirrhosis had a normal arteriogram. The patient with carcinoma of the body of the pancreas with liver metastases showed encasement of the splenic artery, nonfilling of the splenic vein with filling of collateral veins, and defects within the liver in the hepatogram phase.

DISCUSSION

The arteriogram is useful in the jaundiced patient in suggesting whether jaundice is on an obstructive or nonobstructive basis, and if obstructive, in determining the site and extent of the obstructing lesion.

Redman and Reuter[37] showed that gallbladders measuring more than 35 cm.^{2} or having a width of more than 5 cm. may be considered distended, and this distention is usually due to obstruction. Distention of the gallbladder may occasionally be seen

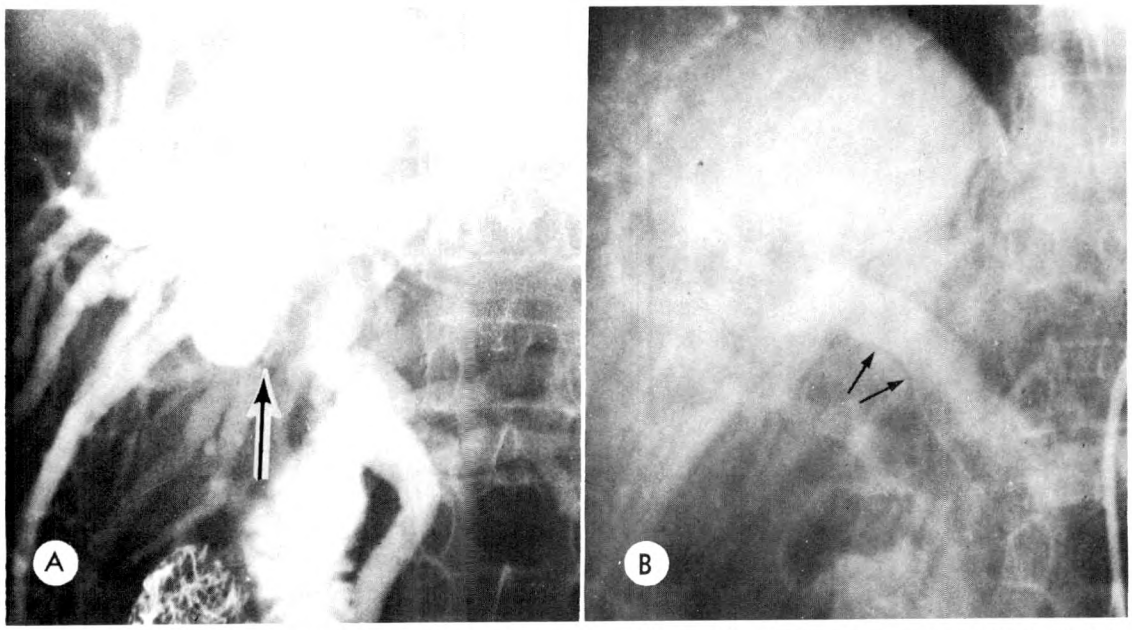


FIG. 9. (*A*) Percutaneous transhepatic cholangiogram in patient with carcinoma of common hepatic duct. Note marked narrowing at confluence of the hepatic ducts (arrow), and nonfilling of the common hepatic duct. Some contrast material passed the obstruction, and the common bile duct is visualized. (*B*) Localized indentation on inferior aspect of portal vein (arrows) which is considered to be related to the tumor mass. Note also dilated ducts which can be correlated with the percutaneous transhepatic cholangiogram. The ducts can be seen alongside the central branches of the portal vein.

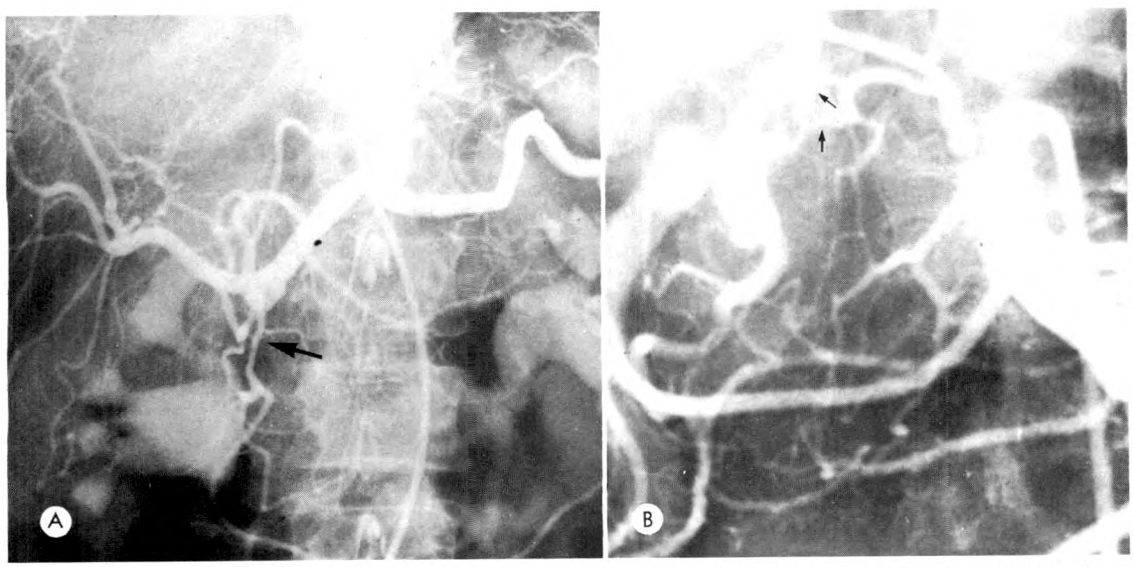


FIG. 10. (*A*) Encasement of proximal gastroduodenal artery (arrow) in patient with carcinoma of the head of the pancreas. (*B*) Irregular encasement of pancreaticoduodenal arteries (arrows) in another patient with carcinoma of the head of the pancreas as demonstrated on AP superior mesenteric arteriogram.

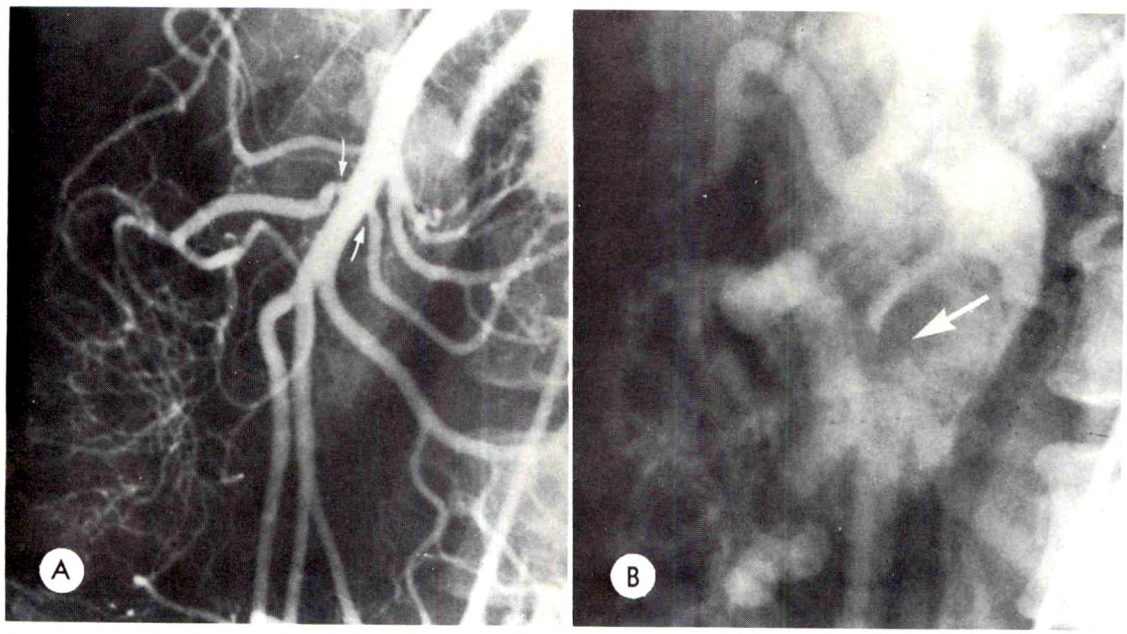


Fig. 11. (*A*) Encasement of the origins of the jejunal and colic arteries (arrows) in a patient with carcinoma of the head of the pancreas as seen on RPO superior mesenteric arteriogram. (*B*) Venous phase shows nonfilling of superior mesenteric vein at the level of arterial encasement (arrow) with collateral filling of the superior mesenteric and portal veins centrally.

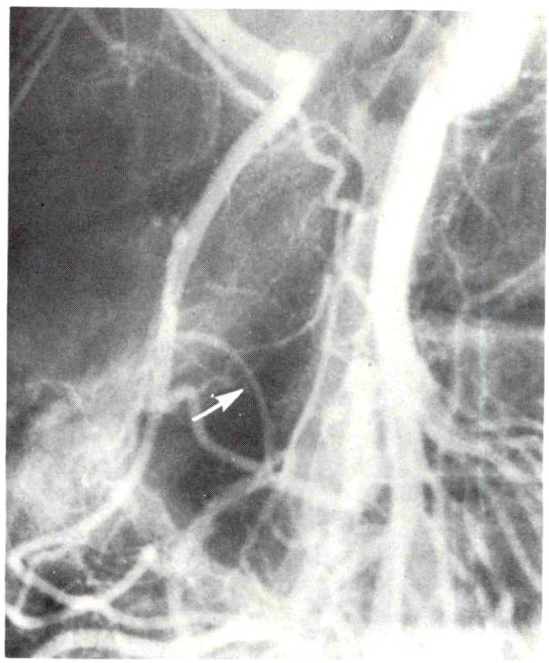


Fig. 12. Displacement and slight narrowing (arrow) of the pancreaticoduodenal arcade in a patient with carcinoma of the papilla of Vater as demonstrated on superior mesenteric arteriogram.

without obstruction. One jaundiced patient in this series with cirrhosis and gallstones had a distended gallbladder without cystic or common bile duct obstruction. We have seen one nonjaundiced patient with gallstones (not in this series) who also had a distended nonobstructed gallbladder. While the cause of gallbladder dilatation is not always certain, gallbladder dilatation in a jaundiced patient is strongly suggestive of distal biliary obstruction. If the gallbladder is enlarged or normal, the cystic artery and gallbladder will almost invariably be demonstrated on the angiogram. On the other hand, cystic artery visualization was seen in only 2 of 18 patients with contracted gallbladders.[53]

In the present series the arteriogram was highly reliable in determining the presence or absence of distal biliary obstruction. It should be noted in this regard that the angiogram is a more reliable indicator of gallbladder distention than physical examination. In one series an enlarged gallblad-

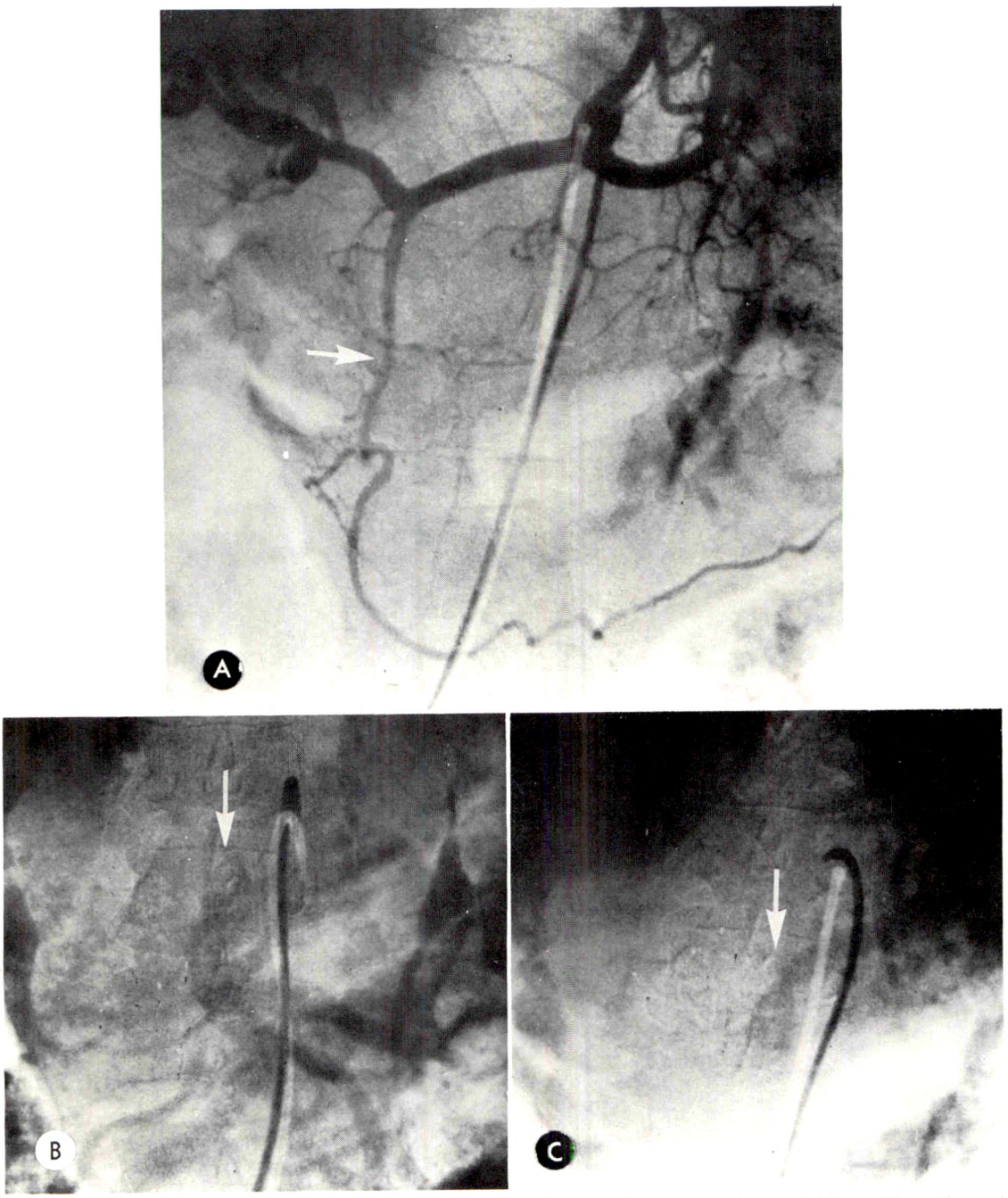


Fig. 13. (*A*) Subtraction technique film of AP celiac arteriogram shows mild stretching of hepatic and gastroduodenal arteries and narrowing of gastroduodenal artery (arrow) in a patient with lymphosarcoma of the pancreas. (*B*) Venous phase of superior mesenteric arteriogram of same patient shows obstruction of superior mesenteric vein (arrow) with nonfilling of the portal vein. (*C*) Venous phase of celiac arteriogram shown in *A* demonstrates obstruction of splenic vein (arrow) with nonfilling of portal vein.

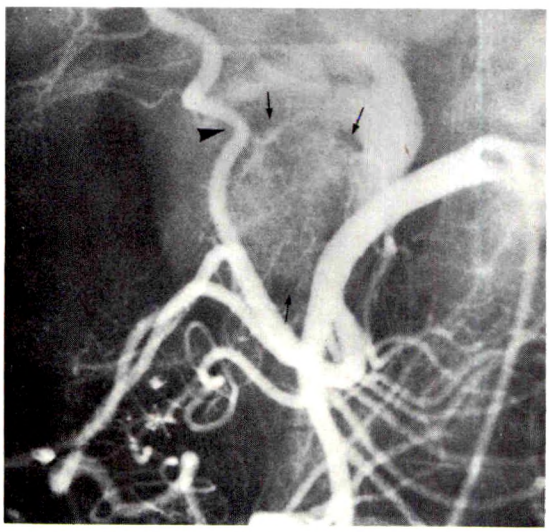


Fig. 14. Slight neovascularity of mucinous adenocarcinoma of the head of the pancreas (arrows) shown on RPO superior mesenteric arteriogram. Note also encasement of the right hepatic artery (arrowhead) which arises from the superior mesenteric artery.

der was palpated in 31 per cent of 90 patients with carcinoma of the head of the pancreas and in 68 per cent of 16 patients with carcinoma of the papilla of Vater.[29] In another series of 152 patients with carcinoma of the head of the pancreas Richards and Sosin[45] found that 17 patients had prior cholecystectomies and that a palpable gallbladder was present on physical examination in only 18 per cent of patients who had not had previous cholecystectomy.

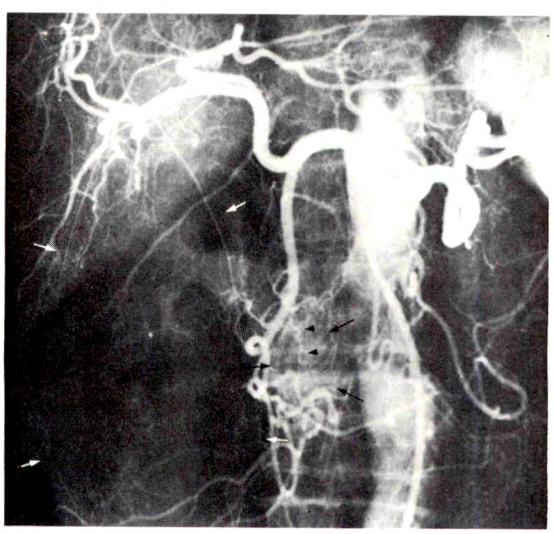


Fig. 15. AP celiac arteriogram shows irregularity of pancreaticoduodenal arteries (arrowheads) and blush (black arrows) of 2 cm. in diameter carcinoma of the common bile duct. Note also displacement of the cystic arteries (white arrows) around a markedly distended gallbladder.

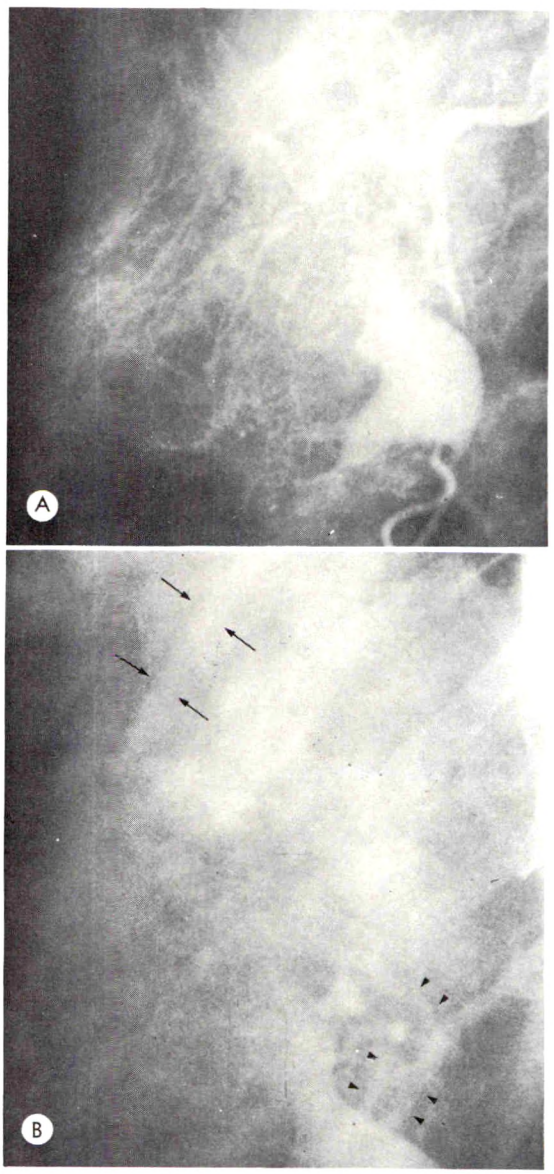


Fig. 16. (*A*) AP hepatic arteriogram shows large area of hypervascularity and tumor staining in the liver and gallbladder bed. The gallbladder outline cannot be delineated. (*B*) Late capillary phase shows early visualization of the hepatic vein (arrows) and early filling of the enlarged pericholecystic veins (arrowheads).

Gallbladder distention was invariably present with a distal obstruction and a normal gallbladder. Surprisingly, the gallbladder was often dilated in the presence of a distal obstruction despite the existence of cholecystitis and cholelithiasis. While this is a small group of patients, the evidence indicates that chronic gallbladder inflammation by no means precludes gallbladder distention, when a distal obstruction is present.

When obstructive jaundice is due to carcinoma of the gallbladder, a distended gallbladder may or may not be seen. Although gallbladder distention is frequently present at surgery, a tumor blush of the gallbladder often merges with a blush in the right lobe of the liver making delineation of the gallbladder difficult (Fig. 16*A*). In some of these and other cases in which the gallbladder outline is not clearly visualized, gallbladder distention may be detected by noting separation and stretching of the branches at the bifurcation of the cystic artery (Fig. 4*A*).

Dilatation or lack of dilatation of the hepatic ducts may also be used as an aid in the differentiation of obstructive from nonobstructive jaundice. A case showing dilated bile ducts on the angiogram was reported by Rossi and Gould,[49] but we feel that this finding has not been sufficiently emphasized to date. As shown above dilatation of the extrahepatic ducts as well as the intrahepatic ducts could often be detected (Fig. 1; 2; 4; 5; 6; and 9). The common hepatic duct lies anterior and to the right of the portal vein.[16,23] Dilatation of the common hepatic duct or a tumor within this duct may displace the portal vein superiorly, posteriorly and/or to the left (Fig. 7; and 9). The right and left hepatic ducts lie anterior to the portal vein[15,23] and may displace the vein posteriorly. A dilated neck of the gallbladder may cause a similar displacement of the portal vein (Fig. 8). The left hepatic duct is fairly constant in its relationship to the left branch of the portal vein with the duct lying above and anterior to the vein,[54] so that this duct may at times be identified by following the course of the left portal vein, when filled, or the left hepatic artery (Fig. 5). Not infrequently the left hepatic duct will bifurcate into 2 branches of equal size. Due to its longer extrahepatic course the left hepatic duct often dilates more than the right hepatic duct when there is a distal obstruction.[54]

The right hepatic duct is less constant in length and in its branching pattern than the left hepatic duct, since there is often an early bifurcation with a short or entirely absent right hepatic duct. Central lucencies related to the right duct, its major branches or the confluence of the right and left ducts were often detected (Fig. 1; 2; 4; 5; and 9). It is important not to misinterpret a central circular lucency within the liver as a metastasis (Fig. 1; and 2*A*). This defect due to duct dilatation has also been reported with liver scanning[25] (Fig. 4*C*). In a jaundiced patient a single central hepatic lucency on angiography is almost certainly related to duct dilatation rather than to liver metastases.

With distal obstruction the ducts are often dilated together with the gallbladder (Fig. 1) or without gallbladder dilatation in cases with previous cholecystectomy and some cases of chronic fibrotic cholecystitis. Dilated ducts may also be seen when the obstruction is proximal to the cystic duct-common duct junction (Fig. 2*C*; 4; and 9*B*). In these cases the differential diagnosis is between a distal obstructing lesion with gallbladder disease (or cholecystectomy), and a more proximal obstructing lesion involving the common hepatic duct. Transhepatic or transjugular cholangiography, which we advocate prior to surgery in all patients operated upon for jaundice will resolve this differential diagnosis.

As mentioned above, hepatic duct distention is not always seen in the presence of obstructing lesions. Dilatation of the intrahepatic ducts and of the extrahepatic ducts in the liver hilus is often seen with mild to moderate ductal distention. Dilatation of the common hepatic duct and common bile duct may also occasionally be seen (Fig. 5;

and 6) or inferred by displacement of the portal vein (Fig. 7). In addition, patients may have marked distention of the gallbladder with no or only slight distention of the intra- and extrahepatic ducts. Hepatic parenchymal compliance appears to play a role in the ability of the intrahepatic ducts to distend. In cirrhotic livers the lack of hepatic compliance will not allow for intrahepatic duct dilatation.[61] The relative and absolute compliance of the gallbladder, extrahepatic, and intrahepatic ducts may well help to determine at which level, if any, dilatation will be present.

SPECIFIC ENTITIES

CARCINOMA OF THE HEAD OF PANCREAS

Arterial encasement is a well known angiographic manifestation of carcinoma of the pancreas, yet few articles have described the frequency of specific artery involvement with this disease.

The arteries which are most frequently involved with carcinoma of the head of the pancreas and of the papilla of Vater are the pancreaticoduodenal arteries. The gastroduodenal and superior mesenteric arteries are often involved with the larger tumors. The incidence of demonstrating the pancreaticoduodenal arteries is dependent upon technique. Reuter[39] states that visualization of all the pancreaticoduodenal arteries can only consistently be obtained with the catheter in the gastroduodenal artery. In his series hepatic artery injection produced no definite improvement in the visualization of these arteries in the head of the pancreas as compared with simultaneous celiac and superior mesenteric injections. Boijsen[8] agrees that the best visualization of the intrapancreatic arteries is obtained by gastroduodenal injection, but also states that a distal hepatic arterial injection will usually give good detail of the arteries in the head of the pancreas. Takashima *et al.*[57] also show good visualization of the arteries in the head of the pancreas with the catheter in the distal hepatic artery, with slight improvement in filling with gastroduodenal injection. Lunderquist[30] on the other hand reported reliable filling of the pancreaticoduodenal arteries by simultaneous celiac and superior mesenteric injection.

In the present series it was seen that sequential celiac and superior mesenteric artery injections often but not consistently filled the pancreaticoduodenal arteries. These arteries were often filled by superior mesenteric as well as celiac injection. Indeed the pancreaticoduodenal arteries were often better filled by superior mesenteric than celiac injection as in the cases shown in Figures 10*B* and 12. The first superior mesenteric injection is performed in the RPO projection to displace the superior mesenteric vein from the spine. If better delineation of the pancreaticoduodenal arteries is desired, another injection with a smaller amount of contrast material is performed in the AP projection, coning down on the head of the pancreas.

The incidence of gastroduodenal and pancreaticoduodenal arterial involvement with carcinoma of the head of the pancreas and papilla of Vater in the current series and in previous series is given in Table III.

TABLE III

ARTERIAL INVOLVEMENT WITH CARCINOMA OF THE HEAD OF PANCREAS AND PAPILLA OF VATER

	Carcinoma of Head of Pancreas		Carcinoma of Papilla of Vater	
	Gastroduodenal	Pancreaticoduodenal	Gastroduodenal	Pancreaticoduodenal
Lunderquist[30]	6/13	14/23	1/3	1/3
Suzuki *et al.*[56]	1/7	3/7	0/7	2/7
Reuter *et al.*[42]			0/2	2/2
Current Series	15/30	15/30	0/1	1/1

Other entities which may cause narrowing of the gastroduodenal or pancreaticoduodenal arteries are pancreatitis, gastric ulcer, carcinoma of the common bile duct, duodenum or stomach, metastases to the pancreas, arteritis and perhaps most importantly atherosclerosis.[31,42,44] Frequently the question arises as to whether narrowing is due to atherosclerosis or pancreatic neoplasm. Unfortunately there is no consistently opacified vein, as there is with the splenic and superior mesenteric arteries, which can be utilized (by the presence or absence of involvement) as an indication whether narrowing of the gastroduodenal or pancreaticoduodenal arteries is due to pancreatic disease or atherosclerosis.

In our experience atherosclerotic narrowing of the gastroduodenal artery is not common and when it does occur the narrowing is slight, but we have seen 1 case with atherosclerotic occlusion of this artery. Reiner and Jimenez[38] have shown pathologically that the gastroduodenal artery may not infrequently be affected by mild atherosclerosis, but pancreaticoduodenal arcade involvement is much less frequent and when present is usually associated with severe atherosclerosis elsewhere in the celiacomesenteric circulation.

Bookstein *et al.*[12] reported that smooth atherosclerotic narrowing of the hepatic or gastroduodenal arteries may simulate carcinomatous involvement, but that mild narrowing of these arteries as the sole manifestation of pancreatic carcinoma is quite unusual. Only 2 of 41 patients with carcinoma of the pancreas demonstrated narrowing of the hepatic or gastroduodenal arteries without associated angiographic abnormalities of neoplasm. Our findings are similar regarding gastroduodenal and pancreaticoduodenal involvement. Of 15 patients with narrowing of the gastroduodenal artery, 13 had major vein involvement; the other 2 patients both had tumor blushes and 1 also had neovascularity. A total of 5 of these 15 patients had neovascularity and/or tumor blushes. Also, of these 15 patients 11 had additional arteries encased (pancreaticoduodenal 9, superior mesenteric and/or branches 5, hepatic 5, and splenic 2).

Pancreaticoduodenal narrowing was present in 15 patients. Only 1 had no additional arterial encasement, neovascularity, venous involvement or tumor blush. Of the remaining 14 patients 12 had major venous involvement, while a tumor blush was present in the other 2 patients and 1 also had neovascularity demonstrated. A total of 7 of these patients had neovascularity and/or tumor blushes. Thirteen patients in this group had involvement of other arteries (gastroduodenal 8, superior mesenteric and/or branches 7, hepatic 6, and splenic 2).

The differential diagnosis of atherosclerosis does not need to be considered when there is stretching and displacement of arteries. The pancreaticoduodenal arcade was stretched and displaced in our single case of carcinoma of the papilla of Vater (Fig. 12) and a similar finding was described by Suzuki *et al.*[56] in 1 of their cases of cancer of the papilla. In these cases the differential diagnosis includes pancreatic pseudocyst, but pseudocyst is usually an untenable diagnosis both angiographically, because of lack of additional findings, and clinically. Carcinoma of the pancreas may also be considered, but carcinoma because of its infiltrating growth pattern usually produces encasement rather than displacement of arteries.

The superior mesenteric artery and the origins of its proximal branches (inferior pancreaticoduodenal, middle colic and jejunal arteries) were narrowed in 13 of 30 cases with carcinoma of the head of the pancreas (Fig. 11). In 4 of these patients the gastroduodenal and pancreaticoduodenal arteries were normal. Buranasiri and Baum[13] showed several instances of involvement of the superior mesenteric artery by tumors of the head of the pancreas. Superior mesenteric artery involvement has been mentioned by other authors, but usually in conjunction with tumors of the uncinate process invading the transverse mesocolon.[5,6] The present study demon-

strates the frequency of involvement of the superior mesenteric trunk and/or its proximal branches by tumors of the head of the pancreas. Because of the frequency with which the superior mesenteric artery and vein (as discussed below) are involved by tumors of the head of the pancreas, superior mesenteric as well as celiac arteriography should be performed on the jaundiced patient.

Certainly abundant neovascularity and dense tumor stains are not characteristic of the ordinary adenocarcinoma of the pancreas and indeed when a vascular lesion is present in the pancreatic region, this suggests a tumor of unusual cell type.[1,11,32,33,46] There is a wide discrepancy among various reports concerning the incidence of neovascularity and tumor blushes with the ordinary adenocarcinomas of the pancreas. In 41 patients with carcinoma of the pancreas Bookstein *et al.*[12] described pathologic vessels in 24 patients and a blush in 4 patients. Lunderquist[30] reported pathologic vessels in 17 patients and a blush in 6 of 23 patients with carcinoma of the head of the pancreas. On the other hand Buranasiri and Baum[13] reported only 1 case each with a blush and new vessels among 50 patients with carcinoma of the pancreas, half of which involved the pancreatic head. The difference in frequency of these angiographic signs is probably related to a combination of factors, both interpretative and technical. At times it is difficult to distinguish new vessels from encased small arteries. Also blushes may be present normally in the pancreas albeit more commonly in the tail. The use of superselective studies and enhancement techniques, such as magnification and subtraction, undoubtedly will result in an increased delineation of tumor vessels and blushes.

In our series of 30 patients with carcinoma of the head of the pancreas neovascularity and/or tumor blushes were identified in 11 patients (Fig. 14). Three of these patients had unusual histologic cell types (squamous cell 2, and mucinous, 1).

Venous involvement with tumors of the head of the pancreas is the rule, and as stated above was present in 26 of 30 patients with carcinoma of the head of the pancreas and in the patient with pancreatic lymphosarcoma. The importance of the venous phase of the angiogram was first stressed by Buranasiri and Baum.[13] In their series of 16 patients with tumors of the head of the pancreas the superior mesenteric and/or portal vein were definitely involved in 12 patients, while superior mesenteric and/or splenic vein involvement were demonstrated in every one of 10 patients with carcinoma of the head and body of the pancreas. To ensure good venous filling, a relatively large bolus of contrast material (50 cc.) is given, and prolonged filming (covering approximately 30 seconds) is obtained. Buranasiri and Baum[13] also state that venous opacification may be enhanced by a relatively slow injection rate (less than 8 cc./sec.) and intraarterial tolazoline injection (25–50 mg.) immediately prior to contrast medium injection.

Ascites was detected on the angiogram by displacement of the liver from the lateral body wall. This was noted in 2 patients, in 1 of whom it was the first clinical indication of ascites. Angiographic evidence of liver metastases was not identified in any patient with carcinoma of the head of the pancreas. When metastases were present at surgery, they were often quite small.

LYMPHOMA

In general lymphomatous tumors are hypovascular, although at times mild hypervascularity may be seen. In our experience with a single lymphosarcoma of the pancreatic region described in this communication and other lymphomatous tumors in the mesentery, there appears to be more stretching and displacement of arteries than encasement, although marked venous obstruction is seen at times (Fig. 13). Similar findings were demonstrated in 2 cases reported by Reuter and Redman.[40] The relative lack of arterial encasement may be due to the fact that many lymphomatous tumors are more cellular and produce less

TABLE IV

ANGIOGRAPHIC FINDINGS WITH CARCINOMA OF THE GALLBLADDER

	Enlarged Cystic Artery	Encased Arteries		Neovascularity	Blush	Encased Veins
		Cystic and Branches	Other			
Abrams *et al.*[2]	5/5	5/5	+	5/5	+	
Deutsch[14]	2/5			2/5	2/5	
Reuter *et al.*[41]	2/5	5/5	4/5	5/5	5/5	
Rösch *et al.*[47]	+		5/7	+	+	2/7
Sato *et al.*[53]		7/16	+	+	13/16	
Current Series*	4/7	4/7	4/7	6/7	6/7	6/7

* Includes 2 cases in which patients did not present with jaundice.

of a desmoplastic response than the typical carcinoma of the pancreas.

In a series of 1,269 patients with lymphosarcoma, including patients with giant follicular lymphoblastoma, lymphosarcoma, and reticulum cell sarcoma, jaundice occurred during the course of the disease in 159 patients (12.5 per cent) and was the first sign of disease in 3 patients.[48] Twenty-two per cent of these patients had chemistries characteristic of obstruction. Of the 44 patients in whom an explanation of jaundice was forthcoming extrahepatic bile obstruction by tumor was the definite cause in 14 and the probable cause in 4 more. Jaundice was due to obstructing common bile duct calculi in 3 patients, to cirrhosis in 3 patients, to hepatitis in 4 patients and to hepatic tumor alone in 12 patients. In another review article Hoster and Dratman[26] stated that jaundice occurred in 3–8 per cent of patients with Hodgkin's disease and that jaundice may be due to pressure of enlarged lymph nodes on the extrahepatic bile ducts.[26]

CARCINOMA OF THE GALLBLADDER

Carcinoma of the gallbladder is usually detected after it has invaded adjacent structures rendering the tumor unresectable. Direct invasion of the liver and obstruction of the extrahepatic biliary ducts are common. Approximately 50 per cent of patients with carcinoma of the gallbladder present with jaundice.[18]

Most authors have described similar angiographic appearances with gallbladder carcinoma.[2,14,41,47,53,55] Table IV is a summary of the findings of the present and previously reported cases. The cystic artery is often enlarged and encasement of this artery or its branches is the rule (Fig. 4). Due to extracholecystic extension, adjacent arterial encasement is also present in the majority of cases, with the hepatic artery and its branches most frequently involved (Fig. 4). New vessel formation is present in the vast majority of patients and this is accompanied by a tumor blush (Fig. 16). Extension to the liver is associated with hypervascularity, but in the unusual avascular carcinoma, avascularity within the adjacent invaded liver parenchyma may be seen.

Since extracholecystic extension is the rule, it is often difficult to outline the gallbladder in its entirety in the capillary phase.

Major venous encasement is quite common with gallbladder carcinoma. The portal vein is the vein most frequently narrowed or occluded, but mesenteric and splenic vein encasements are also common. It is probable that venous involvement is more severe in those patients who present with jaundice, since biliary obstruction indicates a central growth of the neoplasm.

Prominent early filling cholecystic and pericholecystic veins may be seen with carcinoma of the gallbladder (Fig. 16*B*).[14,55] We have also noted early filling of hepatic veins in 2 of the 5 patients reported in this com-

munication (Fig. 16*B*) and in 2 other patients with gallbladder carcinoma who did not present with jaundice.[55]

When large, a carcinoma of the gallbladder which invades the liver may be difficult to distinguish from a hepatoma. The diagnosis of gallbladder carcinoma is favored by an enlarged cystic artery feeding the tumor, less bizarre and prominent neoplastic vessels, and early filling of hepatic and enlarged pericholecystic veins. While gallbladder carcinomas may cause hepatic artery to hepatic vein shunting, hepatomas tend to cause hepatic artery to portal vein shunting.[9,60] There is also a much higher incidence of hepatic and portal vein invasion by hepatomas.

Regarding hepatic vein visualization it should be noted that this may be seen in 6 per cent of normal patients with celiac or hepatic arteriograms.[19] This is not seen in cirrhosis,[19] probably because of the associated hepatic venous encroachment,[28] despite an increase in the size and number of the hepatic arteries,[24] which is probably correlated with an increased arterial blood flow in cirrhosis. Hepatomas are usually superimposed upon cirrhosis, which helps to explain the rarity of hepatic vein visualization with hepatomas.[19] However, at least 1 case of hepatic artery to hepatic vein shunting due to a hepatoma has been described by Rossi and Gould.[49] Hepatic vein visualization is increased in carcinoma of the gallbladder because of the hypervascularity of these tumors and because cholecystic veins drain to hepatic as well as to portal veins.[22]

BILIARY DUCT TUMORS

The arteriograms in our patients with tumors of the hepatic and common bile ducts were usually normal. Three of 5 patients with common hepatic duct tumors and 2 of 4 patients with common bile duct tumors had no arterial, capillary or venous abnormality, and even the positive findings in this group of patients were subtle. The angiographic findings in biliary duct tumors in the current and previous series[27,41,53] are shown in Table v. Reuter *et al.*[41] state that the degree of neovascularity and the tumor staining are correlated with the size of the tumor and that the smaller tumors show mainly arterial encasement. A low incidence of portal vein involvement is noted in all reports of ductal tumors.[10,27,41,53]

The higher percentages of arterial encasement, neovascularity and tumor blushes in the reports of Reuter *et al.*[41] and Kaude and Rian[27] as compared with our series may well be due to a higher percentage of superselective studies by these authors. In the event that the superior mesenteric and celiac arteriograms are entirely normal, an argument could be made for obtaining superselective hepatic and gastroduodenal arteriograms. However, superselective examinations add to the complexity and procedure time of the examination, and increasing procedure time increases the potential complication rate. In addition, small artery abnormalities in the head of the pancreas are not specific for neoplasm and do not in any event indicate nonresectability should a tumor of the head of the pancreas be present. Redman and Reuter[36]

TABLE V

ANGIOGRAPHIC FINDINGS WITH BILIARY DUCT TUMORS

		Arterial Encasement	Neovascularity	Blush	Venous Encasement
Reuter *et al.*[41]	CHD	7/7	6/7	7/7	1/5
	CBD	4/7	3/4	1/4	0/3
Kaude and Rian[27]		13/16	13/16		2/6
Sato *et al.*[53]		1/3			
Current Series	CHD	0/5	0/5	0/5	2/5
	CBD	2/4	0/4	1/4	0/4

CHD = common hepatic duct; CBD = common bile duct.

have shown also that collaterals may develop in the liver hilus secondary to vascular stenosis or ligation, or cirrhosis. These may be difficult to distinguish from arteries infiltrated by neoplasm. In view of these factors an argument could be made for terminating the examination should the celiac and superior mesenteric arteriograms show no abnormality and proceeding to transjugular or transhepatic cholangiography. Normal celiac and superior mesenteric arteriograms for all practical purposes exclude a large carcinoma of the head of the pancreas which might be left unoperated, if the patient were not symptomatic from the elevated bilirubin.

CALCULUS DISEASE AND PANCREATITIS

Patients with calculus disease without pancreatitis have normal arteriograms. This is our experience and also that of Göthlin *et al.*,[20] who described normal selective visceral angiograms in 4 patients with jaundice due to common bile duct calculi. Two of our patients with pancreatitis had pancreaticoduodenal narrowing. One patient was jaundiced on the basis of common duct calculi, while the other had a tumor of the common hepatic duct. In the latter case pancreaticoduodenal narrowing was misinterpreted as indicating the presence of a tumor in the head of the pancreas. In our experience and that of others it may be difficult to differentiate carcinoma of the pancreas from pancreatitis. Both may cause stenosis of the large arteries near the pancreas and of the small intrapancreatic arteries, hypervascularity and tissue staining, as well as major vein stenosis or occlusion.[11,12,34,42] Reuter *et al.*[42] found that normal arteriograms were obtained in 22 per cent of patients with pancreatitis and that the severity of the angiographic findings was related to the duration of the disease rather than to the acuteness of the inflammatory process.

CIRRHOSIS, HEPATITIS, AND CHOLANGITIS

Cirrhosis may or may not be associated with an abnormal angiographic appearance during the arterial, capillary and venous phases. Typically in cirrhosis, biliary as well as portal, there is an increase in width of the hepatic artery, increased arterial collateral flow to the liver, and increased tortuosity of the intrahepatic arteries.[7] The increase in tortuosity of the intrahepatic arteries appears to be related to: (1) a decrease in liver volume;[8] (2) hepatic artery to portal vein communications,[35] so that the hepatic circulation functions as small arteriovenous fistulae;[24] (3) hepatic vein obliteration,[28] causing the portal vein to function as an efferent channel which results in the hepatic artery serving as the only afferent channel; and (4) regenerating nodules may be hypervascular,[17,58] and are supplied primarily by arterial blood.[59]

The hepatographic phase is often not intense during celiac angiography and therefore this phase may not be of help in the diagnosis of cirrhosis.[4,58] The venous phase, however, may be quite important in demonstrating altered venous drainage due to portal hypertension.[51]

No abnormal angiographic pattern has been described in patients with cholangitis or hepatitis except with alcoholic hepatitis in which the arteriogram may simulate a diffuse infiltrating hypervascular neoplasm.[50]

SUMMARY

Analysis of the angiogram of the jaundiced patient is often of help in separating surgical from nonsurgical jaundice.

In the case of obstructive jaundice the angiogram may demonstrate the etiology and extent of the obstructing lesion.

Occasionally, the angiogram will demonstrate a medical cause for jaundice (as cirrhosis) which may not be clinically apparent.

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REFERENCES

1. Abrams, R. M., Beranbaum, E. R., Beranbaum, S. L., and Ngo, L. Angiographic studies

of benign and malignant cystadenoma of pancreas. *Radiology*, 1967, *89*, 1028–1032.

2. Abrams, R. M., Meng, C. H., Firoognia, H., Beranbaum, E. R., and Epstein, H. Y. Angiographic demonstration of carcinoma of gallbladder. *Radiology*, 1970, *94*, 277–282.
3. Alfidi, R. J., Rastogi, H., Buonocore, E., and Brown, H. Hepatic arteriography. *Radiology*, 1968, *90*, 1136–1142.
4. Aronsen, K. F., Lunderquist, A., and Nylander, G. Comparison of celiacography and direct portography in diagnostic evaluation of liver disease. *Radiology*, 1969, *92*, 313–322.
5. Baron, M. G., Mitty, H. A., and Wolf, B. S. Arteriographic appearance of carcinoma of uncinate process of pancreas. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 649–655.
6. Blatt, C. J., Bernstein, R. G., and Lopez, F. Uncommon roentgenologic manifestation of pancreatic carcinoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 119–124.
7. Boijsen, E. Selective angiography of celiac axis and superior mesenteric artery in cirrhosis of liver. *Rev. Internat. Hepat.*, 1965, *3*, 323–336.
8. Boijsen, E. Selective pancreatic angiography. *Brit. J. Radiol.*, 1966, *39*, 481–487.
9. Boijsen, E., and Abrams, H. L. Roentgenologic diagnosis of primary carcinoma of liver. *Acta radiol.* (Diag.), 1965, *3*, 257–277.
10. Boijsen, E., and Reuter, S. R. Combined percutaneous transhepatic cholangiography and angiography in evaluation of obstructive jaundice. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 153–161.
11. Boijsen, E., and Tylen, U. Vascular changes in chronic pancreatitis. *Acta radiol.* (Diag.), 1972, *12*, 34–48.
12. Bookstein, J. J., Reuter, S. R., and Martel, W. Angiographic evaluation of pancreatic carcinoma. *Radiology*, 1969, *93*, 757–764.
13. Buranasiri, S., and Baum, S. Significance of venous phase of celiac and superior mesenteric arteriography in evaluating pancreatic carcinoma. *Radiology*, 1972, *102*, 11–20.
14. Deutsch, V. Cholecysto-angiography: visualization of gallbladder by selective celiac and mesenteric angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 608–616.
15. Elias, H., and Petty, D. Gross anatomy of blood vessels and ducts within human liver. *Am. J. Anat.*, 1952, *90*, 59–111.
16. Fainsinger, M. H. Radiology of intrahepatic biliary tract. *South African J. M. Sc.*, 1950, *15*, 51–66.
17. Farrell, R., Steinman, A., and Green, W. H. Arteriovenous shunting in regenerating liver simulating hepatoma. *Radiology*, 1972, *102*, 279–280.
18. Fortner, J. G., and Pack, G. T. Clinical aspects of primary carcinoma of gallbladder. *A.M.A. Arch. Surg.*, 1958, *77*, 742–750.
19. Glickman, M. G., and Handel, S. F. Opacification of hepatic veins during celiac and hepatic angiography. *Radiology*, 1972, *103*, 565–571.
20. Göthlin, J., Mansoor, M., and Tranberg, Karl-Göran. Combined percutaneous transhepatic cholangiography and selective visceral angiography (SVA) in obstructive jaundice. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 419–425.
21. Gray, R. K., Rösch, J., and Grollman, J. H., Jr. Arteriography in diagnosis of islet-cell tumors. *Radiology*, 1970, *97*, 39–44.
22. Gray's Anatomy. Thirty-fifth British Edition. Edited by R. Warwick and P. L. Williams. W. B. Saunders Company, Philadelphia, 1973, p. 1205.
23. Gray's Anatomy. Thirty-fifth British Edition. Edited by R. Warwick and P. L. Williams. W. B. Saunders Company, Philadelphia, 1973, pp. 710, 712.
24. Hales, M. R., Allan, J. S., and Hall, E. M. Injection-corrosion studies of normal and cirrhotic livers. *Am. J. Path.*, 1959, *35*, 909–941.
25. Heck, L. L., and Gottschalk, A. Appearance of intrahepatic biliary duct dilatation on liver scan. *Radiology*, 1971, *99*, 135–140.
26. Hoster, H. A., and Dratman, M. B. Hodgkin's disease. Part I. *Cancer Res.*, 1948, *8*, 1–48.
27. Kaude, J., and Rian, R. Cholangiocarcinoma. *Radiology*, 1971, *100*, 573–580.
28. Kelty, R. H., Baggenstoss, A. H., and Butt, H. R. Relation of regenerated liver nodule to vascular bed in cirrhosis. *Gastroenterology*, 1950, *15*, 285–295.
29. Klintrup, H.-E. Carcinoma of pancreas. *Acta chir. scandinav.*, 1966, Suppl. 362.
30. Lunderquist, A. Angiography in carcinoma of pancreas. *Acta radiol.*, 1965, Suppl. 235.
31. Olsson, O. Angiography in duodenal carcinoma. *Acta radiol.* (Diag.), 1971, *11*, 177–194.
32. Meaney, T. F., and Buonocore, E. Arteriographic manifestations of pancreatic neoplasm. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 720–725.
33. Meyers, M., and King, M. C. Leiomyosarcoma of duodenum. *Radiology*, 1968, *91*, 788–790.
34. Moskowitz, H., Chait, A., and Mellins, H. Z. "Tumor encasement" of celiac axis due to chronic pancreatitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 641–645.
35. Popper, H., Elias, H., and Petty, D. E. Vascular pattern of cirrhotic liver. *Am. J. Clin. Path.*, 1952, *22*, 717–729.

36. Redman, H. C., and Reuter, S. R. Arterial collaterals in liver hilus. *Radiology*, 1970, *94*, 575–579.
37. Redman, H. C., and Reuter, S. R. Angiographic evaluation of gallbladder dilatation. *Radiology*, 1970, *97*, 367–370.
38. Reiner, L., and Jimenez, F. A. Anatomic aspects of mesenteric arteriosclerosis. In: Vascular Disorders of the Intestine. Edited by S. J. Boley, S. S. Schwartz and L. F. Williams. Appleton-Century-Crofts, Education Division, New York, 1971.
39. Reuter, S. R. Superselective pancreatic angiography. *Radiology*, 1969, *92*, 74–85.
40. Reuter, S. R., and Redman, H. C. Gastrointestinal Angiography. Chapter 4. W. B. Saunders Company, Philadelphia, 1972.
41. Reuter, S. R., Redman, H. C., and Bookstein, J. J. Angiography in carcinoma of biliary tract. *Brit. J. Radiol.*, 1971, *41*, 636–641.
42. Reuter, S. R., Redman, H. C., and Bookstein, J. J. Differential problems in angiographic diagnosis of carcinoma of pancreas. *Radiology*, 1970, *96*, 93–99.
43. Reuter, S. R., Redman, H. C., and Joseph, R. R. Angiographic findings in pancreatitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 56–64.
44. Reuter, S. R., Redman, H. C., Miller, W. J., and Hoskins, P. A. Gastric angiography. *Radiology*, 1970, *94*, 271–276.
45. Richards, A. B., and Sosin, H. Cancer of pancreas. *Ann. Surg.*, 1973, *177*, 325–331.
46. Rösch, J., and Bret, J. Arteriography of pancreas. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 182–193.
47. Rösch, J., Grollman, J. H., Jr., and Steckel, R. J. Arteriography in diagnosis of gallbladder disease. *Radiology*, 1969, *92*, 1485–1491.
48. Rosenberg, S. A., Diamond, H. D., Jaslowitz, B., and Craver, L. F. Lymphosarcoma: review of 1269 cases. *Medicine*, 1961, *40*, 31–84.
49. Rossi, P., and Gould, H. R. Angiography and scanning in liver disease. *Radiology*, 1970, *96*, 553–562.
50. Rourke, J. A., Bosniak, M. A., and Ferris, E. J. Hepatic angiography in "alcoholic hepatitis." *Radiology*, 1968, *91*, 290–296.
51. Ruzicka, F. F., and Rossi, P. Arterial portography: patterns of venous flow. *Radiology*, 1969, *92*, 777–787.
52. Sato, T., Saitoh, Y., Koyama, K., and Watanabe, K. Preoperative determination of operability in carcinomas of pancreas and periampullary region. *Ann. Surg.*, 1968, *168*, 876–886.
53. Sato, T., Watanabe, K., Saitoh, Y., Koyama, K., and Suda, Y. Selective arteriography for gallbladder diseases. *A.M.A. Arch. Surg.*, 1969, *99*, 598–605.
54. Schein, C. J., Stern, W. Z., and Jacobson, H. G. The Common Bile Duct. Charles C Thomas, Publisher, Springfield, Ill., 1966, p. 33.
55. Sprayregen, S., and Messinger, N. H. Carcinoma of gallbladder: diagnosis and evaluation of regional spread by angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 382–392.
56. Suzuki, T., Kawabe, K., Nakayasu, A., Takeda, H., Kobayashi, K., Kubota, N., and Honjo, I. Selective arteriography in cancer of pancreas at resectable stage. *Am. J. Surg.*, 1971, *122*, 302–407.
57. Takashima, T., Yamamoto, I., Mitani, I., and Shin, M. Transfemoral superselective celiac angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 813–817.
58. Viamonte, M., Jr., Warren, W. D., and Fomon, J. J. Liver panangiography in assessment of portal hypertension in liver cirrhosis. *Radiol. Clin. North America*, 1970, *8*, 147–167.
59. Wakim, K. G., and Mann, F. C. Effect of experimental cirrhosis on intrahepatic circulation of blood in intact animal. *Arch. Path.*, 1942, *33*, 198–203.
60. Yü, C. Primary carcinoma of liver (hepatoma): its diagnosis by selective celiac arteriography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 142–149.
61. Zimmerman, H. Personal communication.

RELIEF OF OBSTRUCTIVE JAUNDICE THROUGH PERCUTANEOUS TRANSHEPATIC CATHETER—A NEW THERAPEUTIC METHOD*

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NONSURGICAL decompression of a biliary obstruction by percutaneous transhepatic catheter becomes imperative in selected patients when an immediate or even delayed reconstruction of the biliary system cannot be secured by operation. Both benign stricture and malignant tumor may produce obstructive jaundice; if it is caused by benign stricture of the common duct, it is due to surgical trauma in 97 per cent of the cases.[1] The subsequent, frequently multiple surgical interventions beginning with the primary repair and terminating in hepatojejunostomy, all may lead to stenosis in 30 to 35 per cent of the cases.[2] Recurrent ascending cholangitis, chronic hepatitis, uncontrollable pruritus, depression and occasional portal hypertension further aggravate the clinical condition which as a rule ends in hepatic failure and coma. If, in addition, the transhepatic cholangiogram shows high hepatic obstruction and no accessible segment for reanastomosis, then transhepatic biliary decompression (TBD) remains the only hope and remedy for the desperate condition of the unfortunate patient. This approach was introduced and performed in 11 patients during the last 4 years.

The purpose of this paper is to describe the technique of TBD and demonstrate the value of the procedure by representative cases in benign and malignant stenoses of the biliary system.

METHODOLOGY

After visualization of the biliary system on percutaneous cholangiogram, the films are immediately reviewed with the referring surgeon in order to decide whether reconstruction of the biliary duct would still be feasible or whether insertion of a catheter for biliary drainage is the only alternative to conservative medical management. At this time the biliary system still retains enough contrast medium to be visualized under fluoroscopy. The point of the intended entrance is locally anesthetized, usually within the 9th intercostal space in the midaxillary line, and the skin, subcutaneous tissue and the intercostal muscles are penetrated with a No. 16 needle to prepare a dilated tract within the thoracic soft tissue. This helps to avoid wrinkling and telescoping of the tapered soft tip of the teflon catheter. An 8 inch 18 thin-walled needle sheathed with a No. 7 teflon catheter is next inserted under fluoroscopic control. The tip of the catheter is attenuated and tapered to a 0.35 inch guide wire. It has 4 large side holes spiraling around the distal 5 cm. segment immediately in front of the 5 mm. long, tapered tip. The needle, sheathed with the catheter, is directed into a contrast-outlined anterior segment of the right hepatic duct. Advancement into the liver parenchyma is accomplished by constant rotation of the catheter while the needle is pressed in with variable force. The penetration of a relatively normal liver parenchyma requires only gentle pressure; however, quite forceful manipulation may be necessary to penetrate a fibrotic nodule or neoplastic tumor. After entering the contrast-outlined tributary of the right duct, a couple ml. of contrast

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agent is injected to locate the needle position. Now the teflon tube is advanced and after all the side holes are within the duct the needle is completely withdrawn. At this time a 0.025 inch guide wire having a curved tip is introduced and the teflon catheter is manipulated into a final suitable position within the obstructed portion of the duct. The catheter in this position will provide external biliary drainage with the contents collected in a plastic bag (TA-4 transfer pack). External drainage is not satisfactory in the long run; however, it may be applied for a few weeks prior to a contemplated surgical procedure.

Patients having extensive pruritus or disturbed sensorium due to the high serum bilirubin and impending hepatic coma usually respond very favorably in a few days to external drainage of the obstructed ductal system. Under such conditions we prefer to keep the patient on external drainage for a few days before attempting to advance the catheter through the stricture into the adjacent free biliary segment or anastomosed portion of the small intestine. The probing, dilatation and passage through a stenotic anastomosis usually required an end-open, preshaped, braided catheter. Therefore the relatively rigid teflon tube is replaced by a No. 7 F polyurethane catheter having the tip tapered to No. 5 F. After reaching the post-obstruction area with the tube, a curved-tip guide wire (0.025 to 0.035 inches) is advanced through the tube and the end-open polyurethane catheter is replaced with a teflon No. 7–8 F tube having multiple side holes. Depending on the demonstrated condition of the biliary system visualized on the cholangiogram and its connection to the intestinal tract, it will be necessary to provide a "custom made" catheter tailored to the individual problem. The bile obtained from the obstructed biliary system is frequently infected by a variety of microorganisms such as *Pseudomonas*, *Klebsiella* and *Enterococci*. As a result, even the major branches may be clogged by mucoid aggregates of bile sludge, precipitates and debris giving the initial, false impression of stones on the cholangiograms. Most of these sludge-like substances can be washed out by prolonged irrigation with normal saline solution. It may be necessary to continue irrigation a few days before the duct system is clear. The patient with internal drainage should not be sent home unless the extracted bile content is uniformly clear, the major ducts can be filled with contrast medium and the patency of the tube can be maintained for 24 hours without irrigation. Before discharge from the hospital the patient or a relative must be instructed to handle the daily lavage of the catheter under sterile conditions. The frequency of any follow-up visits depends on the individual case. The tube is changed at 3 month intervals, even in the absence of any symptoms or complaints.

REPORT OF CASES

Case I. V.G. This 23 year old female had cholecystectomy in another hospital in May 1970. Jaundice was noted 2 weeks later and her common duct was explored. Spiking fever, chills, jaundice and a 45 pound weight loss characterized her postoperative course. Following the second operation she was admitted to Ohio State University Hospitals in August 1970 and percutaneous cholangiogram showed obstruction of the common duct (Fig. 1*A*). The patient had severe pruritus, symptoms of depression and bile encephalopathy. A TBD catheter was positioned in the bile duct through an anterior subcostal approach in order to treat her jaundice and relieve her cerebral symptoms before undertaking surgical reconstruction (Fig. 1*B*). She responded well to external drainage and was sent home for 3 weeks. During this period, her symptoms and laboratory findings were markedly improved; her total bilirubin dropped from 22.4 to 11.2 mg./100 ml. The patient underwent an uneventful choledochojejunostomy in September 1970.

Case II. J.R. This 46 year old female underwent multiple surgical procedures in an outside hospital between 1969 and 1971. These included cholecystectomy with common duct exploration during 1969, and repair of the common duct in September 1969. One month later she under-

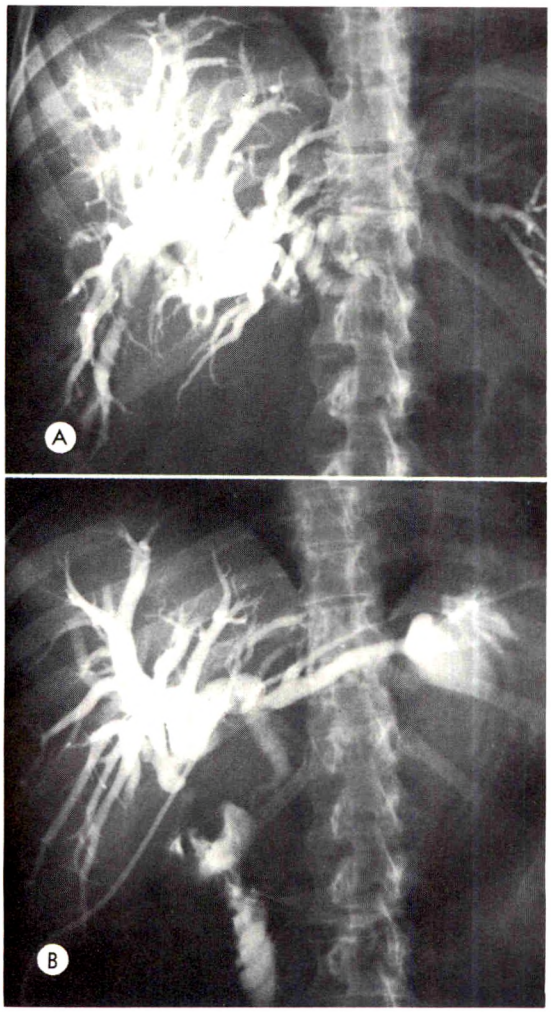


Fig. 1. Case 1. (*A*) Percutaneous transhepatic cholangiogram obtained August 13, 1970, showing complete obstruction of the early common hepatic duct. The intrahepatic ducts are greatly distended; some of the tributaries of the right and several of the left hepatic ducts are not filled. (*B*) Follow-up cholangiogram obtained 6 days later after daily irrigation through the inserted anterior subcostal transhepatic biliary decompression tube (TBD). The biliary system is less distended, much better filled and there is now evident flow through the choledochoduodenal anastomosis (compare with *A*).

went hepatoduodenostomy in Ohio State University Hospital followed by laparotomy for gastric hemorrhage in December 1969. The intestinal adhesions causing bowel obstruction had to be lysed in July, 1970 when her restenosed hepatoduodenostomy and abdominal hernia were also repaired. In January 1971 a Roux-en-Y type of hepatojejunostomy was performed utilizing the inferior segment of the left lobar duct. Unfortunately the obstructive jaundice recurred in 3 months and her direct bilirubin reached 8.9 mg./100 ml. at the time of her readmission on August 7, 1971. A percutaneous transhepatic cholangiogram at this time demonstrated obstruction of the hepatojejunal anastomosis (Fig. 2*A*). A TBD tube was inserted into the right lobar duct; however, it had to be removed 3 days later because of frequent hemobilia requiring transfusions. In view of the multiple unsuccessful surgical reconstructions, her debilitated general condition and her large abdominal incisional hernia, no new surgical intervention could be contemplated and she was sent home on medical therapy.

She was readmitted on June 30, 1972 and a percutaneous transhepatic catheter was successfully placed in the right anterior hepatic radical. The left inferior duct was almost completely obstructed just in front of the anastomosis due to multiple small stones, bile precipitation and debris. After 20 days of external drainage and irrigation, the total bilirubin had been reduced from 8.4 to 2.4 mg./100 ml. and considerable amount of debris had been removed (Fig. 2*B*). The stenotic anastomosis was then catheterized and moderately dilated. After positioning the guide wire into the jejunal segment, a teflon tube having side holes proximal, as well as distal to the site of the hepaticojejunal anastomosis was put in place (Fig. 2*C*). This provided the drainage from the biliary duct to the jejunum and permitted daily irrigation of the biliary system using normal saline solution. As of September 5, 1973 she has been having internal drainage for 16 months. During this period she gained 26 pounds and returned to normal sedentary life. It may be worth mentioning that she has been off any antibiotic medication for the past 15 months, although her bile cultures still show *Klebsiella* and *E. coli* as constant contaminants.

Case 11. B.S. This 39 year old female had cholecystectomy and exploration of the common duct elsewhere in 1955. When the T-tube was removed the common duct happened to be torn necessitating repair. Two years later she underwent a hepatoduodenostomy to relieve obstructive jaundice. Ever since this surgical reconstruction she has been having intermittent jaundice and epigastric and back pain.

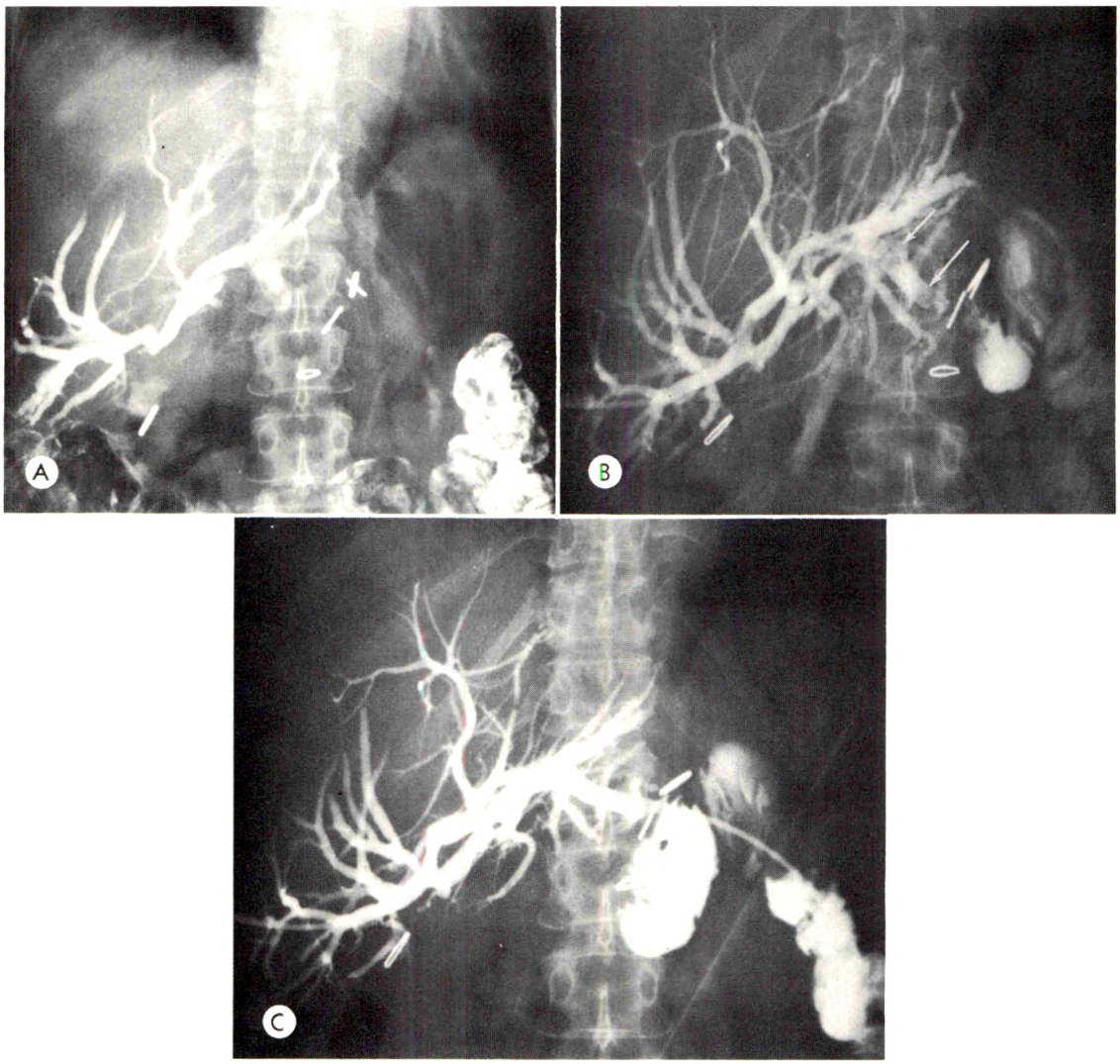


Fig. 2. Case 11. (*A*) Percutaneous transhepatic cholangiogram obtained October 7, 1971, shows an incompletely filled intrahepatic biliary system. The metal clips along the left side of L-1 vertebra indicate the site of the hepatojejunal anastomosis between the left inferior biliary segment and the Roux-en-Y jejunal anastomosis. A long portion of the duct is occluded in front of the anastomosis. (*B*) Cholangiogram on September 17, 1972, following injection through TBD catheter. Although several residual "stones" were still present (arrows), daily irrigations during the last 3 weeks apparently helped to open up many of the biliary tributaries, including the one which leads to the anastomosis with the jejunum. (*C*) Cholangiogram obtained after injection of contrast medium through the TBD tube which was advanced from the right to the left main duct, then through the left inferior segment and *via* the stenotic anastomosis into the efferent loop of the Roux-en-Y connection.

During the last year her condition was aggravated. She lost 22 pounds and developed a painful, enlarged liver as well as incessant gnawing pruritus and lower extremity pustules, requiring repeated and extended hospitalizations in other hospitals. She was admitted to Ohio State University Hospitals on January 29, 1973. Two days later a TBD tube was placed into the right hepatic duct across the stenotic hepatoduodenal anastomosis. The major biliary ducts were filled with numerous stones forming a solid cast (Fig. 3*A*). Her pruritus was completely relieved in 4 days. Her condition further improved and 5 months later, after repeated irrigations, a smaller number of stones and adequate drainage were demonstrated on cholangi-

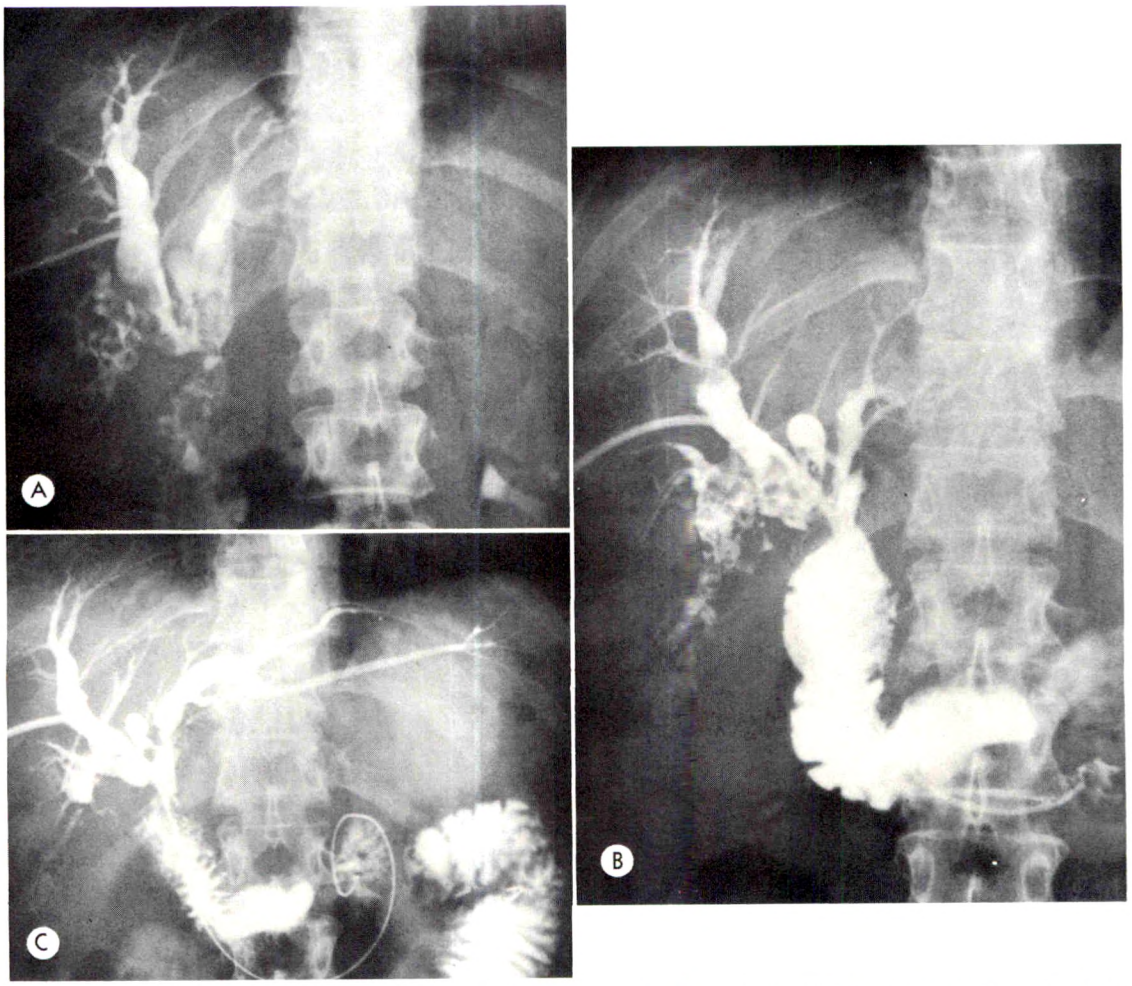


FIG. 3. Case III. (*A*) Cholangiogram obtained February 7, 1973, through the TBD tube placed into the right hepatic duct. The numerous stones from a cast-like mass which distends both right and left hepatic ducts and obstructs the hepatoduodenal anastomosis. (*B*) Follow-up angiogram 5 months later. The TBD tube now is placed across the anastomosis into the duodenum. The volume and number of the stones have appreciably decreased after 5 months of daily irrigation. (*C*) Follow-up cholangiogram after 7 months. Most of the stones have disappeared, the ducts in the left lobe have reopened.

ogram (Fig. 3*B*). During this period she has gained 24 pounds and serum levels of SGOT, SGPT and bilirubin returned to normal. However the alkaline phosphatase responded only moderately from 21.5 to 17 U/100 ml. The markedly enlarged liver that previously reached the iliac crest, now has normal size and her last follow-up cholangiogram demonstrated dramatic improvements. Most of her intrahepatic stones have disappeared, the left lobar duct reopened and all the ducts became less distended. Instead of extensive pain killing medication of 200 mg. of demerol daily and large doses of tranquilizers, she now takes only occasional aspirin tablets. No surgical reconstruction is expected unless the rest of the intrahepatic stones will not be dissolved by internal irrigation.

CASE IV. D.K. This 51 year old male had cholecystectomy and common duct exploration in 1965; 1 year later he had delayed removal of the T-tube. Subsequent recurrent attacks of cholangitis during the next 5 years prompted a re-exploration of the common duct. Stenosis of the duct and a duodenal biliary fistula were identified and repaired by establishing an hepatoduodenal anastomosis. The patient did well until 8 months ago, when he suffered occasional attacks of cholangitis, lasting for 2 or

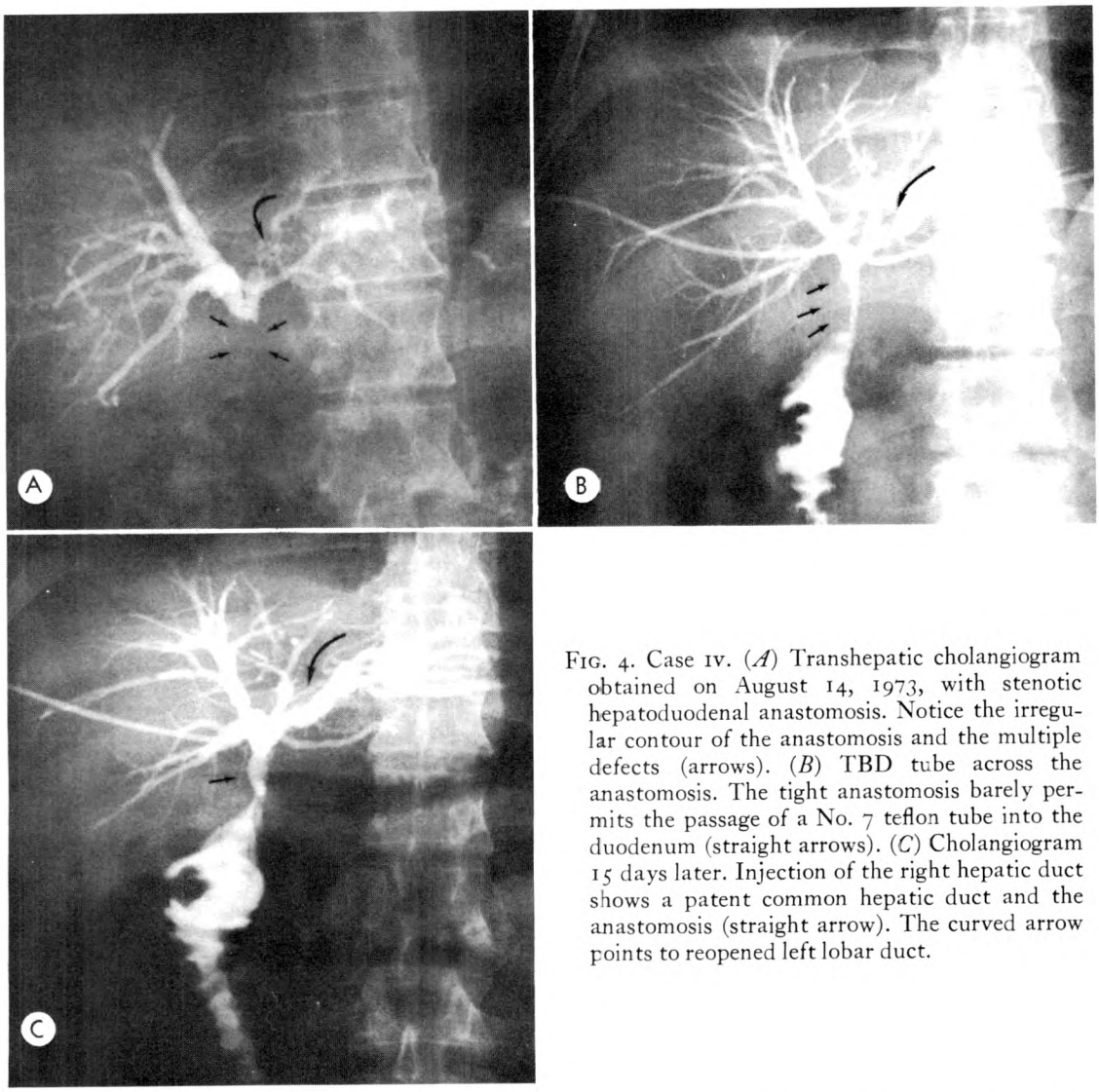


Fig. 4. Case IV. (*A*) Transhepatic cholangiogram obtained on August 14, 1973, with stenotic hepatoduodenal anastomosis. Notice the irregular contour of the anastomosis and the multiple defects (arrows). (*B*) TBD tube across the anastomosis. The tight anastomosis barely permits the passage of a No. 7 teflon tube into the duodenum (straight arrows). (*C*) Cholangiogram 15 days later. Injection of the right hepatic duct shows a patent common hepatic duct and the anastomosis (straight arrow). The curved arrow points to reopened left lobar duct.

3 days. They gradually increased in frequency to approximately one attack every other week. During these periods he had lower extremity myalgia, spiking fever, jaundice and clay-color stools.

After admission to Ohio State University Hospitals on August 8, 1973 his temperature was elevated to 100°F., hemoglobin was 14.4 gm. per cent, white blood cell count 9,500 mm.^{3}, prothrombin time 100 per cent, total serum bilirubin 3.2 mg./100 ml., alkaline phosphatase 16.2 units, SGOT 98 and SGPT 126.

Obstruction of the proximal hepatic duct was demonstrated on the cholangiogram of August 14, 1973. A TBD catheter was positioned just proximal to the stenosis (Fig. 4*A*), and then advanced across the stenosis into the duodenum (Fig. 4*B*). During the next 6 days he had daily irrigation which resulted in removal of more than 400 ml. of sterile, under microscope amorphous appearing, suppurative material. An apparent subhepatic abscess which seemingly communicated through a fistulous tract with the anastomosis was readily drained through the indwelling TBD tube.

During the same 6 days this patient had temperature elevations to 103°F. and some pain in his back and right shoulder. The latter was probably caused by the leakage of small amounts of infected bile along the inserted TBD tube into retroperitoneal area toward the diaphragm (Fig. 4*B*).

His bile cultures from the beginning showed contamination with *Klebsiella* organism. He responded well to antibiotic therapy (Gantamycin 200 mg. q.d.); his fever, shoulder pain and his back pain all disappeared in 6 days. The catheter was then withdrawn and positioned proximal to the stenosis. It drained clear bile to the outside. The external biliary drainage stopped completely on August 29, 1973. A cholangiogram was obtained through the TBD under direct television-controlled monitor. It showed prompt and free flow of the contrast medium through the hepatoduodenal anastomosis (Fig. 4*C*). Evidently the drainage of the old sterile abscess relieved the compression on the duct permitting flow of bile into the duodenum. In addition the irrigation also helped to clear out the interhepatic ducts. The No. 7 drainage catheter was replaced with a No. 5 catheter and even that was removed on September 1, 1973.

Following removal of the catheter there was no bile discharge through the transhepatic duct. The entrance site healed in 3 days and the patient was discharged having no symptoms. Laboratory studies showed normal serum bilirubin, alkaline phosphatase 4.7 units and normal SGOT and SGPT enzyme values. On September 24, 1973 the patient was free of symptoms and returned to normal activity.

MATERIAL, SELECTION OF PATIENTS

Five male and 6 female patients were treated for obstructive jaundice with decompression. Six had benign recurrent stricture of the biliary system; malignant tumor was responsible for the jaundice in 5. Table I summarizes the clinical information on these 11 patients.

All of these surgical patients were primarily referred to us for visualization of the biliary structures by percutaneous cholangiography. In 2 patients with benign stricture the transhepatic drainage tube was inserted to improve the patients' conditions prior to the contemplated surgical repair (Case I and VI). In the remainder of the patients, the condition of the biliary system at the time of cholangiography was thought to preclude further surgical intervention for the immediate or foreseeable future.

The indications to treat benign strictures by this method are listed in Table II. The first indication applied to patients who represent high surgical risks because of their high serum bilirubin content and impaired liver function. The biliary drainage is maintained only for a temporary period of 2 to 4 weeks in preparation for corrective biliary surgery. During this period the serum bilirubin level can markedly be reduced, the intrahepatic ducts freed of the obstructing debris and soft "pseudo-stones" by daily irrigations. Free flow through the opened intrahepatic ducts should be assured prior to repeated biliary reconstructive surgery.

The procedure is most rewarding for the unfortunate patients falling into the second group. These patients are all beyond the reach of surgical reconstruction either because the very last available segment has been utilized for the anastomosis and has

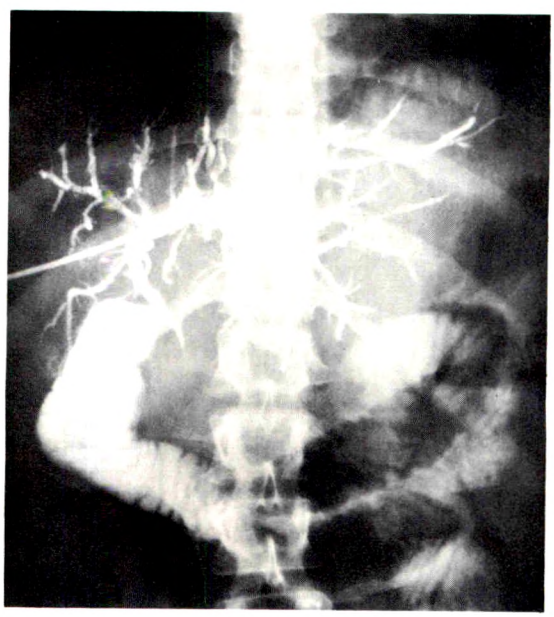


FIG. 5. Case V. Cholangiogram and placement of TBD tube in a 26 year old female, who had 2 years prior to this study a choledochoduodenal anastomosis to relieve her obstructive jaundice caused by sclerosing cholangitis. The over-all narrow ducts have irregular contour and short partially obstructed segments. The common hepatic duct is almost completely occluded. The numerous scattered minute defects represent bile debris. The angiographic findings correlated exactly with the findings at autopsy obtained 4 days later.

Table I

PATIENTS UNDERGOING TRANSHEPATIC BILIARY DECOMPRESSION (TBD)

Case	Age	Sex	Site of Stenosis	Pruritus	Changes in Bilirubin (mg./100 ml.)	Duration of Drainage	Clinical Results	Remarks
				A. Benign Postoperative Strictures				
I. V.G.	23	F	HD and Lt. HD	−	22.4→11.4 21 days	3 weeks	Excellent	Relieved of hepatic encephalopathy
II. J.R.	46	F	Occluded CD jejunal anastomosis	++++	8.4→1.4 26 days	Since June 1972	Excellent	Returned to normal activity
III. B.S.	39	F	HD-duodenal anastomosis	++++	3.8→1.2 12 days	Since February 1973	Good	Many intrahepatic stones
IV. D.K.	51	M	HD-duodenal anastomosis	−−−−	3.2→1.2 22 days	22 days	Excellent, stenosis reopened	Old abscess drained
V. E.S.	26	F	Sclerosing ducts	+++	32.8	4 days	Poor, hepatic coma persisted	Died—renal failure; septicemia
VI. C.M.	48	F	CD-duodenal anastomosis	++	6	1 day	Tube fell out by accident	HD-jejunal anastomosis 1 day later
				B. Malignant Obstructive Lesions				
VII. G.S.	64	M	Biliary Ca Rt. HD intrahepatic	++++	11.5→7.2 55 days	63 days external	Good	Drained only right HD; died—uremia
VIII. T.Th.	33	M	Ca pancreas CD	++++	3.8→1.4 4 days	22 days	Fair	Drainage discontinued, due to hemobilia
IX. L.S.	52	M	Ca pancreas CD	−−−−	13→11.4	21 days internal	Poor	Died—hepatorenal failure
X. B.G.	75	F	Ca pancreas CD	+++	16.2→9.3	12 days internal external	Poor	Has removed tube, died in hepatic coma
XI. M.D.	80	M	Ca colon HD metastasis to porta hepatis	++++	21.8→16 5 days	15 days internal	Good	Symptoms relieved, died at home

Abbreviations: Carcinoma (Ca); Common duct (CD); Hepatic duct (HD); Left (Lt.); Right (Rt.).

subsequently become obstructed (Case II) or other considerations such as excessive high risk factor.

The multiple intrahepatic biliary stones usually cannot be removed at surgery.[2] This method permitted direct irrigation through the tube utilizing normal saline, heparin or bile salt solutions which resulted in dissolution of many large solid stones in 1 of our patients (Case III).

Table II

INDICATIONS FOR TBD IN BENIGN STRICTURES

1. Postoperative biliary duct stricture with complications
2. Restenosed hepatoduodenal or jejunal anastomosis
3. Obstruction and intrahepatic "stones"

The indications for the procedure in malignant disease are listed in Table III. Naturally the procedure in this group of patients is strictly palliative (Fig. 7, *A* and *B*; and 8). Good renal function is a prerequisite in this group of patients. All of these tumors were not amenable to surgery because they were either recurrent, involved multiple organs or had a deep seated location within the liver. Most of these patients were ready to accept jaundice for a long time and only the incessant pruritus and the progressive malaise associated with the impaired liver

TABLE III

INDICATIONS FOR TBD IN MALIGNANT TUMORS

1. Intrahepatic Obstruction
   A. Primary biliary tumors
   B. Single hepatic metastasis
2. Extrahepatic Obstruction
   A. Recurrent primary biliary tumor
   B. Recurrent pancreatic tumor
   C. Single porta hepatis metastasis

function forced them to seek medical relief. A successfully introduced drainage tube usually results in relief of malaise and pruritus in a few days and the rapidly improving liver profile atests to the value of the method. The patient is sent home usually within 10 days with the tube in place and will be instructed in daily irrigations.

CONTRAINDICATIONS TO TBD

Hepatorenal failure rules out intrahepatic positioning of a tube for either external or internal drainage in patients with malignant obstruction. Normal renal function is an essential prerequisite in this group. If renal dialysis is available, it should be initiated before placement of TBD tube in patients with hepatorenal failure, in whom the biliary obstruction is due to benign stricture. Antibiotic coverage before and after the procedure is mandatory in patients with pending or existing renal failure. In our experience septicemia is the most common and tragic complication in this group.

A significant delay in clotting mechanisms should be corrected before transhepatic insertion of the drainage tube.

Sclerosing cholangitis, a poorly understood disease of unknown etiology, causes multiple obstructions within the biliary system without dilatation of the ducts (Case v; Fig. 5). The value of this treatment is questionable, at least in this condition in view of the numerous obstructions within the biliary system these patients have on the cholangiogram and also at autopsy. Unfortunately our patient came to us in hepatic coma and she quickly developed uremia, septicemia and profuse bleeding before she expired on the fourth day of treatment with TBD.

COMPLICATIONS

Most of the patients had fever for a couple of days running up to 102° F. following the cholangiography and placement of the catheter. The temperature elevation can readily be explained first on the basis of temporary bacteremia caused by the backflow of the infected bile into the liver sinusoids with consequent bacteremia. The other possible reason is the occasional escape of bile along the inserted catheter into the retroperitoneal space. None of our patients developed bile peritonitis or required "backup surgery" following placement of the catheter. The patient should be premedicated with antibiotics beginning the night before the intended procedure (Ampicillin 500 mg. for 24 hours). The selection of the proper antibiotic agent for the post-TBD treatment depends on the sensitivity studies of the organisms found in the bile culture. It is interesting to note that the percutaneously inserted TBD tube can safely be maintained within the intrahepatic biliary system and across the hepatojejunal anastomosis for many months without internal medication or antibiotic therapy (Case II and III). These patients had no antibiotic medication for the last 15 and 6 months, respectively.

Hemobilia requiring transfusions was the second most common complication; it occurred in 2 patients (Case II and VIII). The catheter may pass through the lumen of a portal vein branch before it enters into a major bile duct. If a communication is established between these 2 structures, profuse bleeding may occur, which may not be controlled without relocating the course of the catheter. At least it must be assured that no communicating side holes of the TBD tube are left in the vicinity of a hepatic blood vessel. The proper position should be tested using injection of contrast material through the TBD catheter under direct television monitored fluoroscopy and

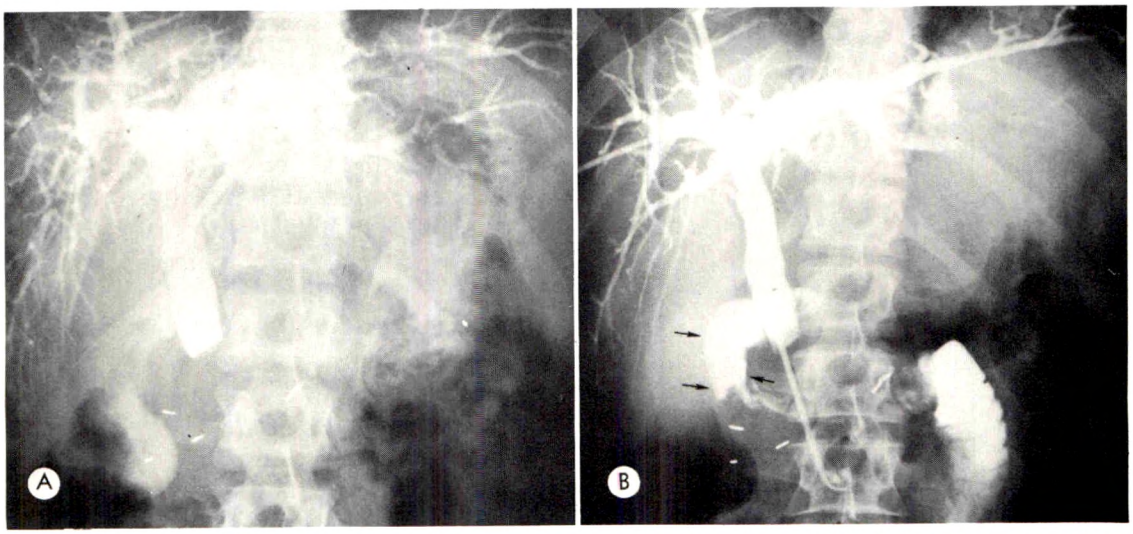


FIG. 6. Case VIII. (*A*) Transhepatic cholangiogram in a 33 year old male who had choledochoduodenostomy $2\frac{1}{2}$ years prior to this study to relieve the obstructive jaundice caused by pancreatic carcinoma. There is complete obstruction of the distal common duct by the tumor. (*B*) TBD tube is advanced through the common duct into the second and third portion of the duodenum. The tumor invades the third and second part of the duodenum. The arrows point to the early second portion of the duodenal loop.

on serial films. No intrahepatic blood vessel should be opacified during the test injection.

Incidental or intentional removal of the tube occurred in 2 patients (Case IV and VIII) requiring 2 separate reinsertions in a comatose patient (Case VIII; Fig. 6, *A* and *B*) with proven pancreatic carcinoma. The proper position of the tube, once it is established, is assured by maintaining a

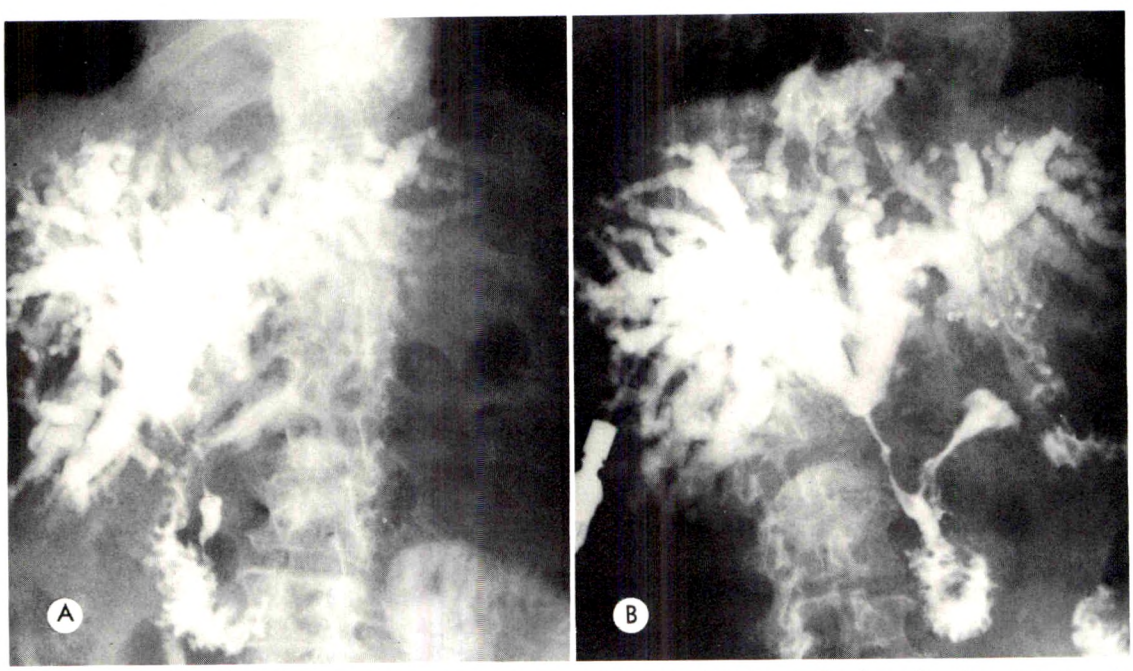


FIG. 7. Case X. (*A* and *B*) Cholangiograms in a 75 year old female with obstructive jaundice due to pancreatic carcinoma which obstructs the common duct. The distal tip of the tube is beyond the site of obstruction.

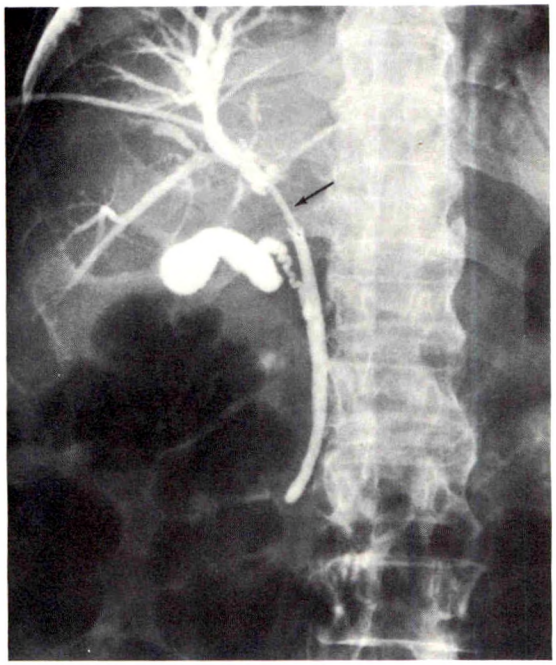


FIG. 8. Case XI. Cholangiogram obtained after injection through the TBD tube which is advanced through the right hepatic duct into the common hepatic duct. The metastatic tumor, located in the porta hepatis, obstructed the right and the left hepatic ducts. The left hepatic duct is still occluded; however, the right hepatic duct drains freely through the tube which is passing through the obstructed segment (arrow).

tight relationship between the skin at the site of insertion and a flat rubber collar which holds the tube in position.

DISCUSSION

Surgical or conservative therapy of a stricture of the common, right or left hepatic ducts, especially if it is located high in the porta hepatis, represents a difficult, occasionally insurmountable problem. Unless the obstruction is relieved and free biliary flow is established it will result in death. Surgical repair is associated with an appreciable mortality and morbidity. The repair of unselected biliary strictures may result in 13 per cent surgical mortality and 25 per cent major complications.[1] An unsatisfactory or bad result was reported to be as low as 25 per cent after the initial repair; the rate of failure increases to 35 per cent, if all of the needed follow-up reconstructions are included.[2] After each failure, the accessible biliary segment becomes shorter with more fibrosis around it. This leads to increased technical difficulties, all of which result in a progressively higher failure rate after each succeeding procedure.

Surgical insertion of various internal stents into the anastomosis to maintain adequate lumen and bile flow has long been advocated as highly beneficial for 6 to 12 months after surgical repair of a stricture. Straight, T shaped and various Y formed tubes made of rubber or silicone have been inserted during surgery, assuming open drainage through the abdominal wall. Surgical insertion of *transhepatic* straight or Y shaped rubber tube, *assuring transhepatic external drainage* in high subhepatic or intrahepatic strictures was also recommended before and used successfully.[3] The treatment of TBD described here is intended to assure adequate biliary drainage through a teflon tube, which is inserted through the anastomosis *without laparotomy*. It is not a diagnostic technique, but rather a therapeutic approach to relieve the jaundice and pruritus, while permitting the liver to recuperate. The amount of bile mud, debris and the stones can and must be removed by irrigations (Case III). The long standing daily irrigations and medication of the intrahepatic ducts through the TBD tube are the *only alternative* to increasing jaundice, which eventually leads to hepatic failure and death.

SUMMARY

The prognosis, following multiple unsuccessful surgical reconstructions of an obstructed biliary system, used to be very disappointing, because of the recurrent hepatitis, increasing jaundice and hepatic failure, which led to eventual coma and subsequent death.

Percutaneous transhepatic decompression of the biliary system, a new thera-

peutic method, may provide a lasting solution or at least temporary relief of the misery of the patient and improve upon the failing condition of the liver.

In patients having malignant obstructive lesions this therapeutic method has very limited value.

Under benign conditions TBD may offer the only life saving treatment with such a satisfactory clinical recovery that patients prefer the inconvenience of an indwelling tube to another surgical repair.*

* Since submitting this paper, the longstanding drainage tubes in Case II and Case III were removed on June 20, 1974, and April 30, 1974, respectively. All stones in Case III have disappeared. Both of these patients remain free of biliary symptoms as of August 10, 1974.

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REFERENCES

1. Smith, R. Hepaticojejunostomy with transhepatic intubation. *Brit. J. Surg.*, 1964, *57*, 186–194.
2. Warren, K. W., and McDonald, W. M. Facts and fiction regarding strictures of extrahepatic bile ducts. *Ann. Surg.*, 1964, *159*, 996–1010.
3. Way, L. W., and Dunphy, J. E. Biliary strictures. *Am. J. Surg.*, 1972, *124*, 287–295.

RECURRENT PYOGENIC CHOLANGITIS IN CHINESE IMMIGRANTS*

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RECURRENT pyogenic cholangitis, also known as cholangiohepatitis, oriental cholangiohepatitis, and Hong Kong disease, is prevalent in South East Asia. In Hong Kong after acute appendicitis and perforated peptic ulcer, it is the third most common cause of an acute abdomen in Chinese people requiring emergency admission.[6] The disease is occasionally seen in North America and we have encountered it in 5 Chinese immigrants over the past 3 years. In 2 patients it was diagnosed preoperatively on the basis of cholangiographic findings.

REPORT OF CASES

CASE I. S.C.L. A 41 year old man presented with acute right upper quadrant pain. He had had similar pain in the past and had had a cholecystectomy and exploration of the common bile duct 2 years previously. On admission, he had scleral icterus and a temperature of 38°C. Abdominal examination revealed right upper quadrant tenderness, but no palpable organs or masses. Pertinent laboratory findings were: hemoglobin 15.1 gm. per cent; white blood cell count (WBC) 12,300 per cu. mm.; bilirubin 5.9 mg. per cent; alkaline phosphatase 180 I.U. (15–80);* SGOT 151 I.U. (1–36); and serum amylase 55 S.U. (40–170).

The intravenous cholangiogram (Fig. 1*A*) and the T-tube cholangiogram (Fig. 1*B*) obtained after his cholecystectomy 2 years previously were reviewed. The abnormalities in the biliary tree were recognized as those of recurrent pyogenic cholangitis.

At operation the common bile duct was dilated to 3 cm. in diameter. It contained many large soft stones up to 2.5 cm. in diameter. Open liver biopsy showed the intermediate and large portal tracts to be notably widened and prominent in contrast to the terminal triads which were small and often difficult to find. In the widened portal tracts, the fibrosis was predominantly periductal and was accompanied by a chronic inflammatory cell infiltrate (Fig. 1, *C* and *D*). The hepatic parenchyma was normal including the terminal liver cell plates. An end-to-side choledochoduodenostomy was performed.

CASE II. F.T.W. A 69 year old woman had had a cholecystostomy 40 years ago and had been troubled with recurrent bouts of epigastric pain since. On admission she was afebrile and clinically anicteric. She had right upper quadrant abdominal tenderness, but no palpable organs or masses. Laboratory work-up revealed hemoglobin 12.6 gm. per cent; WBC 11,600 per cu. mm.; bilirubin 3.7 mg. per cent; alkaline phosphatase 100 I.U. (15–50); SGOT 53 I.U. (1–36); and serum amylase 65 S.U. (48–120).

Intravenous cholangiogram (Fig. 2*A*) showed a markedly dilated common bile duct and stones in the common hepatic and common bile ducts. A preoperative diagnosis of recurrent pyogenic cholangitis was made and confirmed by operative cholangiography (Fig. 2*B*).

At operation, she was found to have choledocholithiasis and a markedly dilated common bile duct. Cultures of bile grew *E. coli*. Cholecystectomy and side-to-side choledochoduodenostomy were performed.

CASE III. M.C. A 67 year old woman had had attacks of severe epigastric pain radiating to her back for many years. On admission, she was clinically jaundiced and had a fever of 38.2°C. Abdominal examination revealed right upper quadrant tenderness with involuntary guarding and a palpable liver edge 2 fingerbreadths below the costal margin. Laboratory values were: hemoglobin 11.2 gm. per cent; WBC 23,900 per cu. mm.: bilirubin 7.6 mg. per cent; alkaline phosphatase 100 I.U. (15–50); SGOT 200 I.U. (1–36); and serum amylase 160 S.U. (40–170).

At operation the common bile duct was found to contain biliary sludge mixed with foul,

* Normal range given in brackets. These vary slightly as the laboratory standards changed during the period of study.

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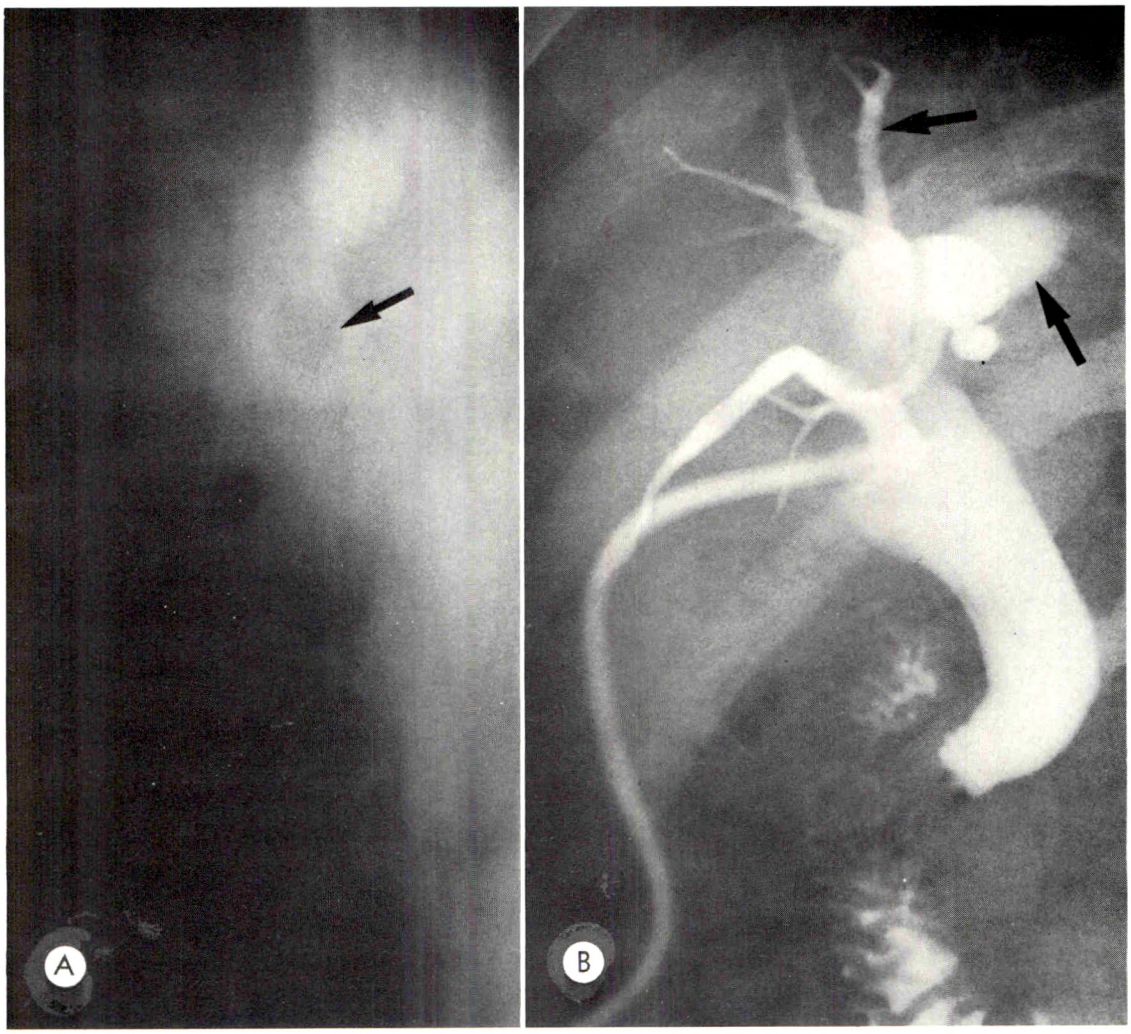


Fig. 1. Case 1. (*A*) Intravenous cholangiogram shows dilated common bile duct and common hepatic duct. A large radiolucent stone (arrow) is demonstrated in the latter. (*B*) T-tube cholangiogram. The common bile duct and intrahepatic duct (lower arrow) are dilated. The upper arrow shows moderately dilated bile duct with short branches arising at right angles to the duct.

murky bile, cultures of which grew *E. coli*. The gallbladder was small and fibrotic. A cholecystectomy was performed and the bile duct was explored and irrigated. T-tube drainage was maintained for 6 weeks. Operative and T-tube cholangiograms (Fig. 3, *A* and *B*) revealed changes of recurrent pyogenic cholangitis.

Case iv. Y.W. A 55 year old woman presented with epigastric pain radiating into her back. A cholecystectomy had been done 12 years previously. Shortly before admission she developed fever and chills and on admission her temperature was 39.7°C. She was clinically jaundiced. Abdominal examination was unremarkable. Her laboratory investigations revealed a hemoglobin of 8.4 gm. per cent, and WBC of 6,600 subsequently rising to 18,900 per cu. mm. Her bilirubin rose to 3.1 mg. per cent. Other values were: alkaline phosphatase 435 I.U. (15–50); SGOT 164 I.U. (1–36); and serum amylase 2,600 S.U. (40–170). Repeated blood cultures were negative. Ova of *Clonorchis sinensis* were present in her stools.

Exploration and T-tube drainage of the common bile duct were performed. At operation, her common bile duct was found to be full of inspissated mucoid material. T-tube cholangiograms (Fig. 4) revealed an abnormal biliary tree with changes typical of recurrent pyogenic

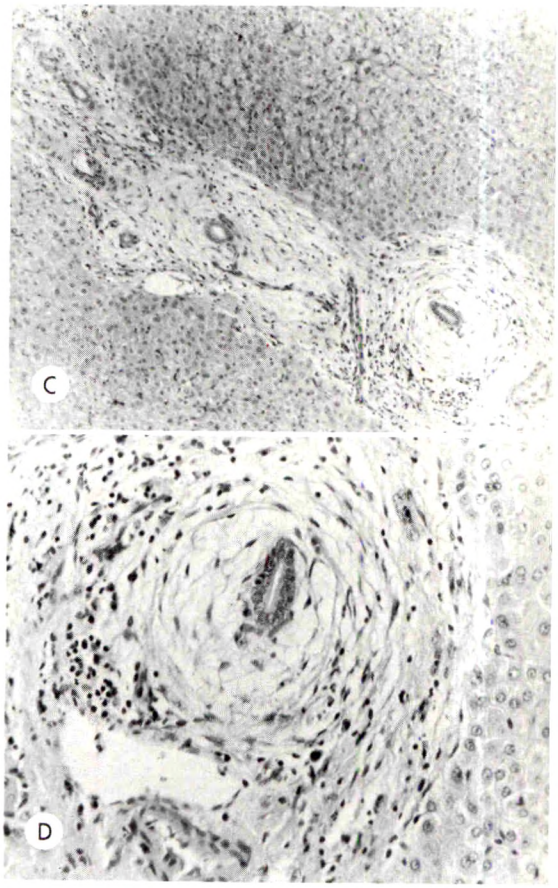


Fig. 1. (*C*) Portal and periportal region of liver. The portal area is widened by fibrosis and is sharply demarcated from the parenchyma. The fibrosis is predominantly periductal and is accompanied by an inflammatory cell infiltrate. The parenchymal liver cells are normal. (Hematoxylin and Eosin, ×210.) (*D*) Higher magnification of a portion shows periductal edema and concentric fibrosis. The inflammatory cells are predominantly lymphocytes and plasma cells. The terminal hepatic plate is intact. (Hematoxylin and Eosin, ×450.)

cholangitis. Liver biopsy showed acute purulent cholangitis.

Case v. Y.G. An 84 year old man presented with vomiting and right upper quadrant pain. He had a history of recurrent pancreatitis. He was anicteric. There was right upper quadrant tenderness with guarding and the liver edge was palpable 2 cm. below the costal margin. Twenty-four hours after admission he developed shaking chills and his temperature rose to 39.8°C. Laboratory values at that time were: hemoglobin 13.7 gm. per cent; WBC 14,100 per cu. mm.; bilirubin 4.0 mg. per cent; alkaline phosphatase 150 I.U. (15–50); SGOT 200 I.U. (1–36); and serum amylase 270 S.U. (40–170). Blood cultures grew gram negative bacilli.

At operation, the gallbladder contained many stones and frank pus. The head of the pancreas was indurated. Cholangiograms revealed an abnormal biliary tree. A cholecystostomy was performed. Three months later the gallbladder was removed and the T-tube cholangiogram (Fig. 5) revealed mild changes of recurrent pyrogenic cholangitis.

DISCUSSION

CLINICAL FEATURES AND LABORATORY FINDINGS

The disease usually occurs in young adults, 30–40 years of age. Males and females are affected equally. Both the age and sex incidence contrast to the usual gallbladder disease which occurs more commonly in older people and females. Clinically, the disease is characterized by recurrent attacks of right upper quadrant pain, fever, chills and jaundice. Each episode may last several days and then subside gradually. However, the initial attack or any subsequent attacks may produce severe septicemia and obstructive jaundice requiring immediate surgical drainage of the common bile duct. Physical examination reveals tenderness and rigidity in the right upper quadrant. The liver is enlarged in about 60 per cent and the spleen is palpable in about 25 per cent of patients.[3]

Our patients presented at a later age than is usual in this disease (4 were over 55). This is probably because milder cases present later, and patients more severely affected are unlikely to emigrate. In addition, diagnosis was delayed in Cases III, IV, and V due to our previous unfamiliarity with the disease.

The laboratory findings reflect the clinical features of sepsis and obstructive jaundice. Leukocytosis is usually present. Serum bilirubin and alkaline phosphatase are both elevated. Other liver function tests are usually normal. Cultures of bile obtained at operation show *E. coli* infection in most patients.

Roentgenologic findings before operation are usually unrevealing. Occasionally, air is seen in the biliary tract because of

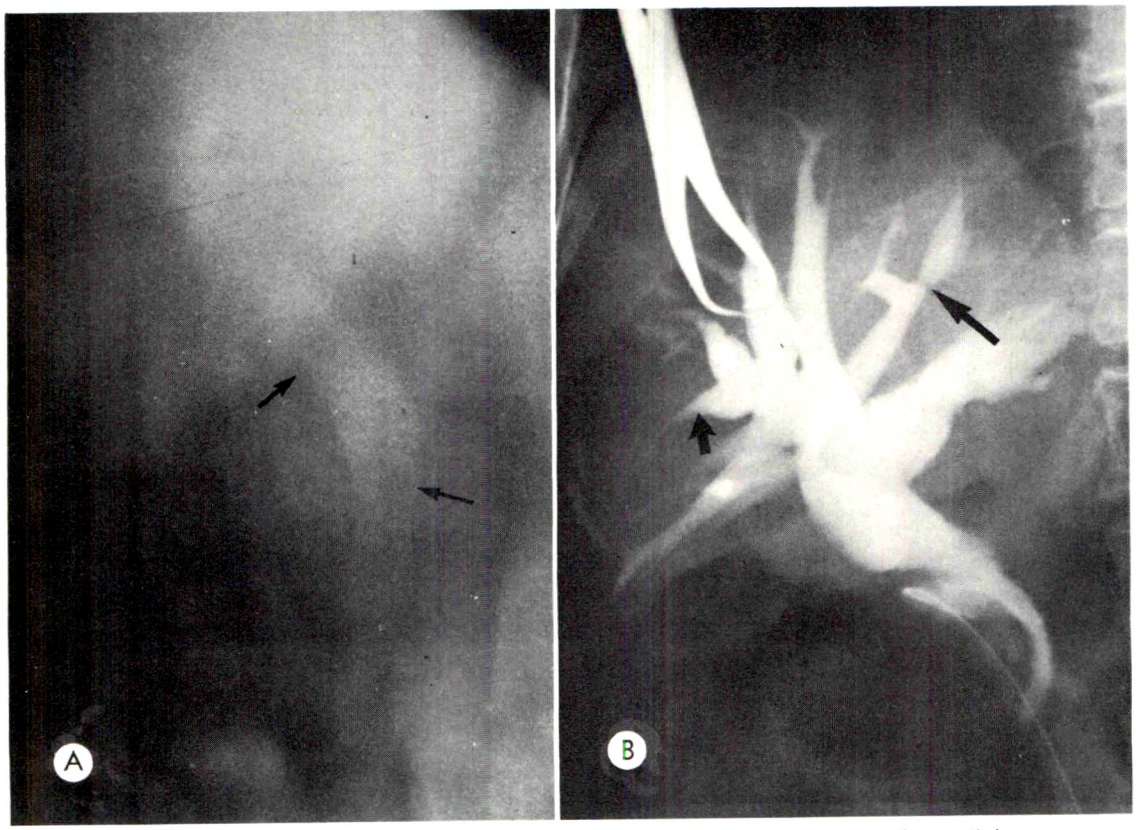


Fig. 2. Case 11. (*A*) Intravenous cholangiogram shows a dilated common bile duct with 2 radiolucent stones, one in the common hepatic duct and the other in the common bile duct (arrows). (*B*) Operative cholangiogram shows markedly dilated intra- and extrahepatic ducts. Note the typical "arrowhead" sign (short arrow) and the stricture (long arrow).

fistula formation or an incompetent sphincter.[4] Oral cholecystography shows either poor function or nonvisualization of the gallbladder because of the elevated serum bilirubin. Intravenous cholangiography sometimes shows a dilated common bile duct with radiolucent stones. Operative and T-tube cholangiograms are characteristic[8] and are described below.

FINDINGS AT OPERATION

At operation the liver is invariably enlarged and congested. Liver biopsy shows distinct features. There is a marked increase in fibrous tissues around the portal tracts, especially around the bile ducts. Infiltration by inflammatory cells is seen in the portal tracts and the adjacent liver parenchyma. The gallbladder may be enlarged and may show chronic inflammatory changes. Stones in the gallbladder are seen in 15–20 per cent of patients. The common bile duct is usually dilated. Gross dilatation up to 10 cm. has been reported. The walls are usually thickened by fibrosis, but occasionally are thin and elastic. Stones are usually present and may be found in any part of the biliary tract.

PATHOGENESIS

The exact pathogenesis of the disease is uncertain, although most authors agree that stones are formed primarily in the bile ducts. Stock and Fung[6] believe that *Clonorchis sinensis* infestation is the primary cause, resulting in stone formation, biliary obstruction, stasis and infection. Ong *et al.*[4] on the other hand believe that the primary cause is portal septicemia resulting from poor eating habits, and that *Clonorchis sinensis* need not be present in all cases of recurrent pyogenic cholangitis.

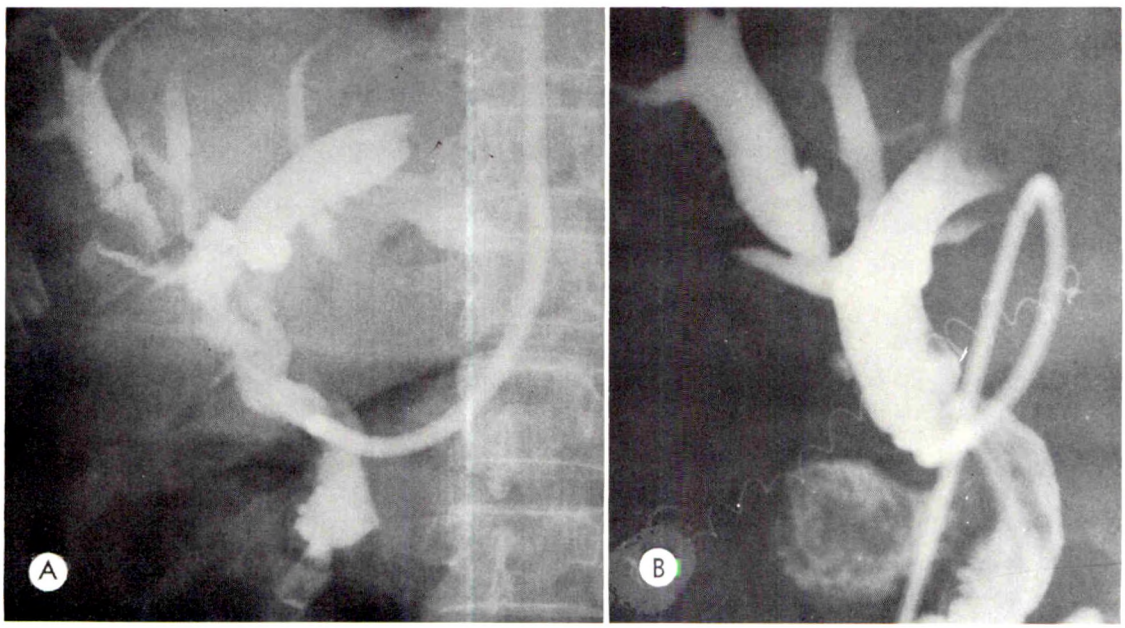


FIG. 3 Case III. (*A*) Operative cholangiogram. (*B*) T-tube cholangiogram. Note that only a few grossly dilated intrahepatic radicals are present. The proximal part of the left hepatic duct is obstructed by a radiolucent stone.

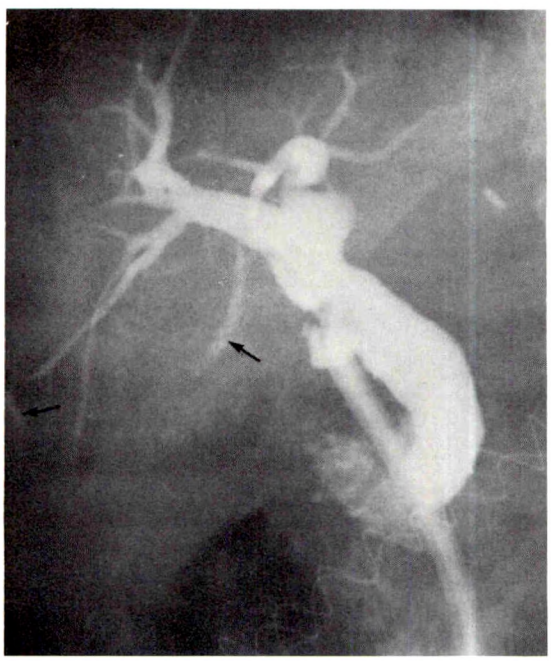


FIG. 4. Case IV. T-tube cholangiogram. Note the lack of branching in the right intrahepatic ducts and the appearance of stretching of these ducts. The left intrahepatic ducts are relatively normal.

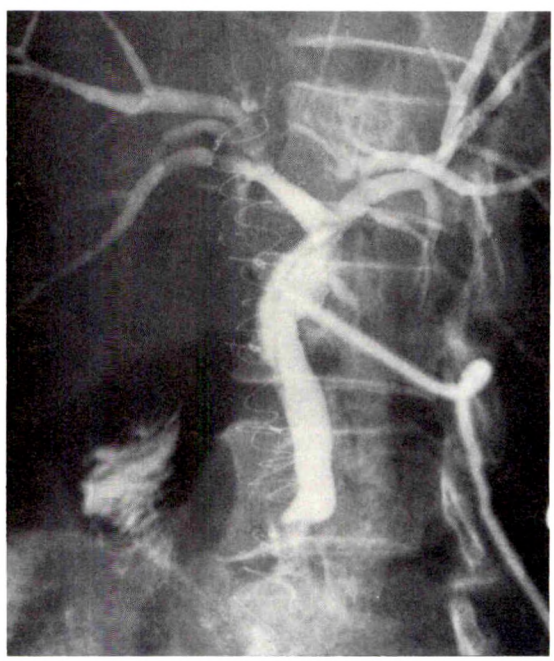


FIG. 5. Case V. T-tube cholangiogram. A dilated common bile duct and mild dilatation of 2 intrahepatic branches are noted (arrows).

CHOLANGIOGRAPHIC FINDINGS

Although characteristic, the roentgenologic manifestations of this disease have been neglected in the literature until recently.[8]

Typical findings include:

1. Decrease in arborization of intrahepatic radicals (Fig. 1*B*; 2*B*; 3, *A* and *B*; and 4*A*).

2. Abnormal branching pattern. The normal branching angles are distorted and may be at right or obtuse angles (Fig. 1*B*). In some cases (Fig. 4*A*) the branches appear to be stretched.

3. Asymmetric changes in the bile ducts. In the same cholangiogram, some intrahepatic ducts may be dilated throughout, while others may show proximal dilatation with rapid tapering towards the periphery. This latter appearance is not unlike that of an arrowhead (arrow in Fig. 2*B*) and we refer to this as the "arrowhead sign."

4. Radiolucent stones are usually present. They may be situated anywhere in the biliary tree (Fig. 1*A*; 2*A*; and 3, *A* and *B*).

5. A dilated common bile duct is usually present. It may measure 3–4 cm. in diameter.

The decrease in the number of intrahepatic radicals is probably due to fibrosis in the portal tracts and the abnormal branching patterns to scarring in the liver. The asymmetric dilatations and strictures are a reflection of chronic inflammatory changes in the intrahepatic bile ducts with or without obstruction. As the degree of obstruction and inflammation varies in different parts of the liver, the changes in the bile ducts differ accordingly.

DIFFERENTIAL DIAGNOSIS

Although the cholangiographic changes in recurrent pyogenic cholangitis are quite characteristic, the following conditions, which produce changes similar in some respects, must be considered in differential diagnosis:

1. *Extrahepatic obstruction.* The hallmark of uncomplicated extrahepatic obstruction is generalized dilatation of the entire biliary tree. The full complement of intrahepatic bile ducts is shown in an operative cholangiogram. Changes of cholangitis may be present, but never to the same degree as in recurrent pyogenic cholangitis, where repeated attacks over a long period of time are the rule.

2. *Sclerosing cholangitis.*[1] This is a disease of unknown etiology associated with submucosal inflammation of portions of the bilt ducts and characterized by fibrosis and stenosis of these regions. Clinically, the patient presents with either intermittent or progressive jaundice. Many patients also have chronic ulcerative colitis. The condition is usually first diagnosed at operation when the common bile duct feels like a thickened cord. Operative or postoperative cholangiography shows the extent of the disease. A characteristic finding is decreased arborization of intrahepatic bile ducts as in recurrent pyogenic cholangitis. However, there is a lack of dilatation proximal to the strictured areas and the common bile duct may also have strictures. Both these features distinguish the condition from the Asiatic disease.

3. *Caroli's disease.* This is a developmental anomaly in which there is segmental saccular dilatation of the intrahepatic bile ducts. This results in stasis, cholangitis, liver abscess and stone formation. The clinical picture is similar to recurrent pyogenic cholangitis, but it occurs in a younger age group. It also carries a much poorer prognosis. Most patients die at an early age as the disease is not amenable to any form of satisfactory treatment. The cholangiographic finding[2] of dilated saccules in intrahepatic bile ducts establishes the diagnosis.

MANAGEMENT

In the western hemisphere, the majority of common bile duct stones originate in the gallbladder, only a few forming primarily in the ducts.[5] The surgical treatment involves exploration of the common bile duct and extraction of the stones. Seldom is a major surgical procedure for internal drainage required.

In recurrent pyogenic cholangitis, repeated attacks are the rule and recurrent formation of stones in the ducts is the main problem. Those who fail to respond to conservative treatment usually require a permanent, adequate drainage procedure; *e.g.*, choledochoduodenostomy, choledochojejunostomy or sphincteroplasty.[3,7] This is best done at the first operation, but if the patient's condition is too poor for a major procedure, cholecystostomy or T-tube drainage can be performed, and elective, definitive, surgical drainage postponed until later.

CONCLUSIONS AND SUMMARY

Recurrent pyogenic cholangitis is a common disease in the Far East, and is occasionally seen in Asian immigrants in North America.

Five such patients are reported.

Knowledge of the characteristic cholangiographic appearances will enable the radiologist to make this diagnosis and alert the clinician to the recommended mode of management.

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REFERENCES

1. Krieger, J., Seaman, W. B., and Porter, M. R. Roentgenologic appearances of sclerosing cholangitis. *Radiology*, 1970, *95*, 369–375.
2. Mujahed, Z., Glenn, F., and Evans, J. A. Communicating cavernous ectasia of intrahepatic ducts (Caroli's disease). Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 21–26.
3. Ong, G. B. Study of recurrent pyogenic cholangitis. *A.M.A. Arch. Surg.*, 1962, *84*, 199–225.
4. Ong, G. B., Lee, T. C., Low, G., and Iu, P. Y. Air in biliary passages of choledochoduodenal origin. *Brit. J. Surg.*, 1962, *50*, 172–178.
5. Sabiston, D. C. Editor. Textbook of Surgery. W. B. Saunders Company, Philadelphia, 1972.
6. Stock, F. E., and Fung, J. H. Oriental cholangiohepatitis. *A.M.A. Arch. Surg.*, 1962, *84*, 409–412.
7. Stock, F. E., and Tinckler, L. F. Choledochoduodenostomy in treatment of cholangiohepatitis. *Surg., Gynec. & Obst.*, 1955, *101*, 599–606.
8. Wastie, M. L., and Cunningham, I. G. E. Roentgenologic findings in recurrent pyogenic cholangitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 71–77.

Vol. 122, No. 2

EXPERIENCE WITH ENDOSCOPIC RETROGRADE PANCREATICOGRAPHY*

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AFTER introduction of retrograde endoscopic pancreaticography by Oi *et al.*[16] and Tagaki *et al.*[21] in 1970, the examination was quickly accepted for the differential diagnosis of pancreatic diseases.

Since the beginning of 1971, we have performed 650 examinations and wish to present a report of our experience. The technique used was similar to that of Oi *et al.*,[16] Demling and Classen[9] and, in 1972, Weiss and co-workers.[24]

COMPLICATIONS

There are few reports of complications in the literature: Gaucher *et al.*,[12] Stadelmann *et al.*,[19] and Cotton *et al.*[8] describe perforations of the pancreatic duct by cannulation, with extravasation of the contrast medium but no further complications. An elevation of the amylase and the lipase, correlated to the degree of filling of the duct, is often seen; however, it is relatively harmless without other clinical signs. The most important complications are: a supervening acute pancreatitis, with severe clinical findings;[10,15] formation of a pseudocyst;[11] and, rarely, mortality.[19] In 650 examinations we had 1 mortality after the filling of a pseudocyst of the pancreas: death occurred 8 days after the procedure. An operation had not been performed. Cotton *et al.*[8] reported a similar incident.

These incidents demonstrate the necessity of immediate operation after pancreaticographic filling of a necrotic cavity, to prevent infection of the cavity by contrast medium.

NORMAL PANCREATIC DUCT SYSTEM AND NORMAL MOTILITY OF THE PAPILLA OF VATER

The anatomy of the pancreatic duct system is composed of magistral arborization. Fifteen to thirty equally calibrated side ducts, seen only partially during retrograde pancreaticography (Fig. 1), empty into the main duct, Wirsung's duct or the duct of Santorini. We use the term "rami pancreatici"[2] to describe these second degree ducts. Occasionally, one of these ducts has a larger caliber within the body of the pancreas, and several side ducts may empty into this duct. Such a "ramus corporis" then drains a somewhat bulging parenchymal area (Fig. 2).

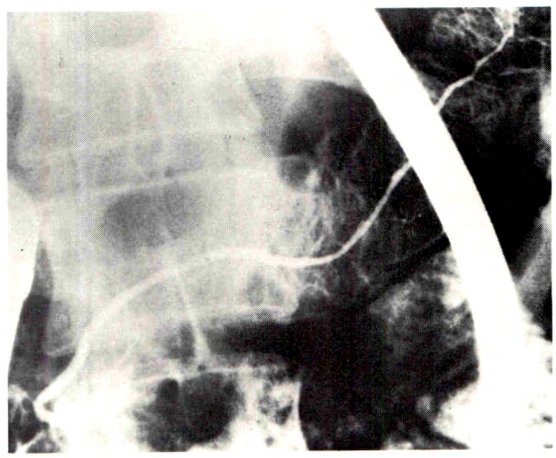


FIG. 1. Normal pancreatic duct system with representation of the Wirsung's duct and numerous rami pancreatici.

Santorini's duct is visualized only in a certain percentage of cases during pancreaticography, as in only 31 per cent does it communicate with Wirsung's duct.[13] It is located craniad to Wirsung's duct and empties in about 40 per cent of the cases through the papilla minor into the duodenum. It takes up a larger "ramus capitis," draining, with a branching duct system, the largest part of the pancreatic head. However, in 41 per cent, this "ramus

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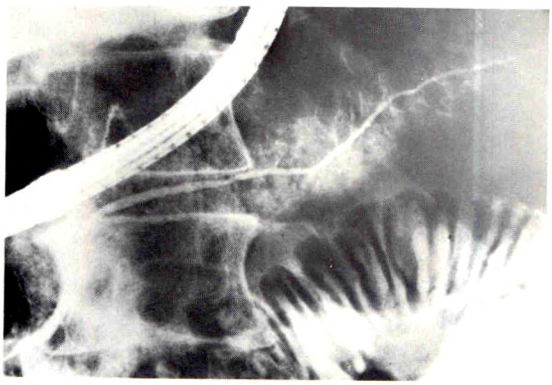


Fig. 2. Especially large ramus corporis between the pancreatic body and the tail, draining a bulging pancreatic area.

capitis" does not empty into the duct of Santorini but into Wirsung's duct. Only then can it be visualized during retrograde pancreaticography, since the filling pressure obtained in Santorini's duct is not sufficient to completely fill the duct. For the third degree branches we use the term "ductuli," and for the branches of these ductuli the term "canaliculi." The lumen of the normal Wirsung duct, measured on 100 retrograde pancreaticograms, was, on the average, 2–3 mm. with a range between 1 and 6 mm. For different parts of the pancreas, Classen *et al.*[6] gave figures of between 2 and 7 mm. for the head, between 2 and 5 mm. for the body, and between 1 and 2.5 mm. for the tail of the pancreas. Corresponding to the magistral arborization type, the lumen of the main duct is almost equal in the head and the body. Only in the tail of the pancreas does the lumen decrease. There is ectasia of the duct, if the lumen increases towards the tail. The lumen of the common bile duct is normally twice as large as that of the pancreatic main duct. This comparison is important for the diagnosis of different lesions of papillary stenosis, as on the one side the common duct and on the other side the pancreatic duct can be more affected, and thus more dilated in the prestenotic segment.

In retrograde pancreaticography it is important to know that among the different variations (papilla major, minor and duct systems), the Wirsung duct and the common bile duct empty together into the duodenum in only 85 per cent. This means that in at least 15 per cent, a simultaneous filling of the common bile duct and the Wirsung duct is impossible. However, since the rate of a simultaneous filling of both ducts by pancreaticography is usually lower, we should look for technical reasons related to anatomic structures of the papilla. In rare cases a pancreatic duct system is filled from the papilla major, draining only the head of the pancreas (Fig. 3) (this was noted in 4 of our 650 examinations). It is important to know this variation in order to avoid diagnostic errors.

The anatomic structure of the papilla of Vater, with a circular muscle at the distal end of both common bile and pancreatic ducts as well as around the opening of the papilla with longitudinal and spiral muscle fibers, explains the normal function of the sphincter of Oddi, which can be seen during the evacuation of contrast medium from both ducts.[3] During contraction of the sphincter muscle, the evacuation of the ducts is stopped, "contraction phase" (Fig. 4*A*). Usually the contraction is simultaneous in the common bile and the pancreatic ducts; seldom it is alternating. During relaxation of the circular muscles, there is either a common or a separate

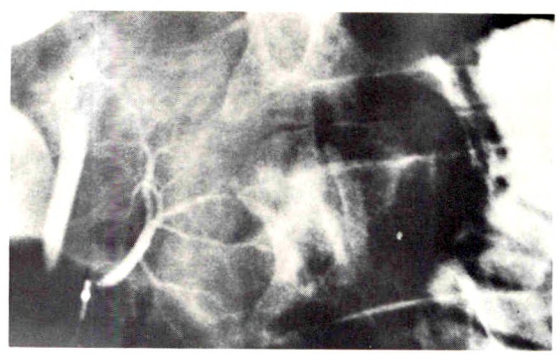


Fig. 3. Retrograde filling of a pancreatic duct system through the papilla major, draining exclusively the pancreatic head. No representation of the duct system in the body and the tail.

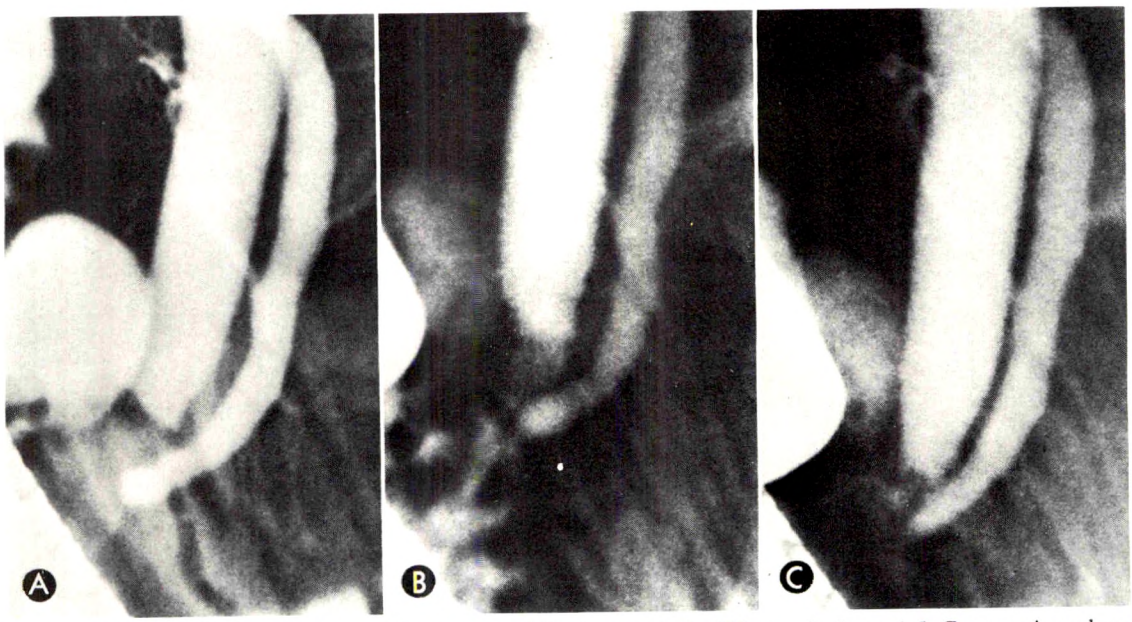


Fig. 4. Simultaneous evacuation of the common bile duct and the Wirsung's duct. (*A*) Contraction phase with back-flow of the contrast medium into the common bile and the pancreatic duct. (*B*) Evacuation phase with expulsion of contrast medium out of the common bile and pancreatic duct into the duodenum. (*C*) Resting phase with stasis of contrast medium in the common bile and the pancreatic duct.

evacuation of the different ducts, "evacuation phase" (Fig. 4*B*). The evacuation and the contraction phases last about 1–3 seconds. The phase in which there is neither evacuation nor contraction is the "resting phase" (Fig. 4*C*).

Evacuation of the whole pancreatic duct system, after removal of the catheter from the papilla of Vater, takes between 2 and 7 minutes on the average.

PAPILLARY STENOSIS AND PAPILLARY SPASM

Corresponding to the anatomic situation, the stenosis can involve either the common bile and Wirsung's duct, or the terminal portion of the common bile duct and the terminal portion of the pancreatic duct separately, or all 3 together and to a different degree. The stenosed duct is usually narrowed to fiber size, its outline is irregular or smooth, and its course is tortuous or stretched. The stenosis or the stenosed segment is not always visualized on roentgenography: in the prestenotic segment there is ectasia of the common bile duct or the pancreatic duct, or of both (Fig. 5).

Functionally, a delay of up to 15 minutes in contrast medium evacuation is seen in papillary stenosis.

In some types of papillary stenosis that are observed with the described symptoms of stenosis, *i.e.*, prestenotic ectasia and delay of evacuation, there is an evident hypermotility limited to the more frequent and delayed contractions, as well as an overlapping of duodenal peristalsis on sphincteric contraction. As a sign of strong contraction, the sphincter muscle bulges into the common bile duct, giving the appearance of a concave shape to the contrast medium in the common bile duct (Fig. 6*A*). Contrary to stenosis due to inflammatory reactions, the contours are smooth and stretched along the course of the common bile duct (Fig. 6*B*). Here evidently the normal functional moment of the stenosis in the sense of a spasm is very important.

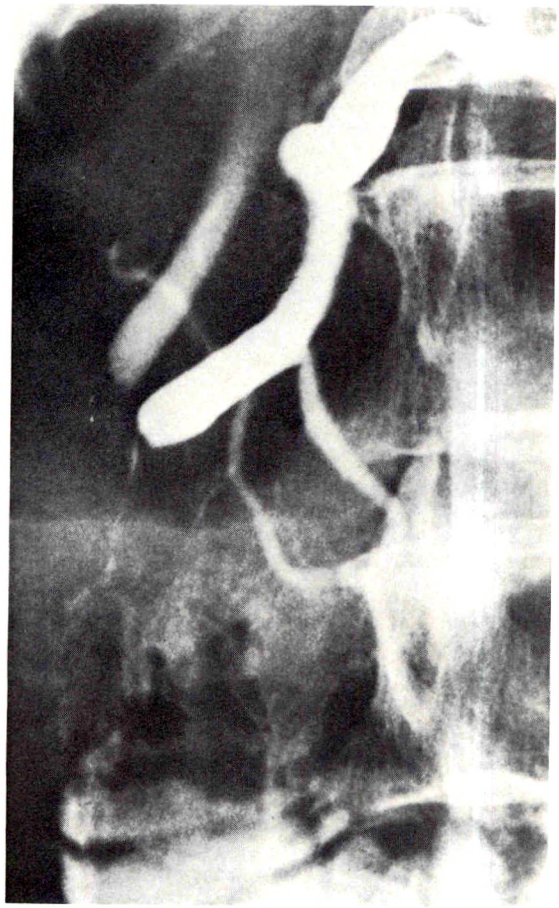


Fig. 5. Papillary stenosis with prestenotic ectasia, more pronounced in the pancreatic than in the common bile duct.

MALFORMATIONS

Malformations of the pancreas can only be visualized on retrograde pancreaticography if they communicate with the duct system. In 1 of 650 cases we found little round excavations the size of a pinhead at the ends of the finest ductuli and canaliculi, corresponding to the image of dysontogenetic cysts. The remainder of the duct system was entirely intact and the patient was without any complaints (Fig. 7).

CHANGES OF THE DUCT SYSTEM WITH AGE

According to our measurements in 100 normal cases, the lumen of the pancreatic main duct of a normal pancreas enlarges with age. The outlines become irregular and somewhat tortuous in their course. Similar changes are seen in the side ducts. The whole duct system demonstrates the same deformities as seen during the early stages of chronic pancreatitis.

CHRONIC PANCREATITIS

It is our experience that the changes of the duct system increase with the duration of chronic pancreatitis. In the first 2 years there are often, as in acute pancreatitis, no deformities at all. However, thereafter the effects of local fibrosis and cirrhosis of the pancreatic parenchyma, usually seen during the course of chronic pancreatitis, are reflected on the duct system. We find the same deformities as in the elderly patient with a normal pancreas: irregularities of the contours and the course of the main duct and the side ducts. In the further course of the disease, these deformities become pronounced, showing alterations of the lumen with single or multiple stenosis[26] (Fig. 8). The ductuli and canaliculi become coarse and cyst-like. The outstanding finding is ectasia, which is also seen in the main duct (Fig. 9). In 45 cases of chronic pancreatitis, ectasia was noted in 25. The more there are different symptoms and the more they are pronounced, the more the diagnosis of chronic pancreatitis can be documented by retrograde pancreaticography. Simultaneous narrowing of the common bile duct was found in 8 of 45 cases, when the common bile duct had an intrapancreatic course; Classen and Demling[5] found this narrowing in 16 of 64 cases.

In pancreatitis calculosa, the calcifications are either in the parenchyma of the pancreas or in the ducts. In retrograde pancreaticography, only the latter can be shown: extraductal stones have no effect on the pancreatic duct system. The intraductal stones are usually located in the Wirsung duct and are visualized as contrast medium filling defects. Only once did we find stones in a ramus pancreaticus, dilated to a small cavity (Fig. 10). At the same time the whole duct system shows more or

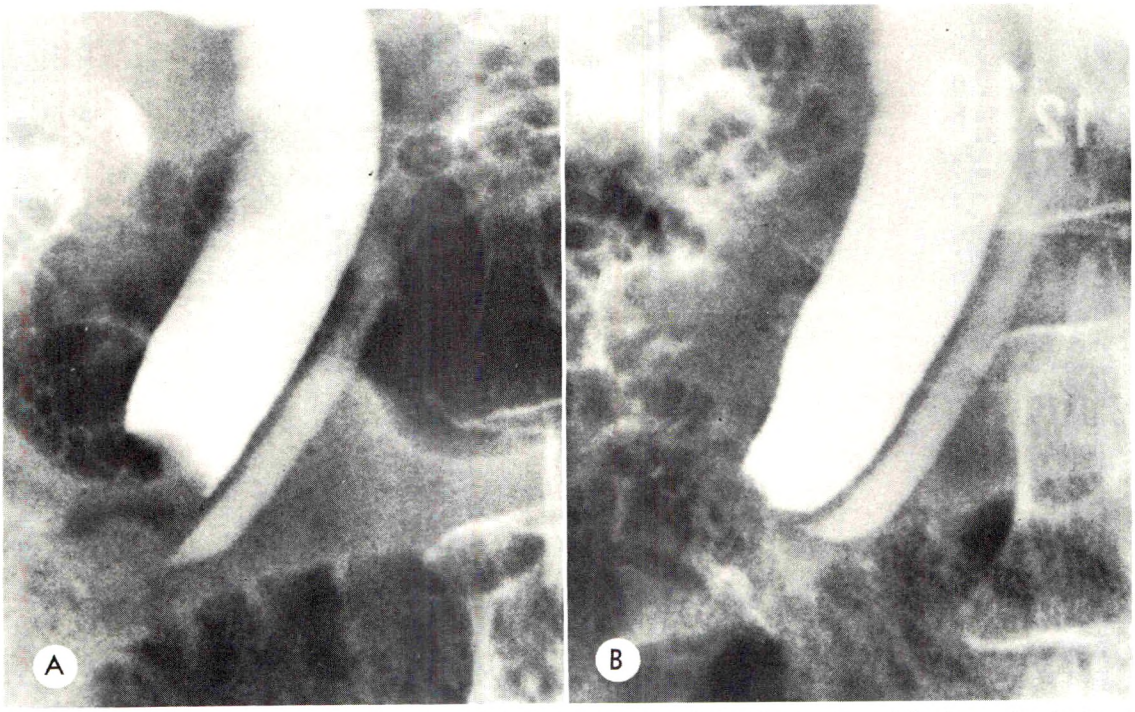


Fig. 6. (*A*) Papillary spasm with bulging of the sphincter muscle in the common bile duct, simulating a stone obstruction. (*B*) After release of the spasm smooth contours of the terminal common bile duct are seen.

less the changes of chronic pancreatitis. Rarely, the lithiasis of the Wirsung or the common bile duct is associated with a papillary stenosis. In this case the lithiasis can be the result or the cause of the papillary stenosis.

PANCREATIC ABSCESS AND PSEUDOCYST

Direct proof of the exact localization of an intrapancreatic abscess reflects an excellent progress in the diagnosis, first of all for the operative treatment. However, only those necrotic processes which are in com-

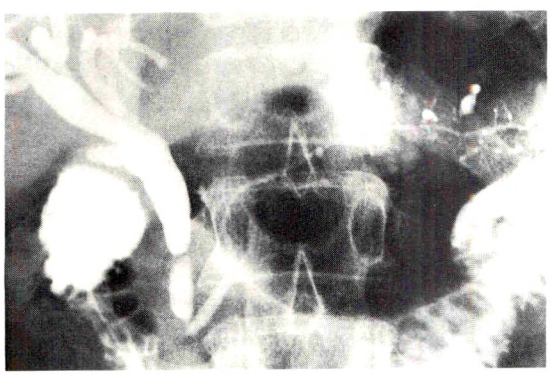


Fig. 7. Multiple small dysontogenetic cysts in the endings of the ductuli and canaliculi.

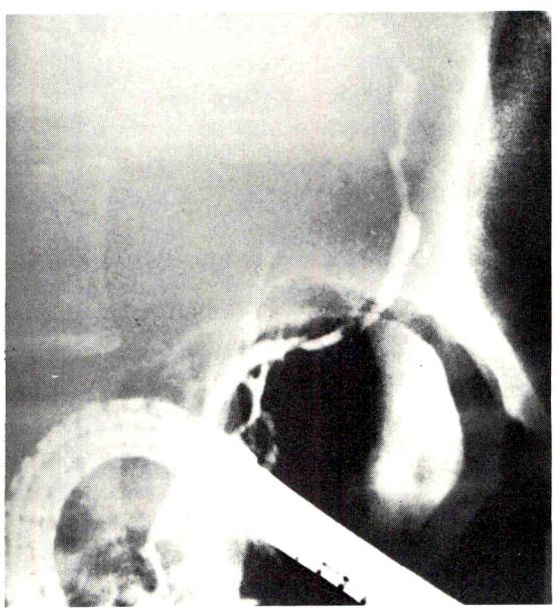


Fig. 8. Chronic pancreatitis with slight ectasia and multiple stenoses in the Wirsung's duct.

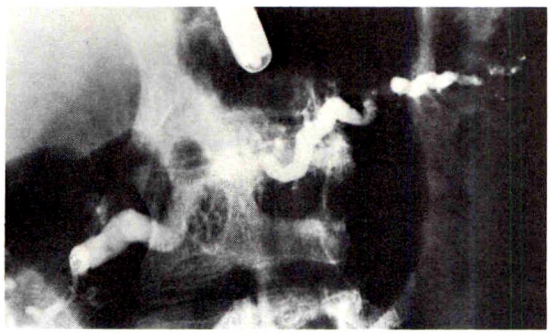


Fig. 9. Chronic pancreatitis with generalized ectasia and irregular course of the Wirsung's duct and with pronounced deformity of the rami pancreatici.

munication with the duct system can be shown by retrograde pancreaticography. In case of an abscess, there is a circumscribed contrast medium extravasation (one should better call it extraductation), usually of the size of 1–2 cm., sometimes with irregular contrast medium filling defects due to necrotic material or sequestra in the necrotic cavity (Fig. 14). The neighboring ductuli and canaliculi are deformed as in chronic pancreatitis. The rest of the duct system can be entirely normal. The diagnosis of abscess, as a space-occupying lesion, is confirmed by displacement of a larger duct (Fig. 11).

In pseudocyst there is, corresponding to the pathologic anatomic finding, a diffuse "extraductation" of contrast medium out of an autodigested or perforated duct. In our cases this was mostly the main duct or the 2nd degree duct (ramus). The extraductated contrast medium is quickly diluted in the large pseudocyst filled with fluid and, therefore, the exact size of the cavity can not be delineated (Fig. 12). One should not try this in any case, for the danger of infection is unnecessarily increased. Sometimes there is also a simultaneous perforation of 2 ducts (Fig. 12). The visualization of the perforated duct sometimes extends beyond the perforation itself (Fig. 13); occasionally, it ends at the site of the perforation (Fig. 14). As in the pancreatic abscess, the rest of the duct system can be entirely normal, or changed as in chronic pancreatitis.

In our 11 cases the necrotic process originated 5 times in the head, 3 times between the head and the body, twice in the body, and twice between the body and the tail of the pancreas.

If the pseudocyst loses its communication with the duct system, proof by pancreaticography becomes impossible.

Carcinoma of the Pancreas and of the Papilla

Carcinomas of the pancreas and the papilla can not be differentiated by endoscopy or by pancreaticography. They are dealt with here together. We examined a total of 14 carcinomas; in 2 of them a cannulation was not possible for a car-



Fig. 10. Multiple stones in the Wirsung's duct and in a ramus, which is dilated to a small cavity.

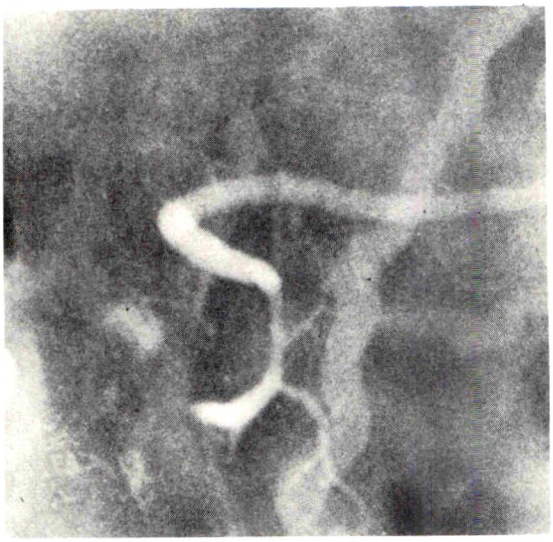


Fig. 11. An abscess in the head of the pancreas of the size of about 2 cm., demonstrated by an accumulation of contrast medium and displacement with narrowing of the neighboring Wirsung's duct.

cinoma infiltrating the papillary region and occluding the opening; the diagnosis could be made by biopsy and brushing. In detail we found the following signs:

Occlusion of the main duct (9; Fig. 15)
Stenosis with prestenotic ectasia (3; Fig. 16)
Displacement of the side ducts (2)
Lacunary contrast medium extravasation into the parenchyma (1; Fig. 17)
Displacement of the Wirsung duct and stenosis of the common bile duct in the prepapillary area (1).

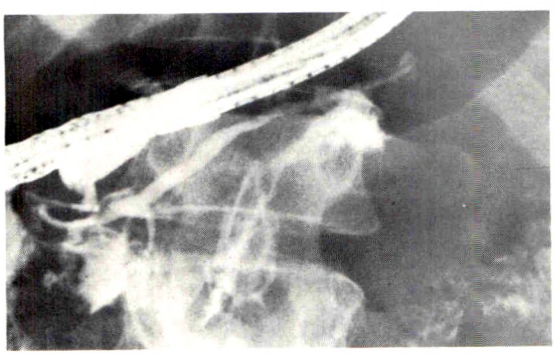


Fig. 12. Large pseudocyst, communicating with the perforated ramus capitis inferior and the Wirsung's duct in the area of the body of the pancreas.

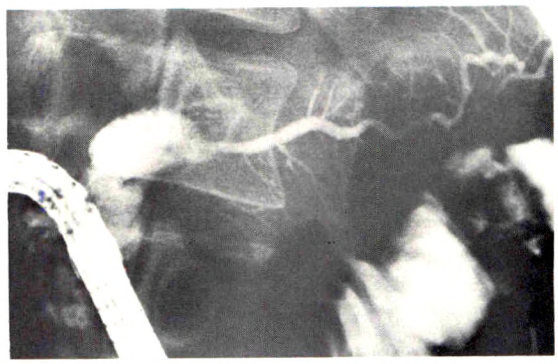


Fig. 13. Necrotic cavity in the head of the pancreas; the necrosis didn't destroy completely the pancreatic main duct.

At postmortem examination we found the following additional signs:[1]

Stenosis without prestenotic ectasia
Cystic deformation of the side ducts
Rarefaction of the side ducts
Bizarre lacunary ectasia.

Besides these changes in the pancreatic duct system the carcinoma can also pro-

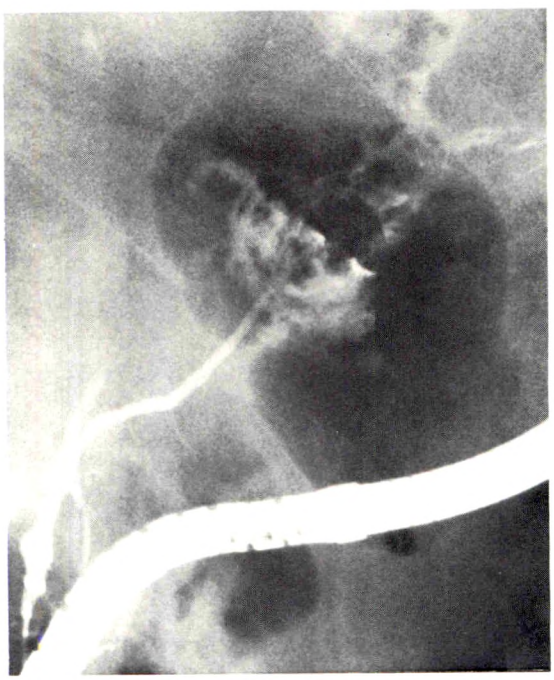


Fig. 14. Necrotic cavity in the pancreatic body, destroying completely the Wirsung's duct. The contrast medium filled defects in the cavity show necrotic material and sequestra.

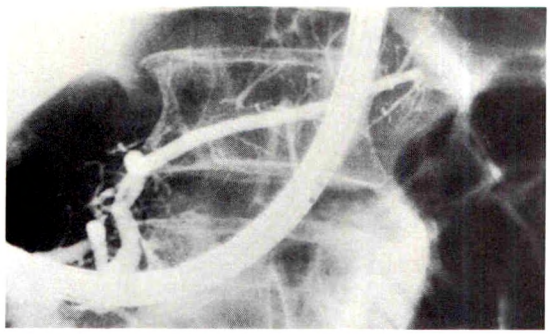


FIG. 15. Pancreatic carcinoma with complete obstruction of the Wirsung's duct between the body and the tail of the pancreas. There is a normal appearance of the pancreatic duct system up to the obstruction. Histologic examination: adenocarcinoma.

duce displacements of the common bile duct, when it is located in the head of the pancreas.

Since these signs alone are not specific of carcinoma, and since the occlusion of the pancreatic main duct often is seen in the advanced stage of stenosis, it seems unsuitable to differentiate the types of carcinomas by signs or symptoms. Because the location of the carcinoma influences the changes produced, it seems more appropriate to differentiate between the carcinoma originating in the pancreatic duct and the carcinoma originating in the parenchyma of the pancreas. The first usually results in total obstruction of the main duct, and a complete occlusion of the main duct is demonstrated by pancreaticography. The stenosis with prestenotic ectasia belongs to this type. The carcinoma originating in the parenchyma causes more often a stenosis without the following: prestenotic ectasia; displacement of the main and side ducts; and rarefaction of the side ducts.

The differentiation of carcinoma from chronic pancreatitis is very difficult by a single sign or symptom.[15,19,20] In carcinoma sometimes one can see no ectasia of the poststenotic duct segment, contrary to chronic pancreatitis where there are generalized ectasias (pre- and poststenotic) with solitary or multiple stenoses. In the carcinoma originating from the main duct, the duct system from the papilla to the obstruction is often completely normal.

A combination of displacement and stenosis of a duct is seen also in the pancreatic abscess, although an irregular contour of the stenosis favors a carcinoma.

The synopsis of the clinical findings and other subsequent examinations usually lead to the right diagnosis.[5–7]

SUMMARY

Review of our experience with the endoscopic retrograde pancreaticography is based on 650 examinations.

An anatomic nomenclature of the pan-

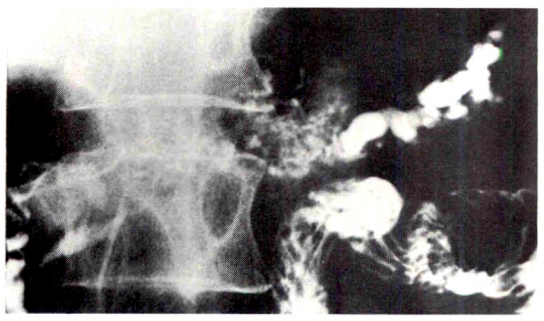


FIG. 16. Carcinoma between the head and the body of the pancreas with irregular stenosis and prestenotic ectasia of the Wirsung's duct. Histologic examination: adenocarcinoma.

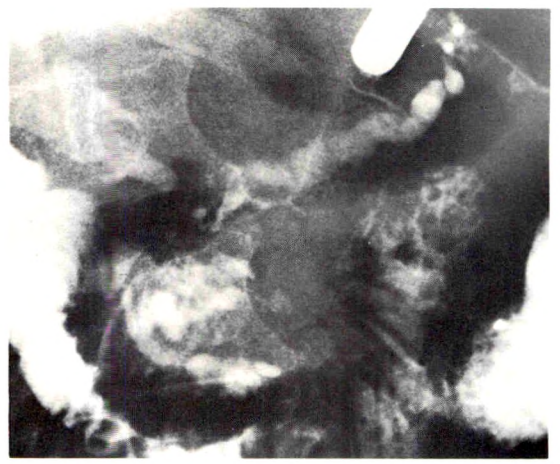


FIG. 17. Carcinoma of the head of the pancreas with stenosis of the Wirsung's duct and contrast medium extravasation into the surrounding pancreatic parenchyma. Histologic examination: adenocarcinoma with liver metastasis.

creatic duct system is proposed. In the course of the normal evacuation of the pancreatic duct 3 different phases are distinguished. In papillary stenosis and in papillary spasm these phases are changed pathologically and the total evacuation time is prolonged. In chronic pancreatitis there are irregularities of the outline of the course of the duct with progress of the disease; there are ectasias, solitary or multiple stenoses, and deformities of the side ducts.

Stones in the pancreatic main duct appear as defects in the contrast medium filling.

The special value of the diagnostic proof of pancreatic abscesses and pseudocysts shown by contrast medium extraductation is discussed as regards surgical intervention.

The duct changes seen in pancreatic carcinoma are often similar to those of chronic pancreatitis.

The normal appearance of the pancreatic main duct and side ducts between the tumor and papilla, and evaluation of all clinical and radiologic findings frequently allow the right differential diagnosis.

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REFERENCES

1. Anacker, H. Die pathologischen Veränderungen des Pankreasgangsystems im Röntgenbild. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1962, *96*, 455–470.
2. Anacker, H. Röntgenanatomie des Pankreas. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1961, *94*, 1–13.
3. Anacker, H., Weiss, H.-D., and Kramann, B. Die normale und die pathologische Entleerung des Ductus pancreaticus, insbesondere bei der Papillenstenose. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1973, *118*.
4. Anacker, H., Weiss, H.-D., and Wiesner, W. Das pankreatikographische Bild der entzündlichen Pankreasprozesse. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1972, *117*.
5. Classen, M., and Demling, L. Retrograde Cholangiographie beim Verschlussikterus. *Radiologe*, 1973, *13*, 35–40.
6. Classen, M., Hellwig, H., and Rösch, J. Anatomy of the pancreatic duct: duodenoscopic-radiological study. *Endoscopy*, 1973, *5*, 14–17.
7. Classen, M., Anacker, H., Stadelmann, O., Seifert, E., and Fritsch, E. Diagnosen von Tumoren der Papilla Vateri und der Bauchspeicheldrüse durch endoskopisch-radiologische Cholangio-Pankreatikographie. Symposium Radiologicum Pancreatis, München, 1973.
8. Cotton, P. B., Beales, J. S. M., and Cole, J. A. Hazards of endoscopic retrograde cholangio-pancreatography. In: Endoscopy of the Small Intestine with Retrograde Pancreato-Cholangiography. Edited by L. Demling and M. Classen. Georg Thieme, Stuttgart, 1973.
9. Demling, L., and Classen, M. Duodenojejunoskopie. *Deutsche med. Wchnschr.*, 1970, *95*, 1427–1428.
10. Fujita, R., Kumura, F., Hasegaway, Sohma, S., and Kidokoro, J. Endoscopic pancreatocholangiography. Second European Congress of Digestive Endoscopy, Paris, July, 1972.
11. Galvan, A., and Klotz, A. P. Is Transduodenal Pancreatography ever Contraindicated? American Society for Gastrointestinal Endoscopy, Dallas, May, 1972.
12. Gaucher, P., Jeanpierre, R., Watrin, B., Bigard, M. A., Bas, M., Vicari, F., Laurent, J., and Heully, F. Analyse critique des aspets radiologiques fournie par la Wirsungographie et la cholangiographie par duodénoscopie chez les malades atteints de pancréatite chronique. Second European Congress of Digestive Endoscopy, Paris, July, 1972.
13. Gualdi, G., and Messe, G. Rilievi anatomo-radiografici sul sistema escretore del pancreas con particolare riferimento ai canali accessori della testa. *Nuntius radiol.*, 1959, *25*, 391.
14. Hara, Y., and Ogoshi, K. Retrograde pancreatography via fiber-duodenoscopy. In: Endoscopy of the Small Intestine with Retrograde Pancreato-Cholangiography. Edited by L. Demling and M. Classen. Georg Thieme, Stuttgart, 1973.
15. Ogoshi, K., and Hara, Y. Retrograde pancreatocholedochography. *Japan J. Clin. Radiol.*, 1972, *17*, 455–466.
16. Oi, I., Kobayashi, S., and Kondo, T. Endoscopic pancreatocholangiography. *Endoscopy*, 1970, *2*, 103–106.
17. Oi, I. Duodenoscopy during pancreatic disease. *Arch. mal. app. digesto*, 1972, *61*, 349–354.
18. Reuter, S. R., Redman, H. C., and Joseph,

R. R. Angiographic findings in pancreatitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 56–64.

19. Stadelmann, O., Deyhle, P., Löffler, A., Hügel, H., Miederer, S. E., and Fumagalli, I. Endoscopic pancreatography in the diagnosis of tumors. In: Endoscopy of the Small Intestine with Retrograde Pancreato-Cholangiography. Edited by L. Demling and M. Classen. Georg Thieme, Stuttgart, 1973.
20. Stadelmann, P., Deyhle, P., Fumagalli, I., Miederer, S. E., Preter, B., Sobbe, A., Löffler, A., and Jenny, S. The Efficiency of Duodenoscopy in the Clinical Diagnosis Procedure. Second European Congress of Digestive Endoscopy, Paris, July, 1972.
21. Tagaki, K., Ikeda, S., Nakagawa, Y., Kumakura, K., Marnyama, M., Someya, N., Takada, T., Takekoshi, T., and Kin, T. Endoscopic cannulation of ampulla of Vater. *Endoscopy*, 1970, *1*, 107–114.
22. Tylen, U., and Arnesjö. Angiographic diagnosis of inflammatory disease of pancreas. *Acta radiol.* (Diag.), 1973, *14*, 215–240.
23. Weiss, H.-D., Anacker, H., Wiesner, W., and Rupp, N. Duodenoscopic pancreatography. *Radiology*, 1973, *106*, 333–339.
24. Weiss, H.-D., Anacker, H., and Wiesner, W. Die Technik der Duodenoskopie mit retrograder Pankreato-Cholangiographie. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1972, *116*, 517–522.
25. Weiss, H.-D., Anacker, H., Wiesner, W., and Kramann, B. Duodenoskopische Pankreatikographie-Pankreasangiographie: eine Vergleichsstudie bei chronischer Pankreatitis. In: Fortschritte der gastroenterologischen Endoskopie. Bd. 4. Herausg. von H. Lindner. Witzstrock, Baden-Baden, 1973.
26. Wiesner, W., Weiss, H.-D., and Anacker, H. Erste Erfahrungen mit der duodenoskopischen Pankreatikographie in der Diagnostik der chronischen Pankreatitis. *Deutsche med. Wchnschr.*, 1972, *97*, 991–994.

PANCREATIC PSEUDOCYST

A REVIEW OF 17 CASES WITH EMPHASIS ON RADIOLOGIC FINDINGS*

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MILWAUKEE, WISCONSIN

PANCREATITIS is a frequent result of chronic alcohol abuse, particularly in males over the age of 40 years. In acute pancreatitis, elevation of serum amylase in the presence of characteristic abdominal pain facilitates the diagnosis on the basis of clinical evidence, and the radiologic findings, although supportive, often are not diagnostic.

Pseudocyst of the pancreas occurs as a complication of subacute or chronic pancreatitis, at which time the serum amylase may have returned to normal levels, and the patient may be subjectively asymptomatic. In this phase of pancreatitis, the radiologic findings are of relatively greater significance.

In this paper, the clinical findings are briefly summarized in Table IA. The radiologic abnormalities were identified on plain film roentgenography of the abdomen, chest roentgenography, upper gastrointestinal study, cholecystography, barium enema examination, excretory urography and angiography. The radiologic findings are tabulated in Table IB. All cases were proven by surgery or autopsy. These patients were admitted to the Veterans Administration Hospital, Milwaukee, Wisconsin, or to the Milwaukee County General Hospital between 1967 and 1972.

Of the 17 cases reviewed, 8 cases are selected for more detailed presentation to illustrate the radiologic findings.

TABLE IA

SUMMARY OF CLINICAL FINDINGS IN 17 PATIENTS WITH PANCREATIC PSEUDOCYST

Case No.	Age (yr.)	Sex	Alcoholism	Abdominal Pain	Nausea Vomiting	Weight Loss	Gastrointestinal Bleeding	Serum Amylase (μ/ml.)
I	44	M	++	+	+		+ occult	48
II	82	M	?					50
III	42	M	++					35
IV	38	M	++	+			++ per varices	122
V	53	M	+	+		+		822
VI	43	M	+	+		+	+ per varices	220
VII	41	M	+	+				68
VIII	41	M	+	+		+	++ emesis	357
9	46	M	+	+				926
10	40	M	+	+	+	+		737
11	54	M	++					279
12	31	M	++	+	+			1,460
13	53	M	++					—
14	41	M	+	+	+			2,217
15	34	M	++					400
16	39	M	?	+			+ occult per ulcer	1,440
17	60	M	++	+				1,399

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TABLE IB

SUMMARY OF RADIOLOGIC FINDINGS IN 17 PATIENTS WITH PANCREATIC PSEUDOCYST

Case No.	Age (yr.)	Sex	Chest Roentgen-gram	Plain Abdominal Roentgenogram				IVP	GB	UGI	Colon	Angiogram
				Calcification	Mass	Abscess	Psoas Margin					
I	44	M	neg.		+	+	bilat. obscured			retroinfragastric mass	pushed down	
II	82	M	left effusion		+		normal	poorly visualized		retroinfragastric mass		displacement of left hepatic artery
III	42	M	neg.				normal	normal		retrogastric mass	neg.	avascular mass above spleen, splenic vein thrombosis
IV	38	M	neg.	+	+		normal	ureteral notching		retrogastric mass		stretching of left hepatic a., splenic vein thrombosis
V	53	M	neg.				normal	normal		anterior gastric mass		
VI	43	M	neg.		+		left obscured			gastric varices	displaced splenic flexure	
VII	41	M	neg.		+		normal	left poorly visualized		deformed antrum	neg.	
VIII	41	M	neg.				normal	normal	oral, fair visualization	widening of C loop		stretching of gastroduodenal a., and pancreaticoduodenal a.
9	46	M	left effusion				normal		IVC, bladder faint, duct neg.	irregularity along greater curve	neg.	
10	40	M	neg.				left obscured	normal	IVC, bladder faint, duct nonvisualized	mass along lesser curve	colon cut-off	
11	54	M	neg.	+			bilat. obscured			retrogastric mass	colon cut-off	
12	31	M	neg.				normal			mass on left side of stomach, coarse duodenum		
13	53	M	neg.		+		normal	normal		retrogastric mass	pushed down	
14	41	M	neg.		+		normal			retrogastric mass	pushed down	
15	34	M	left effusion	++	+		normal	normal		deformed fundus	pushed down	
16	39	M	neg.		+		normal			infragastric mass	pushed down	
17	60	M	neg.		+		left obscured	normal	IVC, bladder faint, duct angulated	retrogastric mass	pushed down	

IVP = intravenous pyelogram; GB = gallbladder; UGI = upper gastrointestinal; IVC = intravenous cholangiogram; a. = artery.

REPORT OF CASES

Case I. This 44 year old, white male entered the hospital because of suspected vocal cord tumor; however, biopsy proved to be negative. The patient gave a history of excessive use of alcohol. On physical examination, he was found to have soft tissue masses in the left upper quadrant and left lower quadrant of the abdomen, gynecomastia, spider angiomata and palmar erythema. Laboratory examination indicated elevated blood sugar with a curve suggesting diabetes mellitus. The serum amylase was within normal limits. The upper gastrointestinal study (Fig. 1*A*) revealed retrogastric and infragastric mass. Barium enema examination (Fig. 1*B*) demonstrated caudad displacement of the transverse colon. These findings were interpreted as pseudocyst of the pancreas. When surgery was performed, a large cystic mass was found occupying the lesser peritoneal sac and extending into the retroperitoneal space, displacing the stomach anteriorly and the colon inferiorly. The mass extended from the left diaphragm inferiorly into the pelvis. Six thousand cubic centimeters of purulent material was aspirated from the cyst, and cystgastrostomy was performed. One month later the patient developed gastric outlet obstruction, and pylorojejunostomy was performed with relief of the symptoms. Four months later he developed diffuse mid-abdominal pain. Physical examination revealed a firm, tender mass in the right groin. A roentgenogram of the abdomen showed an abscess of the right lower quadrant. When this abscess was drained, it was found to extend into the right retroperitoneal space. Postoperatively, the patient is doing well.

Comment. Pancreatic abscess is a relatively rare complication of pancreatic pseudocyst. It probably occurred in this patient because the original pancreatic pseudocyst was found to be infected, and, following surgery, there probably was residual pus or infected pseudocyst fluid within the retroperitoneal space. Normal serum amylase values, and diabetes, are often seen during the end stage of chronic relapsing pancreatitis. Cystgastrostomy is one of the well accepted surgical procedures.

Case II. This 82 year old male entered the hospital in a coma following an automobile accident. Carotid arteriography failed to show any abnormality. Chest roentgenogram revealed left pleural effusion. Laboratory examination indicated a high serum calcium (17 mg. per cent) which was thought to be due to hyperparathyroidism. Subsequently a parathyroid adenoma was surgically removed. During the course of hospitalization, the patient developed an epigastric mass.

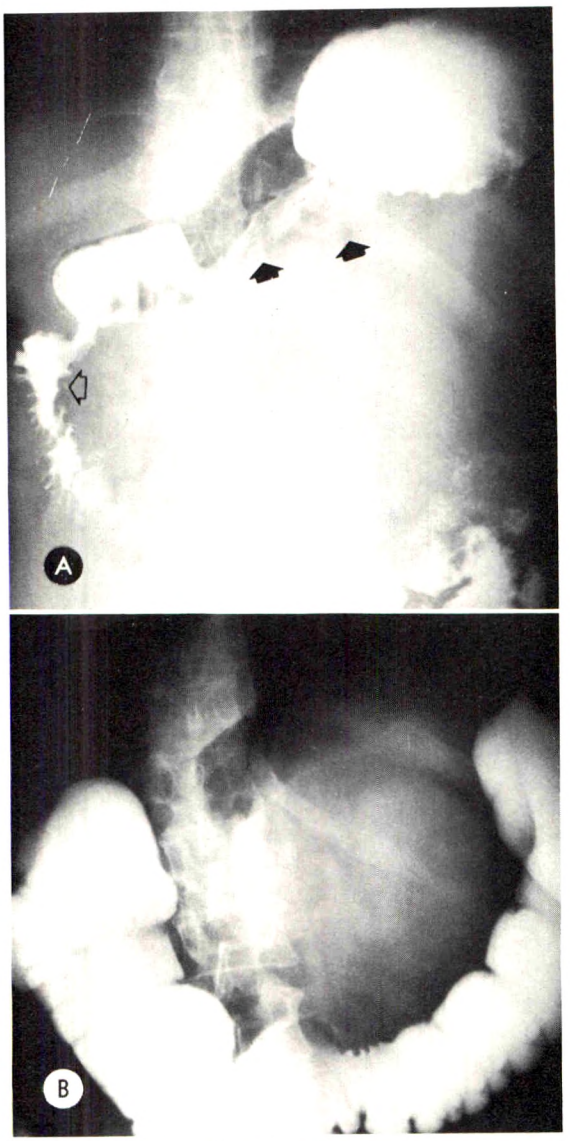


Fig. 1. Case I. (*A*) Frontal roentgenogram of the stomach reveals a large infragastric mass pushing the body of the stomach superiorly (▲). The loop of the duodenum is widened, and the duodenal mucosa is edematous (△). The pancreatic pseudocyst was located in the lesser peritoneal sac. (*B*) Barium enema roentgenogram shows caudad displacement of the transverse colon.

Upper gastrointestinal study revealed a large epigastric mass, pushing the stomach superiorly

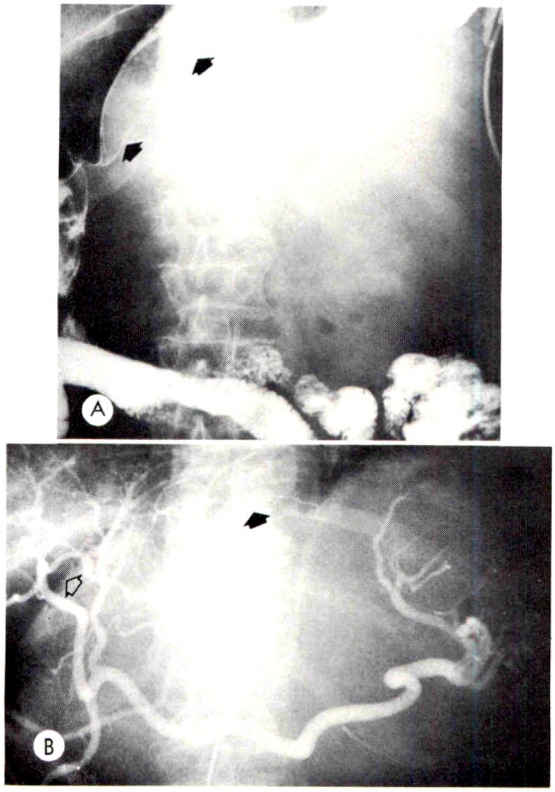


FIG. 2. Case II. (*A*) Frontal roentgenogram of the stomach shows a large mass causing compression and elevation of the stomach. The smooth outline of the stomach suggests extragastric mass (▲). (*B*) Celiac arteriogram shows stretching and displacement of the left gastric artery by an avascular mass (▲). Air in the stomach outlines displacement of the stomach by a smoothly rounded mass situated inferior to the stomach (△).

(Fig. 2*A*). Celiac arteriography was performed and revealed some stretching of the left gastric artery. No tumor vessels or staining were identified (Fig. 2*B*). At surgery a large cyst containing 2,000 cc. of thin, grayish fluid was found in the lesser peritoneal sac compressing the stomach superiorly and anteriorly, and displacing the transverse colon and the ligament of Treitz inferiorly. The pancreas was generally edematous. Ascitic fluid was present. Cystgastrostomy was performed. Three weeks after surgery the patient died. At autopsy, 3 more small, pancreatic pseudocysts were found within the head and tail of the pancreas. The largest pseudocyst was found in the body of the pancreas, and it extended into the lesser sac. The cause of the patient's coma was thought to be due to obstructive hydrocephalus secondary to leptomeningitis following traumatic subarachnoid hemorrhage.

Comment. This patient was exceptionally old considering that the other patients in this series were all between 30 and 50 years of age. It was the opinion of the pathologists that this patient's pancreatitis was due to a diffuse inflammatory process involving the pancreas, rather than trauma. Primary hyperparathyroidism may be associated with pancreatitis.[2] Angiography was not contributory in reaching the diagnosis and was done to look for a vascular tumor or hematoma.

CASE III. This 42 year old white male had a history of excessive intake of alcohol. He was admitted for evaluation of a left upper quadrant abdominal mass, which had been noted 5 months prior to admission when he had been admitted to another hospital for a hemorrhoidectomy. In other respects, he was asymptomatic at the time of admission. Physical examination revealed a fullness in the upper left quadrant of the abdomen, but no tenderness was present.

Laboratory examination revealed SGOT 130 units/ml., alkaline phosphatase of 200 units (King Armstrong), total bilirubin 1.0 mg. per cent, serum amylase 35 units/ml. Chest roentgenography and barium enema examination were normal. Upper gastrointestinal study revealed a slight indentation of the posterior aspect of the gastric fundus due to extrinsic pressure. Celiac angiography demonstrated a suprasplenic mass with compression of the superior aspect of the spleen. The venous phase showed a nonfunctioning splenic vein with extensive collateral venous flow (Fig. 3). At surgery a pancreatic pseudocyst containing 300 cc. of nonpurulent fluid was discovered posterior and superior to the spleen and communicating with the tail of the pancreas. The remainder of the pancreas was firm to palpation and apparently without active pancreatitis. The splenic vein was noted to be occluded.

Comment. Pancreatic pseudocyst situated between the spleen and the left diaphragm is an unusual location. Because of the roentgenographic appearance of an avascular mass adjacent to the spleen, a preoperative diagnosis of hematoma within the splenic capsule was favored, although pseudocyst of the pancreas was considered as a possibility because of the history of alcoholism and nonfunctioning splenic vein which was proved at surgery to be due to splenic vein thrombosis.

Case iv. This 38 year old Negro male had a long standing history of excessive use of alcohol. In December 1971, he was admitted to the hospital because of seizures and abdominal pain. In April 1972, he was again admitted because of massive upper gastrointestinal bleeding. At laparotomy the bleeding was found to be coming from varices of the stomach fundus. The bleeding was controlled by oversewing the stomach fundus. At surgery a large pancreatic pseudocyst was found extending from the left lobe of the liver. Fluid aspirated from the cyst was negative for amylase and bacteria. Postoperative upper gastrointestinal examination revealed a large mass posterior to the stomach (Fig. 4*A*). An excretory urogram revealed irregularity and localized displacements of the left ureter (Fig. 4*B*). Subsequently these irregularities of the ureter were proved by angiography to be due to periureteral dilated collateral veins secondary to splenic vein thrombosis. Splenic arteriography revealed occlusion of the splenic vein (Fig. 4*C*) with collateral venous flow extending inferiorly in the mesenteric and retroperitoneal veins. Celiac arteriography revealed stretching and displacement of the left hepatic artery. The liver scan was done using Tc^{99m} sulphur colloid, and, although there was slight uptake by the spleen, there was no uptake in the left lobe of the liver. During a second surgical exploration, a large necrotic cystic mass, approximately the size of a bowling ball, was found adherent between the left lobe of the liver and the spleen. Splenic vein thrombosis was noted, and splenectomy was performed. It was felt that the left lobe of the liver was involved by the cystic mass. Drains were placed in the left lobe of the liver and in the left upper quadrant of the abdomen. One month later the patient left the hospital in good condition.

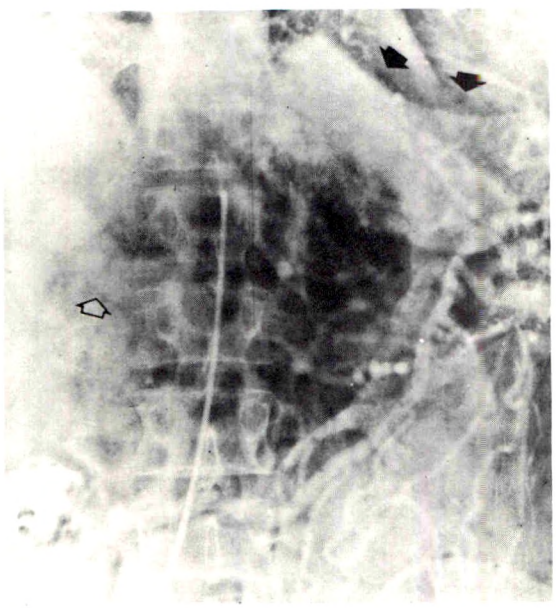


Fig. 3. Case iii. Celiac arteriogram, venous phase, demonstrates the absence of filling of the splenic vein. The presence of extensive collateral circulation and visualization of the portal vein (⌂) is diagnostic of splenic vein thrombosis. Note the smoothly outlined defect along the superior aspect of spleen representing mass effect by pseudocyst of pancreas (◆).

Comment. This patient initially presented with massive upper gastrointestinal bleeding from gastric varices apparently secondary to splenic vein thrombosis. Splenic vein thrombosis is a common complication of chronic pancreatitis and can be seen in at least 5 per cent[14] of patients with pancreatitis. Ureteral notching due to periureteral dilated collateral veins is a rare finding.[5] This finding is generally considered to be due to renal vein thrombosis,[1] but in this case, was caused by splenic vein thrombosis.

Case v. This 54 year old white male had had a long history of excessive use of alcohol. Three months prior to admission he noted a moderate epigastric mass. Upper gastrointestinal study revealed the mass to be anterior to the stomach (Fig. 5). At surgery, a cystic mass was found adjacent to the abdominal wall anterior to the stomach. The cyst had a thick wall which was opened inferiorly and posteriorly. Three hundred to four hundred cubic centimeters of turbid fluid was aspirated. The cyst opening was anastomosed to the jejunum for internal drainage in the form of a Roux-Y cyst jejunostomy.

Comment. An unusual finding in this patient was the location of the pancreatic pseudocyst anterior to the stomach. Pancreatic pseudocyst is more often located posterior to the stomach, inferior to the stomach, lateral to the stomach, or between the liver and the spleen.[28] Roux-Y cyst jejunostomy is one of the frequent methods of internal drainage.

Case vi. This 44 year old white male alcoholic was admitted following 3 days of left up-

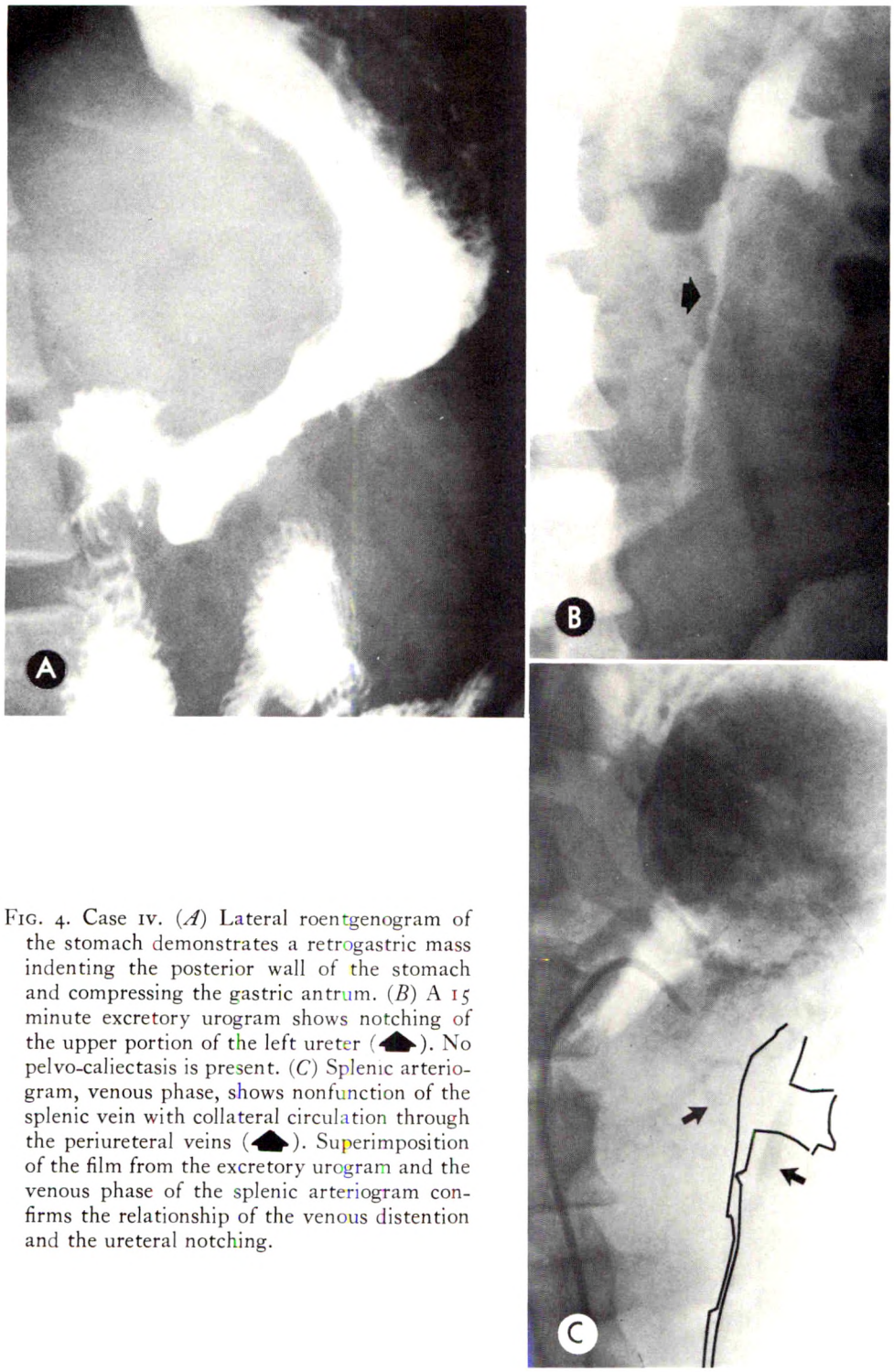


FIG. 4. Case IV. (*A*) Lateral roentgenogram of the stomach demonstrates a retrogastric mass indenting the posterior wall of the stomach and compressing the gastric antrum. (*B*) A 15 minute excretory urogram shows notching of the upper portion of the left ureter (▲). No pelvo-caliectasis is present. (*C*) Splenic arteriogram, venous phase, shows nonfunction of the splenic vein with collateral circulation through the periureteral veins (▲). Superimposition of the film from the excretory urogram and the venous phase of the splenic arteriogram confirms the relationship of the venous distention and the ureteral notching.



Fig. 5. Case v. Lateral roentgenogram of the stomach demonstrates a large extragastric mass, which is anterior to the stomach. This is quite an unusual location for pseudocyst of the pancreas.

per quadrant abdominal pain. Four months prior to admission he had been surgically treated at another hospital for pancreatic pseudocyst. During the interval, he had developed several episodes of mild upper gastrointestinal bleeding. He did not appear to be in acute distress; however, palpation of the abdomen revealed an exquisitely tender mass in the left upper quadrant below the left costal margin, measuring approximately 8 × 10 cm. in size. The hematocrit was 17.3 per cent, hemoglobin 5.9 gm. per cent, white blood cell count 3,900 mm.^{3} and serum amylase 220 units/ml. Upper gastrointestinal study was done, revealing the mass to be situated anterior and to the left of the stomach. Thickened mucosal folds were present throughout the gastric fundus, consistent with gastric varices (Fig. 6*A*). One week after admission, the patient's abdominal mass suddenly disappeared, and his pain became more severe. Emergency surgery was done, revealing a ruptured pancreatic

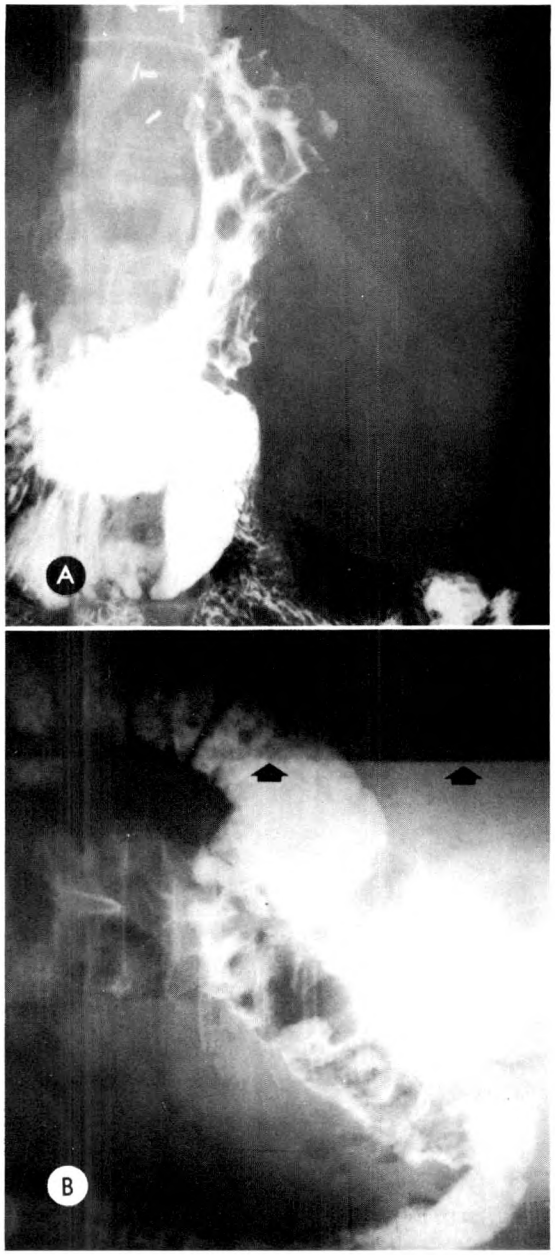


Fig. 6. Case vi. (*A*) Posteroanterior roentgenogram of the stomach shows irregular nodular filling defects in the gastric fundus consistent with gastric varices. The stomach is displaced toward the right by a large mass in the left upper quadrant of the abdomen. (*B*) Left lateral decubitus roentgenogram of the abdomen after cystgastrostomy, at which time there was rupture of the pancreatic pseudocyst. Note the air-fluid level within the peritoneal cavity (◆).

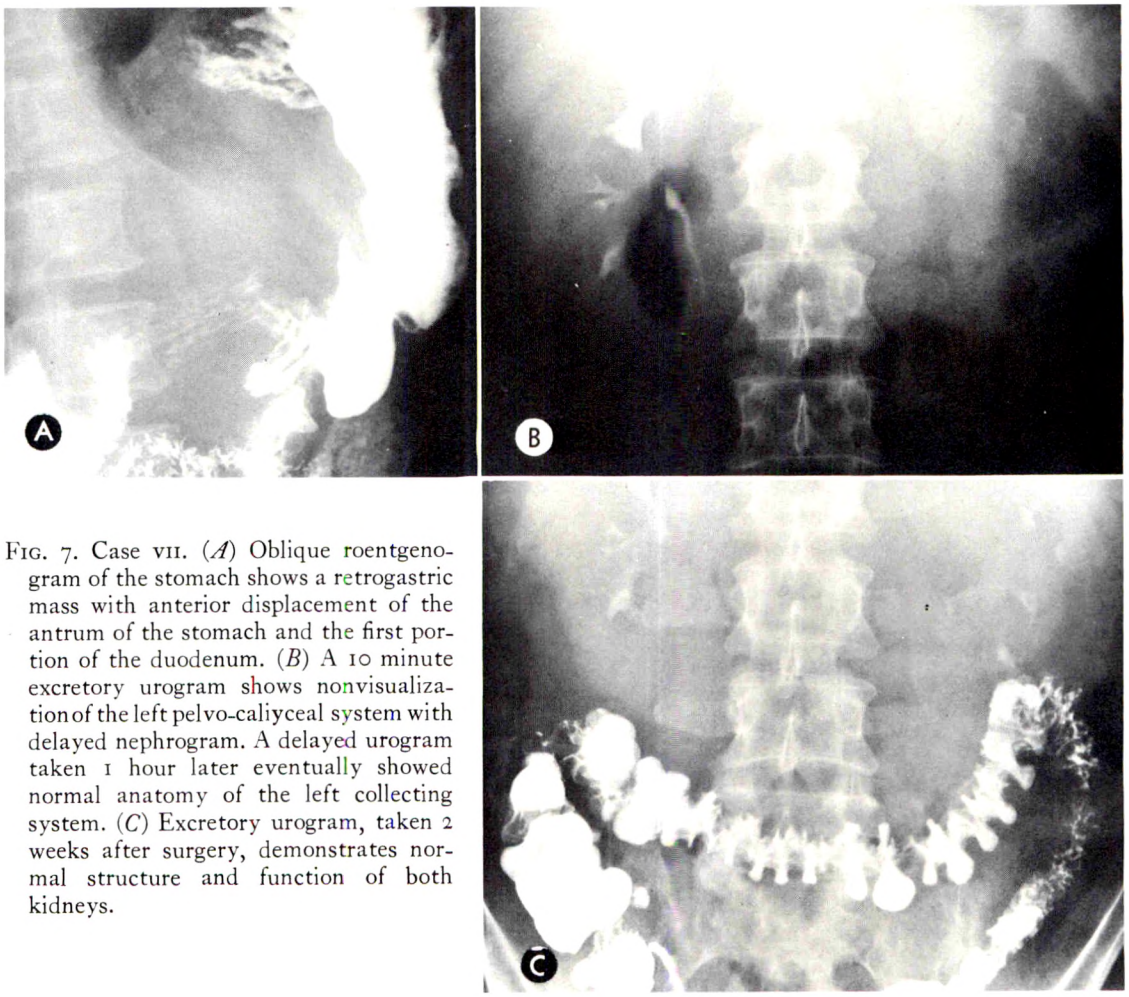


Fig. 7. Case vii. (*A*) Oblique roentgenogram of the stomach shows a retrogastric mass with anterior displacement of the antrum of the stomach and the first portion of the duodenum. (*B*) A 10 minute excretory urogram shows nonvisualization of the left pelvo-caliyceal system with delayed nephrogram. A delayed urogram taken 1 hour later eventually showed normal anatomy of the left collecting system. (*C*) Excretory urogram, taken 2 weeks after surgery, demonstrates normal structure and function of both kidneys.

pseudocyst. The pseudocyst was situated between the pancreas, stomach, anterior abdominal wall, and spleen. Cystgastrostomy was performed. During surgery he was noted to have gastric varices, splenomegaly, and splenic vein thrombosis.

Postoperatively, a left lateral decubitus roentgenogram of the abdomen was taken and showed air-fluid level within the abdomen (Fig. 6*B*). Subsequently, the patient developed respiratory complications and died 2 weeks after rupture of the pseudocyst.

Comment. This is the second patient in this series to have bleeding from gastric varices secondary to splenic vein thrombosis caused by pancreatitis. Spontaneous rupture of pancreatic pseudocyst is an abdominal catastrophe with an ominous prognosis. If the pseudocyst ruptures internally into the gastrointestinal tract, prognosis is somewhat better.[8,13]

Case vii. This 41 year old white male alcoholic entered with a complaint of epigastric pain. Upper gastrointestinal study revealed a retrogastric mass (Fig. 7*A*). Intravenous pyelography was done revealing a delayed excretion of contrast material from the left kidney (Fig. 7*B*). At surgery a cystic mass containing 800 cc. of dark colored fluid was found posterior to the stomach. The kidney was felt to be normal in consistency and size by palpation. Intravenous pyelography performed 2 weeks after surgery showed equal function of both kidneys (Fig. 7*C*). It was felt that the poor excretion from the left kidney was due to transient, vascular impairment, either by vessel spasm or compression by the pancreatic pseudocyst.

Case viii. This 41 year old white male alcoholic entered the hospital complaining of pain in the right upper quadrant. A right upper

quadrant mass was palpable. Gastrointestinal study revealed marked widening of the second portion of the duodenum (Fig. 8*A*). Abdominal angiography indicated stretching of the vessels in the region of the head of the pancreas (Fig. 8*B*). At surgery, a pancreatic pseudocyst containing 500 cc. of fluid was found within the head of the pancreas, displacing the duodenum to the right and anteriorly. A Roux-Y cyst-jejunostomy was performed, and the patient is doing well postoperatively.

Cases 9 to 17 are summarized on Tables IA and IB.

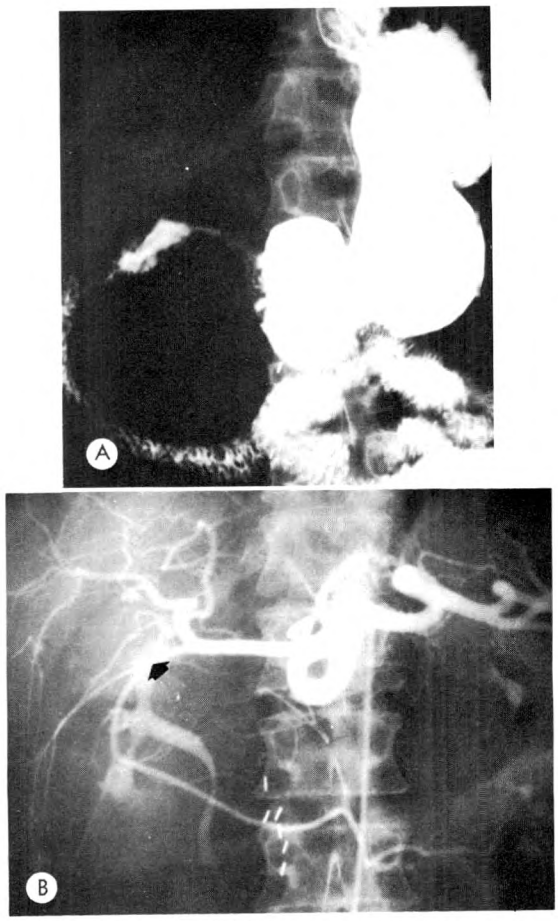


FIG. 8. Case VIII. (*A*) Posteroanterior roentgenogram of the stomach shows a markedly widened C-loop of the duodenum consistent with a mass in the head of the pancreas. The mucosal pattern of the duodenum appears quite normal. (*B*) Celiac arteriogram shows stretching and displacement of the gastroduodenal artery (◀) and pancreaticoduodenal arteries, indicating enlargement of the head of the pancreas. No abnormal vessels are identified within the mass.

DISCUSSION

Twenty-three radiologic manifestations of pancreatitis have been reported.[29] None of these manifestations are sufficiently definitive to provide a precise diagnosis. Pseudocyst of the pancreas, on the other hand, is not difficult to diagnose. The combination of pancreatic calcification and a mass posterior to the stomach is strongly suggestive of the diagnosis of pancreatic pseudocyst.

AGE AND SEX

Most of the patients in this series were between 40 and 50 years of age. The youngest was 31 years and the oldest was 82 years of age. They were all male. Two-thirds of our cases are from the Veterans Administration Hospital, where the patient population is predominantly male; however, pancreatitis is more common in males. Ninety-three out of 100 cases of pancreatitis reported by Netto *et al.*[19] were males.

ETIOLOGY

Fifteen of 17 patients reported in this series gave a history of excessive intake of alcoholic beverages. Of the remaining 2, 1 who was 82 years old and in a coma (Case 11) was unable to provide an adequate history. In Case 16, history about alcoholic intake was not described. In the series of Sarles *et al.*,[26] 99 out of 100 cases of chronic pancreatitis patients were alcoholics.

Gallstones were not found in any of our patients, either at surgery or at autopsy. According to Netto *et al.*,[19] who reported 100 cases of chronic pancreatitis, 22 per cent were alcoholics. 42 per cent had gallbladder disease, and 8 per cent had both gallbladder disease and a history of alcoholism.

There appears to be more than a coincidental relationship between primary hyperparathyroidism and pancreatitis. In one series of 155 cases of primary hyperparathyroidism, 11 patients were found to have pancreatitis, and of these, 81 per cent were noted to have pancreatic calcifications.[2] The patient in our series (Case 11), who had

a combination of pancreatitis and hyperparathyroidism, did not have pancreatic calcifications.

CLINICAL FINDINGS

Five of the patients had no abdominal symptoms, the others complained of abdominal pain (12 cases), upper gastrointestinal bleeding (5 cases), nausea and vomiting (4 cases), and weight loss in excess of 20 pounds (4 cases). Serum amylase value was normal in 5. These findings are tabulated in Table 1A.

RADIOGRAPHIC FINDINGS

Chest roentgenography. In this series, 3 patients (18 per cent) had evidence of left pleural effusion. Margulis and Burhenne,[14] report an incidence of 6 per cent pleural effusion in patients with pancreatitis. Additional manifestations include limitation of movement of the left hemidiaphragm and elevation of the left hemidiaphragm. In severe cases, both hemidiaphragms may be involved.[10,20,23,29]

Plain film roentgenography of the abdomen. Pancreatic calcifications were identified on the roentgenograms of 3 of the patients in this series (18 per cent), ages 38, 54, and 34. Reported incidence of pancreatic calcifications in patients with chronic pancreatitis is as follows: 17–24 per cent by Bockus;[2] 24 per cent by Netto *et al.*,[19] and 49 per cent by Sarles *et al.*[26]

In 10 of the patients in this series (59 per cent), a soft tissue mass could be visualized on plain roentgenograms of the abdomen. Usually the mass was apparent because of displacement of the stomach or colon. In 3 of our cases, an abscess was formed by infection of the pancreatic pseudocyst. In 1 of these cases, the soap-bubble appearance was apparent on plain roentgenogram of the abdomen. Pancreatic abscess is a serious condition with fever and a high mortality rate.[6,23] Case 1 had an infected pancreatic pseudocyst and, later, developed peritoneal and retroperitoneal abscesses. The other 2 patients died following rupture of the pancreatic pseudocyst. According to Littmann *et al.*,[13] there is a 5 per cent incidence of rupture of pancreatic pseudocyst. The cyst may rupture into the stomach or duodenum, in which case it is not as severe as a rupture which results in spill of the cyst contents into the peritoneal cavity.[8,13]

Retroperitoneal effusion of cyst fluid may obscure the outline of the left kidney and psoas muscle. This finding has been reported in a study by Meyers *et al.*[16] and others.[20,23,29] Obliteration of the retroperitoneal fat planes may be even more obvious, if the patient is emaciated due to pancreatic digestive insufficiency. In our series, the renal and psoas muscle outlines were obliterated in 5 cases (29 per cent): 3 cases on the left side only, and 2 cases bilaterally. According to Stein *et al.*[29] 17 out of 61 cases of acute pancreatitis (28 per cent) had loss of renal and psoas muscle outlines. Excretory urography was performed in 10 of the patients in this series. All were normal except 1 (Case 10), who had poor concentration in the left kidney. Following surgery, kidney function returned to normal, and the patient is doing well years later. Pancreatic pseudocyst has been found to cause a pressure effect upon the renal vessels in other series.[1,15,20] Notching of the ureter was found in 1 of our patients (Case IV), and this was later shown to be caused by dilated collateral veins adjacent to the ureter secondary to splenic vein thrombosis. This manifestation was described by Chait *et al.*[5] as occurring in portal hypertension. Other conditions which produce ureteral notching may be renal artery stenosis[15] or renal vein thrombosis.[1]

Gallbladder examination was performed in 4 of our cases, 3 by intravenous administration of cholografin, and 1 by oral administration of telepaque. In all of these patients, there was faint visualization of the gallbladder, but no stones were identified. In Case 17, the typical kneebend deformity of the distal common bile duct was found to be due to displacement by a mass in the head of the pancreas. This finding has been described in pancreatitis.[14,23,25,31]

The position of the colon was studied either by barium enema, residual barium in the colon from previous intestinal

studies or by intestinal gas. In 11 cases, there was found to be caudal displacement of either the transverse or the splenic flexure by a soft tissue mass. Three cases were noted to have an irritable splenic flexure with a typical colon cut-off sign.[4,27] The colon cut-off sign has been described as an abrupt termination of the column of gas within the colon lumen, due to an extension of the inflammatory process of the pancreas through the transverse mesocolon or phrenocolic ligament, causing irritability, as is described elsewhere.[4]

Upper gastrointestinal study. The location of the mass of pancreatic pseudocyst in relation to the stomach is summarized in the diagrams shown in Figure 9. The most frequent location of the cyst is retrogastric, and the least frequent location of the cyst is anterior to the stomach.[2,20] In 1 case (Case VIII), the pseudocyst was located in the head of the pancreas, causing widening of the duodenal loop, and 2 other patients had widening of the duodenal loop secondary to a large extrapancreatic pseudocyst (Case I and Case II).

Angiography. The value of angiography in pancreatitis is well established.[1,3,9,11,12,17,22,24] Angiography was performed in 4 patients in our series, and 3 of them showed stretching or displacement of some of the branches of the celiac axis, left hepatic artery, gastroduodenal artery, or superior pancreaticoduodenal arteries, as has been described by others.[9,11,21,22,24] Pooling of the contrast medium in microaneurysms, areas of hyperemia or decreased vascularity, vessel encasement and deformity has been described elsewhere.[9,12,24] Because the splenic vein is situated in close anatomic relationship to the pancreas, it is frequenly involved by pancreatitis, with the occurrence of either thrombosis, displacement, or deformity of the splenic vein.[1,11,14,22,24,26,30] In our series, 3 patients were noted to have splenic vein thrombosis. Study of the venous phase of celiac angiography will provide valuable diagnostic information.[1,24]

Volume of the pancreatic pseudocysts in this series varied from 200 ml. to 6,000 ml. The pseudocysts were located outside the

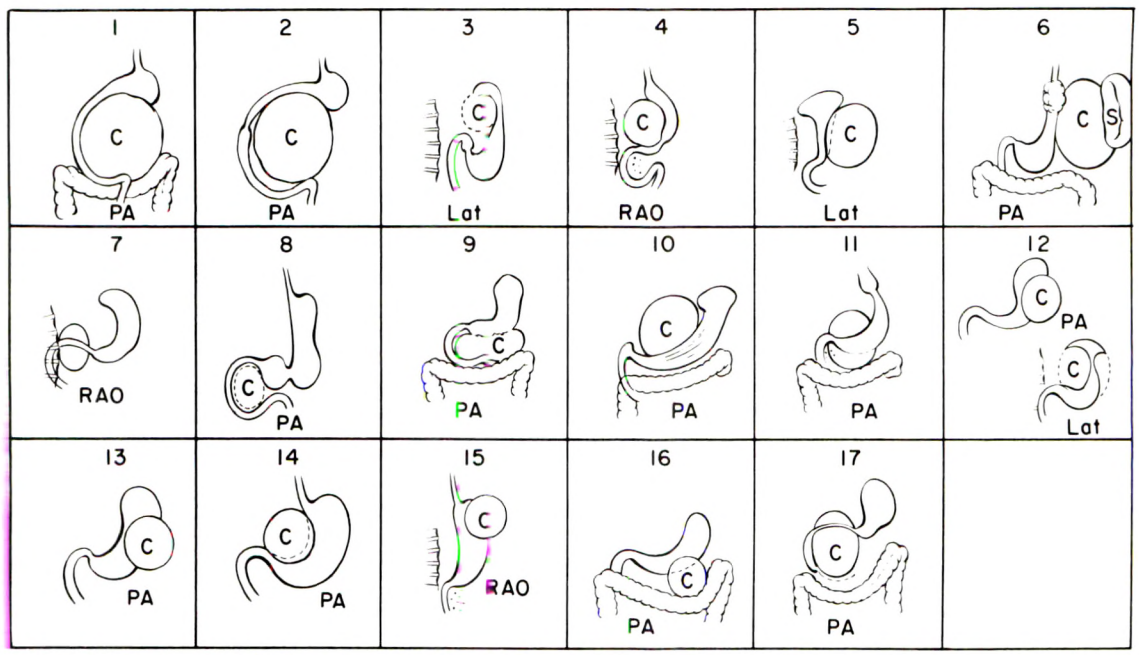


Fig. 9. Diagrams showing the location of the mass of pancreatic pseudocyst in relation to the stomach in the 17 cases studied. PA = posteroanterior; Lat = lateral; RAO = right anterior oblique.

pancreas with the exception of 1 case (Case 10), in which the cyst was located within the head of the pancreas. In a series of 26 surgically proven cases of pancreatic pseudocysts reported by Sarles *et al.*[26] the pseudocyst was found to be extrapancreatic in 11, and intrapancreatic in 16.

The anatomic location of the pseudocyst was: within the lesser peritoneal sac, 11 cases; in the peritoneal cavity, 3 cases; retroperitoneal, 1 case; and within the head of the pancreas, 1 case. The anterior surface of the pancreas forms a portion of the lesser peritoneal sac, which explains the greater frequency of pancreatic pseudocyst in this location.[18] The operative procedures performed were as follows: cystgastrostomy, 10 cases; external drainage, 2 cases; resection of the pseudocyst, 2 cases. The preferred method of treatment is total excision,[30] but in most cases this is impossible and, therefore, internal and external drainage is performed.[7,30]

SUMMARY

Seventeen cases of pseudocyst of the pancreas were reviewed and a summary of the clinical manifestations and radiologic findings is given.

Preoperative diagnosis of pancreatic pseudocyst can be accurately made with the aid of radiologic examination.

Plain film roentgenograms of the abdomen revealed evidence of the pseudocyst mass in most cases (59 per cent). Upper gastrointestinal study was of value in providing more precise information relative to the location of the pseudocyst. Barium enema examination revealed evidence of extrinsic pressure on the transverse colon or irritation of the splenic flexure.

Various other radiologic manifestations are described.

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REFERENCES

1. Abrams, H. L. Angiography. Second edition. Volume II. Little, Brown & Company, Boston, 1972, pp. 953–1088.
2. Bockus, H. L. Gastroenterology. Volume III. W. B. Saunders Company, Philadelphia, 1964, pp. 917–931.
3. Boijsen, E., and Tyler, U. Vascular changes in chronic pancreatitis. *Acta radiol.* (Diag.), 1972, *12*, 34–48.
4. Brascho, D. J., Reynolds, T. N., and Zonca, P. Radiographic colon cut-off sign in acute pancreatitis. *Radiology*, 1962, *79*, 763–768.
5. Chait, A., Matasar, K. W., Fabian, C. E., and Mellins, H. Z. Vascular impressions on ureters. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 729–749.
6. Farringer, J. L., Robbins, L. B., and Pickens, D. R. Abscess of pancreas. *Surgery*, 1966, *60*, 964–970.
7. Galligan, J. J. Pancreatic pseudocyst in childhood. *A.M.A. Am. J. Dis. Child.*, 1966, *112*, 479–482.
8. Guyer, P. B., and Amin, P. H. Radiological demonstration of spontaneous rupture of pancreatic pseudocyst. *Brit. J. Radiol.*, 1970, *43*, 342–343.
9. Kadell, B. M., and Riley, J. M. Major arterial involvement by pancreatic pseudocyst. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 632–636.
10. Kaye, M. D. Pleuropulmonary complications of pancreatitis. *Thorax*, 1968, *23*, 297–306.
11. Lechner, G., and Pokieser, H. Ergebnisse angiographischer Untersuchungen bei Pankreatitis. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1971, *114*, 49–57.
12. L'Herminé, C., Gautier-Benoit, C., Vankemmel, M., and Lemaitre, Les érosions artérielles des pseudo-kystes pancréatiques. *Ann. de radiol.*, 1971, *14*, 55–63.
13. Littmann, R., Pochaczevsky, R., and Richter, R. M. Spontaneous rupture of pancreatic pseudocyst into duodenum. *A.M.A. Arch. Surg.*, 1970, *100*, 76–78.
14. Margulis, A. R., and Burhenne, H. J. Alimentary Tract Roentgenology. Volume II. C. V. Mosby Company, St. Louis, 1967, pp. 857–895.
15. Meschan, I. Roentgen Signs in Clinical Practice. Volume II. W. B. Saunders Company, Philadelphia, 1966, pp. 1183–1213.
16. Meyers, M. A., Whalen, J. P., Peelle, K., and Berne, A. S. Radiological features of extraperitoneal effusions. *Radiology*, 1972, *104*, 249–257.
17. Nebesar, R. A., and Pollard, J. J. Critical evaluation of selective celiac and superior mesenteric angiography in diagnosis of pan-

creatic disease. *Radiology*, 1967, *89*, 1017–1027.
18. Netter, F. H. The CIBA Collection of Medical Illustrations. Volume 3. Part 3. 1964, *24*, 143–146.
19. Netto, A. P., Dreiling, D. A., and Baronofsky, I. Relationship between pancreatic calcification and cancer of pancreas. *Ann. Surg.*, 1960, *151*, 530–537.
20. Poppel, H. Roentgen manifestation of pancreatitis. *Seminars in Roentgenol.*, 1968, *3*, 227–241.
21. Ranniger, K., and Saldine, R. M. Arteriographic diagnosis of pancreatic lesions. *Radiology*, 1966, *86*, 470–474.
22. Reuter, S. R., Redman, H. C., and Ramon, R. J. Angiographic findings in pancreatitis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 56–64.
23. Robbins, L. L. Editor. Golden's Diagnostic Roentgenology. Williams & Wilkins Company, Baltimore, 1968, pp. 14.125–14.140.
24. Rösch, J. Angiography in diagnosis of pancreatic disease. *Seminars in Roentgenol.*, 1968, *3*, 296–309.
25. Sachs, M. D. Routine cholangiography: operative and postoperative. *Radiol. Clin. North America*, 1966, *4*, 547–560.
26. Sarles, H., Sarles, J. C., Camatte, R., Muraitore, R., Gaini, M., Guien, C., Pastor, J., and LeRoy, F. Observations in 205 confirmed cases of acute pancreatitis, recurring pancreatitis and chronic pancreatitis. *Gut*, 1965, *6*, 545–559.
27. Schwartz, S., and Nadelhalft, J. Simulation of colonic obstruction at splenic flexure by pancreatitis: roentgen features. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *78*, 607–616.
28. Shockman, A. T., and Marasco, J. A. Pseudocysts of pancreas. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 628–638.
29. Stein, G. N., Kalser, M. H., Sarian, N. N., and Finkelstein, A. Evaluation of roentgen findings in acute pancreatitis. *Gastroenterology*, 1959, *36*, 354–361.
30. Warshaw, A. L., McChesney, T., Evans, G. W., and McCathy, H. F. Intrasplenic dissection by pancreatic pseudocyst. *New England J. Med.*, 1972, *287*, 72–74.
31. Wise, R. E. Cholangiography and duct pancreatography in diagnosis of pancreatic diseases. *Seminars in Roentgenol.*, 1968, *3*, 288–295.

SELECTIVE ARTERIOGRAPHY COMBINED WITH HYPOTONIC DUODENOGRAPHY FOR PANCREATIC LESIONS*

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IN a previous article,[14] we reported the basic study on the diagnostic usefulness of combined selective arteriography and hypotonic duodenography for carcinoma of the pancreas. Since then, we have routinely used this new combined technique in over 100 patients who were suspected of having a pancreatic lesion. By employing this procedure, much more valuable information could be obtained than by the simple summation of the findings noted on the 2 roentgenograms obtained separately.

The purpose of the present communication is to report a group of patients illustrating the significance of this examination.

The technique, the details of which were described elsewhere,[14] is briefly as follows: In the prone position, 2 per cent Xylocaine solution is infused through the tube introduced into the duodenum, for the purpose of anesthesia of the anterior surface of the duodenal mucosa. With the tube still in place, the patient is turned to the supine position, and selective angiography of the celiac trunk and superior mesenteric artery is performed in both anteroposterior and right oblique projections. After angiography, the catheter is kept in the orifice of the artery and hypotonic duodenography is routinely done using an anticholinergic drug and Xylocaine solution. During the phase of insufflation, when the duodenum is semi-opacified and visualized in double contrast, arteriography is performed with the catheter remaining in the celiac trunk. In most of the patients, the catheter is advanced further into the common hepatic artery and superselective angiography is added.

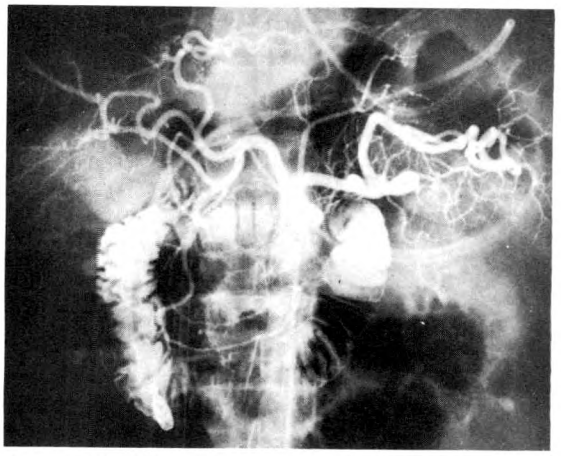


FIG. 1. Combined roentgenogram of a normal subject. In addition to the combination of a celiac angiogram and hypotonic duodenogram, an oral cholecystogram is also superimposed. The normal correlative anatomy of vascular, duodenal and biliary systems is visible.

Figure 1 shows the combined roentgenogram of a normal subject. The topographic relationship between the duodenal loop and the arteries is clearly visible. In addition to the combination of these 2 images, the gallbladder visualized by oral cholecystography is also superimposed in order to show the normal correlative anatomy of the vascular, duodenal and biliary systems.

All the patients illustrated below underwent laparotomy about 1 week after the examination.

REPORT OF CASES

CASE 1. A 41 year old female had a $2\frac{1}{2}$ months' history of progressive jaundice. Selective arteriography showed irregular stenosis of both anterior superior and posterior superior pancreaticoduodenal arteries, but neither pro-

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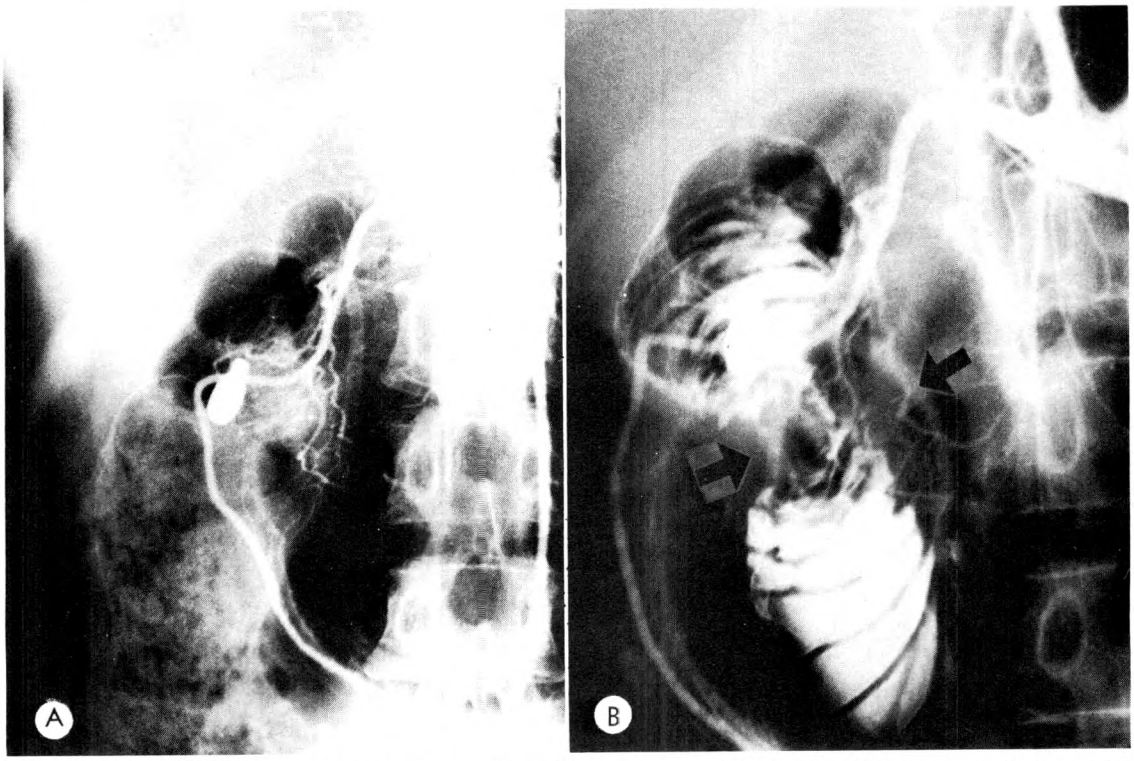


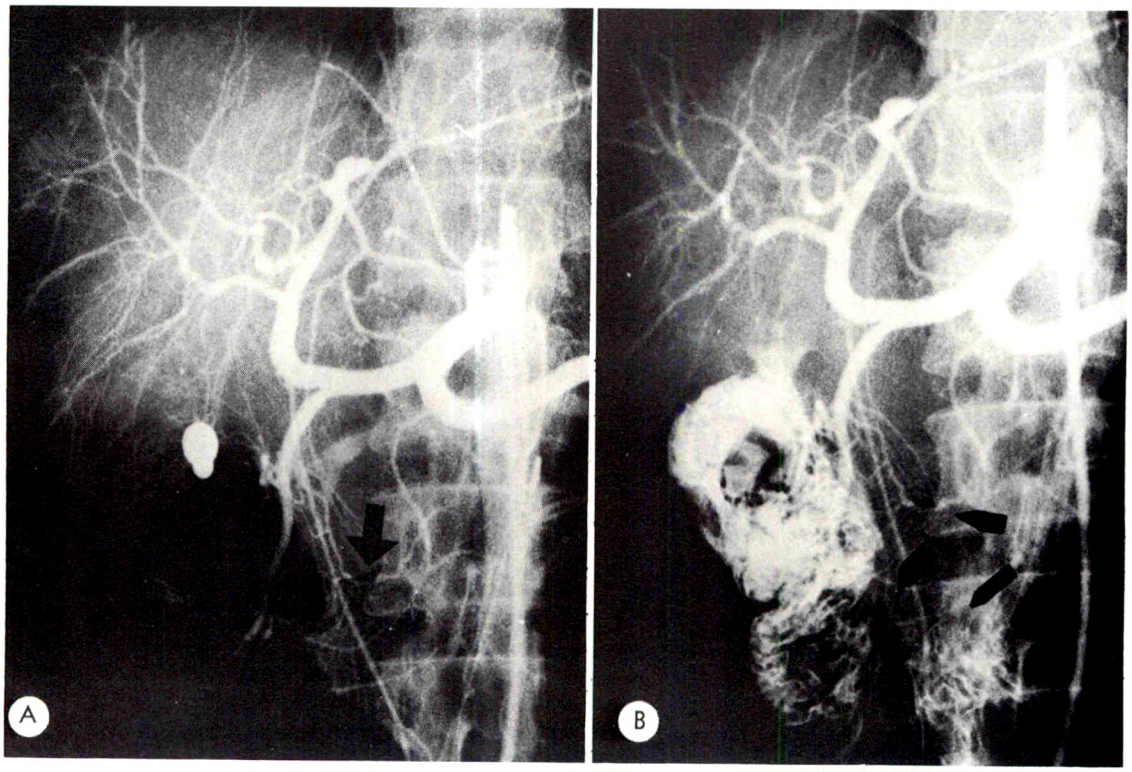
Fig. 2. Case 1. (*A*) Simple arteriogram of a patient with carcinoma of the head of the pancreas. The anterior superior and posterior superior pancreaticoduodenal arteries show irregular stenosis. (*B*) The duodenogram is superimposed. The extent of the tumor can be estimated by the mucosal destruction and vascular infiltration (arrows).

liferated tumor vessels nor tumor stain were demonstrable (Fig. 2*A*). Otherwise no evidence of abnormality was revealed in the pancreatic vessels. The simultaneously projected duodenogram showed the circumferential constriction with an incisura defect of the mid-descending duodenum, suggestive of direct invasion by the pancreatic lesion toward the duodenum (Fig. 2*B*). The size of the tumor, estimated by the combined vascular and duodenal images, was 4.0 by 3.5 cm. As to operability, we predicted that resection of the tumor might be feasible, because no invasive findings were noted in the other vessels, such as the dorsal pancreatic and inferior pancreaticoduodenal arteries.

Laparotomy revealed the mass extending from the head of the pancreas toward the duodenal mucosa. Pancreaticoduodenectomy was performed. The size of the tumor, measured directly on cut section, was 4.5 by 3.5 cm. Adenocarcinoma of the pancreatic duct epithelium was the histologic diagnosis.

Comment. In this patient, a diagnosis of carcinoma of the pancreas could be made by simple arteriography alone, but combined roentgenography made it possible to demonstrate the extent and outline of the lesion. This contributory information could not be gained by the simple summation of the findings noted on each roentgenogram separately.

Case II. A 45 year old female presented with jaundice of 1½ months' duration. On angiography, the posterior inferior pancreaticoduodenal artery showed a ragged contour, indicative of carcinoma originating on the posterior surface of the head of the pancreas (Fig. 3*A*). In this roentgenogram alone, the growth of the tumor could not be assessed, because tumor stain was not visible. On combined duodenography, the destroyed mucosal margin along the lower descending and horizontal duodenum was noticeable (Fig. 3*B*). By the combination of these vascular and duodenal images, the extent of the tumor could be evaluated, suggesting that the lesion was too far advanced to permit its radical resection. Figure 3*C* shows schematic illustration of this combined roentgenogram,



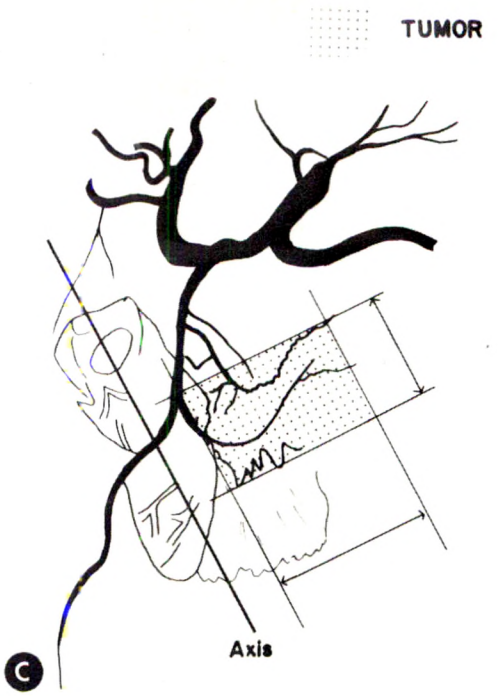


FIG. 3. Case II. (*A*) Simple arteriogram of a patient with carcinoma of the head of the pancreas. The posterior inferior pancreaticoduodenal artery is invaded (arrow). (*B*) The duodenogram is combined. The mucosal margin along the lower descending and horizontal duodenum is destroyed. By the combination of vascular and duodenal changes (arrows), the growth of tumor can be assessed. (*C*) Schematic illustration of the estimated extension of the tumor.

in which the outline of the tumor is estimated.

Surgery revealed carcinoma arising from the lower portion of the head of the pancreas with metastasis to the pericholedochal lymph nodes, causing obstructive jaundice. Biliodigestive anastomosis was performed.

Comment. The net size of this tumor could not be measured because the lesion was unresectable, but the location, extent and growth of the tumor shown in the combined roentgenogram were correlated with those found at surgery. Thus, the complementary nature of the combined vascular and duodenal roentgenograms contributed to a more accurate visualization of the lesion within the gland.

Case III. A 58 year old male had combined selective arteriography and hypotonic duodenography for longstanding painless jaundice. As shown in Figure 4, the gastroduodenal artery is deviated medially, the posterior pancreaticoduodenal arcade is irregularly invaded, and the cystic artery is remarkably distended, indicating the presence of carcinoma of the pancreas. In the combined duodenogram, the upper descending duodenum exhibits tubular constriction, which proves to be caused by the distended cystic artery. Our diagnosis was carcinoma of the head of the pancreas without duodenal infiltration, which was later confirmed by laparotomy.

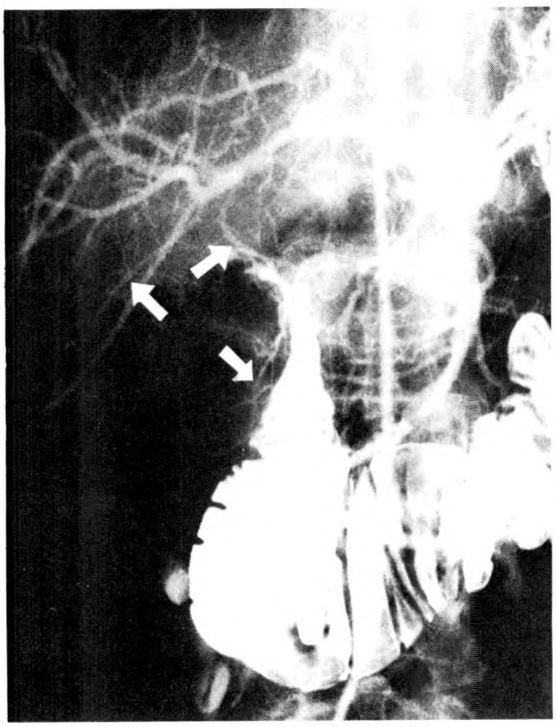


Fig. 4. Case III. Combined roentgenogram of a patient with carcinoma of the head of the pancreas. The upper descending duodenum reveals tubular constriction, which proves to be caused by the distended cystic artery (arrows), rather than by the infiltration of tumor. Medial deviation of the gastroduodenal artery and irregular stenosis of the posterior pancreaticoduodenal arcade are also seen.

Comment. By use of the combined procedure, it could be suggested that deformity of the duodenum was due to compression by the enlarged gallbladder, rather than due to invasion by the tumor, because the distended cystic artery corresponded to the area with impressed duodenal margin.

Case IV. A 65 year old male was admitted to our hospital with slight jaundice and dull abdominal pain of 3 months' duration. On simple arteriography, there was no pathology to indicate with confidence the diagnosis, except for the very meager findings in the finer branches of the pancreaticoduodenal arcades and distention of the cystic artery. Combined duodenography showed smoothly marginated double contour on the medial aspect of the descending duodenum, which corresponded to the area of the above mentioned minimal vascular lesions (Fig. 5). The final diagnosis was carcinoma of the ampulla of Vater, for which pancreaticoduodenectomy was performed. The size of the tumor was 2.5 by 2.0 cm.

Comment. On the angiogram, identification of finer tumor vessels in the pancreas is often difficult, even by use of the superselective and pharmacodynamic techniques. In this situation the simultaneously projected image of the duodenal mucosa is of considerable help to determine whether these subtle vessels are normal or pathologic. Assessment of these minimal findings cannot be made by the separately taken roentgenograms, because there is an unexpectedly wide variation in the relative position of the duodenum with respect to the pancreatic vessels.[14] As illustrated in this case, the slight abnormalities suspected by angiography could be further ascertained by the combined projection of the hypotonic duodenogram.

Case V. A 69 year old male came to our clinic with the complaint of longstanding jaundice. Selective celiac angiography showed no

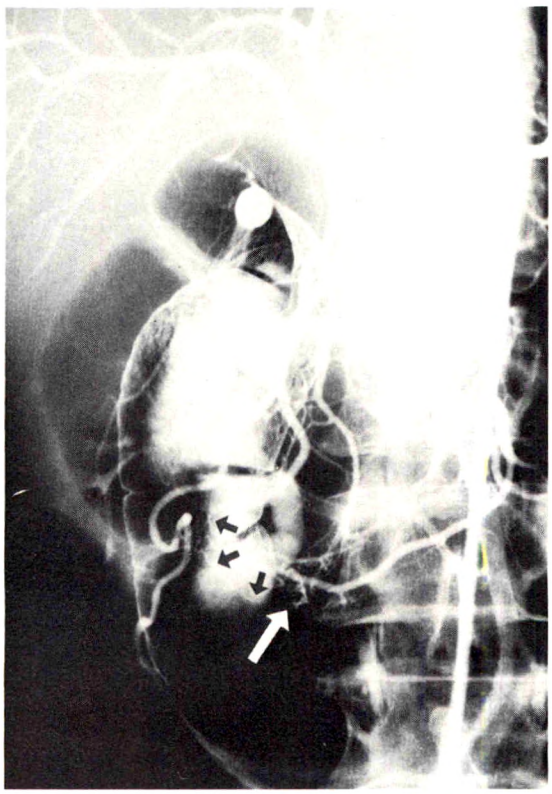


Fig. 5. Case iv. Combined roentgenogram of a patient with carcinoma of the ampulla of Vater. The smoothly marginated double contour of the medial descending duodenum is revealed on the duodenogram (black arrows), where slight changes in the finer vessels are shown (white arrow).

evidence of abnormality along the vessels of the pancreas, gallbladder and liver (Fig. 6*A*). On hypotonic duodenography combined with common hepatic arteriography, no additional pathology was noticed in the duodenal mucosa or the finer vessels supplying the corresponding portion (Fig. 6*B*). The pancreaticoduodenal region was free of pathology. On laparotomy, no changes were found in the pancreas, duodenum and extrahepatic biliary tract. Operative cholangiography also revealed normal appearance of the intrahepatic bile duct. The final diagnosis was cholangiolitic hepatitis, which was confirmed by the biopsied specimen of the liver.

Comment. Even when there is no evidence of abnormality in the arteriogram, the angiographers sometimes hesitate to say that there is no pathology present. By means of combined roentgenography, we were able to conclude with confidence that this jaundiced patient was free of a pancreaticoduodenal lesion.

Discussion

Despite the recent advances in gastroenterology, the early detection of carcinoma of the pancreas is still a challenging problem. During the past decade, various attempts were made to define details of this elusive organ, and their diagnostic usefulness has been compared with each other. Satisfactory information, however, could not be attained by use of only one method alone. Eaton and his colleagues[3] performed several diagnostic procedures on the same patients with pancreatic disease. According to their results, the most appropriate approaches for pancreaticoduodenal carcinoma appeared to be duodenography and angiography, the diagnostic accuracy of which was 75 and 80 per cent, respectively. Each technique has its own advantage and disadvantage, which can be supplemented and complemented by each other. On the basis of the results obtained by the preliminary study,[14] we began to use the new combination of arteriography and duodenography for all patients suspected of harboring pancreatic lesions.

The usefulness of the combined roentgenogram can be summarized as follows.

1. Precise location of the vessels supplying the pancreas can be identified by the simultaneous projection of the duodenal loop.

As has been widely accepted, the anatomy of the pancreatic vessels varies so much that it is difficult to assess the detailed localization of the lesion within the gland by the sole use of angiography. The combined projection of the duodenal image is of greatest help, because the duodenal loop can reveal the profile of the head of the pancreas.

2. The extent of the pancreatic lesion can be fairly accurately estimated by the simultaneously projected vascular and duodenal images.

As already described in the previous report,[14] there is an unexpectedly wide

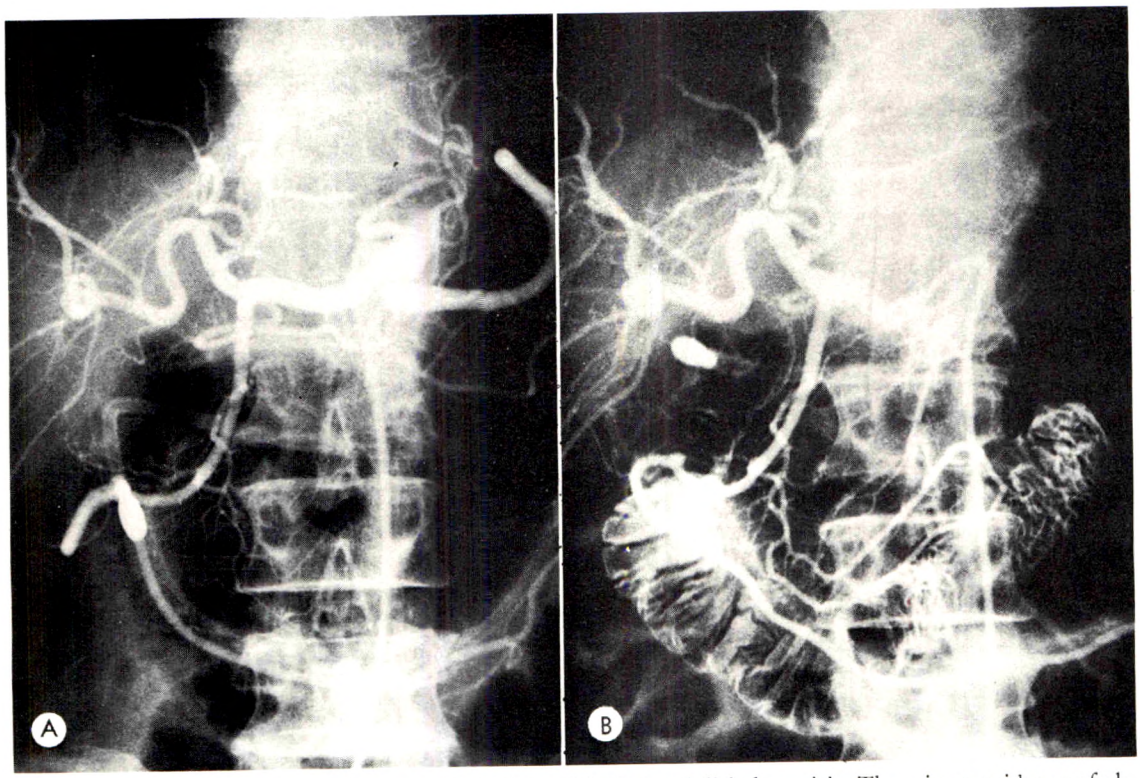


Fig. 6. Case v. (*A*) Simple arteriogram of a patient with cholangiolitic hepatitis. There is no evidence of abnormality. (*B*) The hypotonic duodenogram is superimposed. No additional pathology is noted in the duodenal mucosa or the finer vessels.

variation in the relative position of the pancreatic vessels with respect to the duodenal loop, even in the normal subject. For this reason, satisfactory information on the size, extent, form and growth of the lesion cannot be gained by the summation of the findings noted on 2 roentgenograms taken separately.

3. Identification of the minimal abnormalities suspected by angiography can be enhanced by the hypotonic duodenogram imaged on the same film.

It is often confusing and perplexing to interpret the subtle changes in the finer pancreaticoduodenal vessels. The simultaneously projected duodenogram plays an important role when it is difficult to determine whether these vascular findings are pathologic or normal.

4. Combined arteriography and duodenography might enable the surgeon to devise the appropriate therapeutic plan.

One cannot perform pancreaticoduodenectomy without severing the gastroduodenal artery, the site of which can be distinctly predicted prior to surgery in the combined roentgenogram. The aberrant hepatic artery originating in the superior mesenteric artery is the most frequently encountered variation.[5,10] During operation upon the pancreas, an effort should be made to preserve this variant artery, because it runs upward along the dorsal surface of the pancreas. The combined roentgenography makes it possible to reveal the relative position of this variant artery with respect to the duodenum and pancreatic vessels. This information can help surgeons to plan the operative procedure and to save time. For the selective demonstration of the aberrant hepatic and pancreaticoduodenal arteries, we use the technique with double catheters and vasoconstrictive agents, the details of

which were described previously.[11] Apart from simplification of the operative problems, angiography of the pancreas has additional advantage and usefulness in the surgical field, which were also discussed in our previous reports.[9,12,13,15,16]

Other combinations of 2 different procedures have been attempted by some investigators: the combination of cholangiography and splenoportography was reported by Miller *et al.*,[6] and Sato *et al.*[8]; the combination of duodenography and scintigraphy by Baum and Howe,[1] and McCarthy *et al.*[4]; the combination of arteriography and cholangiography by Boijsen and Reuter[2]; and the combination of duodenography and cholangiography by Raia,[7] and Baum and Howe.[1] All of these combined procedures have their specific advantage. In comparison with them, our method appears to be much safer and practicable, although some problems still remain. The following illustration suggests the advisability of percutaneous cholangiography in combination with the present arteriography and duodenography. Figure 7, *A* and *B*, shows the roentgenograms on which the vascular, duodenal and biliary systems are visualized simultaneously. This is the patient with carcinoma of the pancreas, who underwent choledochoduodenostomy prior to the examination. The contrast medium infused into the duodenum entered the common bile duct through the anastomosis. Arteriography shows rough tapering of the common hepatic, gastroduodenal, superior mesenteric and aberrant right hepatic arteries and their branches; duo-

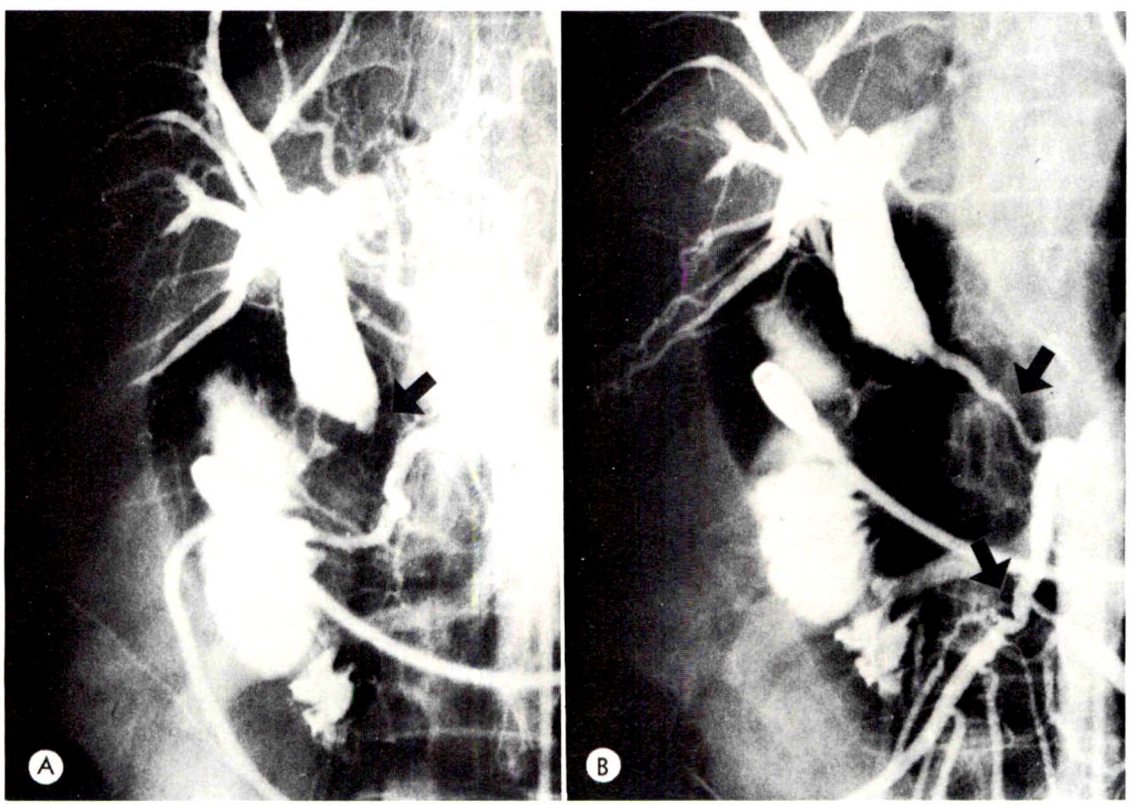


Fig. 7. (*A*) Combined roentgenogram of a patient with carcinoma of the pancreas. In addition to vascular invasion of the gastroduodenal artery and mucosal destruction of the duodenal margin, complete obstruction of the terminal bile duct (arrow) is simultaneously visualized. Thus, the combination of vascular, duodenal and biliary images yields a clearer demonstration of the tumor extension. (*B*) The angiogram of the superior mesenteric artery is superimposed. The remarkable infiltration by the tumor is shown (arrows). The upper arrow indicates the invaded aberrant right hepatic artery.

denography demonstrates the deep filling defect with an irregularly eroded base, and sharp spicules along the concavity of the duodenal loop; and cholangiography discloses complete obstruction of the terminal bile duct. As a result, the boundaries of the tumor mass in the gland can be more distinctly delineated. Thus, combination of 3 different images on the same film might yield a further dimension of the radiologic evaluation of the pancreas.

SUMMARY

Based on the facts specified in the preliminary study, we routinely made use of combined selective arteriography and hypotonic duodenography for the examination of patients suspected of harboring a pancreatic lesion.

This diagnostic procedure is more advantageous than the summation of the findings noted on 2 roentgenograms taken separately: precise location of the vessels supplying the pancreas can be identified; the extent of the pancreatic lesion can be visualized; minimal changes in the finer vessels can be confirmed; and an appropriate therapeutic plan can be established. These facts were documented in a group of patients.

The further addition of cholangiography to the combined selective arteriography and hypotonic duodenography is suggested.

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REFERENCES

1. Baum, M., and Howe, T. Hypotonic duodenography in diagnosis of carcinoma of pancreas and its further use when combined with percutaneous cholangiography and pancreatic scintiscanning. *Am. J. Surg.*, 1968, *115*, 519–525.
2. Boijsen, E., and Reuter, S. R. Combined percutaneous transhepatic cholangiography and angiography in evaluation of obstructive jaundice. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 153–161
3. Eaton, S. B., Fleischli, D. J., Pollard, J. J., Nebesar, R. A., and Potsaid, M. S. Comparison of current radiologic approaches to diagnosis of pancreatic disease. *New England J. Med.*, 1968, *279*, 389–396.
4. McCarthy, D. M., Kreel, L., Agnew, J. E., and Bouchier, I. A. D. Value of hypotonic duodenography as adjunct to pancreatic scanning. *Gut*, 1969, *10*, 665–673.
5. Michels, N. A. Blood Supply and Anatomy of Upper Abdominal Organs with Descriptive Atlas. J. B. Lippincott Company, Philadelphia, 1955.
6. Miller, H., Del Guercio, L. R. M., Cohn, J. D., Feins, N. R., and Coomaraswamy, R. P. Transhepatic cholangiography combined with splenoportography in biliary pancreatic carcinoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *96*, 468–476.
7. Raia, S. Percutaneous cholangiography and simultaneous duodenography. *Surgery*, 1966, *60*, 1125–1128.
8. Sato, T., Kakizaki, G., Saito, Y., Maki, T., and Koyama, K. Combined method for diagnosis in malignant jaundice. *A.M.A. Arch. Surg.*, 1967, *95*, 207–216.
9. Suzuki, T., Kawabe, K., Nakayasu, A., Takeda, H., Kobayashi, K., Kubota, N., and Honjo, I. Selective arteriography in cancer of pancreas at resectable stage. *Am. J. Surg.*, 1971, *122*, 402–407.
10. Suzuki, T., Nakayasu, A., Kawabe, K., Takeda, H., and Honjo, I. Surgical significance of anatomic variations of hepatic artery. *Am. J. Surg.*, 1971, *122*, 505–512.
11. Suzuki, T., Imamura, M., Kawabe, K., and Honjo, I. Selective demonstration of variant hepatic artery. *Surg., Gynec. & Obst.*, 1972, *135*, 209–215.
12. Suzuki, T., Kuratsuka, H., Uchida, K., Matsumoto, Y., and Honjo, I. Carcinoma of pancreas arising in region of uncinate process. *Cancer*, 1972, *30*, 796–800.
13. Suzuki, T., Kawabe, K., Imamura, M., and Honjo, I. Survival of patients with cancer of pancreas in relation to findings on arteriography. *Ann. Surg.*, 1972, *176*, 37–47.
14. Suzuki, T., Uchida, K., Kuratsuka, H., Kawabe, K., Imamura, M., and Honjo, I. Usefulness of combined selective arteriography and hypotonic duodenography in evaluation of cancer of pancreas. *Ann. Surg.*, 1972, *176*, 791–797.
15. Suzuki, T., Kuratsuka, H., Uchida, K., Nagamatsu, Y., and Honjo, I. Correlation between clinical aspects and location of lesion in carcinoma of head of pancreas. *Am. J. Surg.*, 1973, *125*, 546–550.
16. Suzuki, T., Kitagawa, S., and Honjo, I. Role of splenic arteriography in evaluation of malignant tumors in body of pancreas. (In press.)

CURRENT ASPECTS OF ULTRASONIC SCANNING OF THE PANCREAS*

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SONOGRAPHY today affords a simple and reliable means of demonstrating various disorders of the upper abdomen, especially space-occupying lesions. The basic principles of the method and the particular points of our approach are extensively described elsewhere.[1,4,7,9,11,12]

Ultrasonic diagnosis readily supplies definite evidence of a pathologic process, but the interpretation of an abnormal ultrasonic scan presents difficulties. To interpret the scan it must be possible to sort out the topographic relationships and relate the pathologic structures to the various organs.

Figure 1, *A* and *B*, illustrates the problem by means of 2 simple examples: in both sonograms, showing transverse sections through the upper abdomen, there are dorsally the negative contrasts of the spine and the aorta; alongside these the zone of shadow dorsal to the stomach; and on the right the profiles of the liver and the right kidney. The pathologic evidence is unmistakable in both sonograms, which are of 2 different patients: the large approximately round clear area to the right in the abdomen corresponds in both cases to a cystic tumor, the presence of which can be readily demonstrated by a sonar study. *But to which organs are these 2 lesions to be assigned?* This is the question which brings us face to face with the difficulties of ultrasonic diagnosis of the upper abdomen. In Figure 1*A*, the complete series of craniocaudal sections shows that the cyst is not directly connected with any of the organs demonstrable by sonography. Surgery revealed that what was involved was a simple cyst of the falciform ligament. In Figure 1*B*, serial sections show that the stalk of the

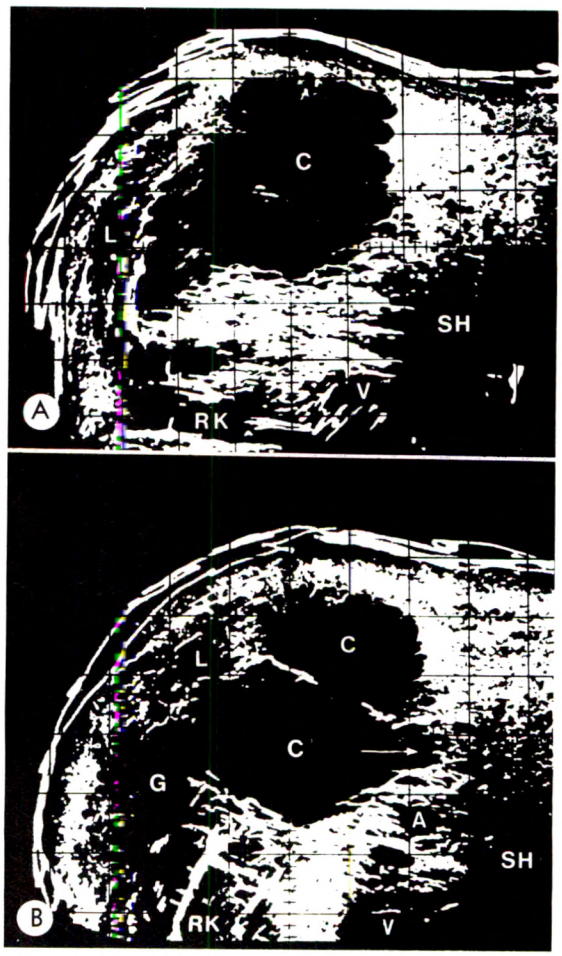


FIG. 1. Two examples of a cystic tumor in the right upper abdomen. (*A*) A cyst of the falciform ligament. (*B*) A pseudocyst of the pancreas.

C = cyst; L = liver; G = gallbladder; RK = right kidney; A = aorta; V = vertebral column; SH = zone of shadow dorsal to the stomach; Arrow = stalk of the cyst running into the region of the pancreas.

Both of these sonograms, and all those that follow, are transverse sections through the upper abdomen made with the patient in the supine position. The right side of the body is at the left of the scan. (Picker-Laminograph, Model 102, Option 3. Transducer 2MHz.)

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loculated cyst runs directly into the region of the pancreas. The diagnosis, or, more accurately the assignment of the stalk of the cyst to the pancreas, is possible only if the topography of the pancreas in the sonogram is known. The criteria used to describe the region of the pancreas in sonography will be discussed.

TOPOGRAPHY UNDER NORMAL CONDITIONS

The most important landmark for topographic orientation in the area of the pancreas is the *portal vein*, which is shown by splenoportography in Figure 2. Its oblique course from caudal left to cranial right can readily be reconstructed on serial section scans. Figure 3 illustrates a normal appearance.[8] The pancreas itself cannot be specifically visualized in the sonogram. It produces a diffuse, mosaic-like area of echoes in a triangle formed by the posterior

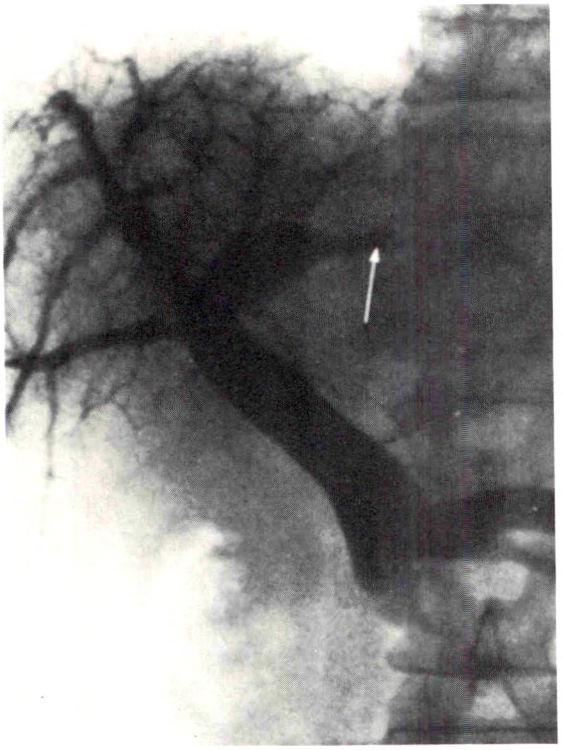


Fig. 2. Normal splenic portogram illustrating the oblique course of the portal vein. Arrow = ramification of the portal vein in the left lobe of the liver (see Fig. 3, No. 1).

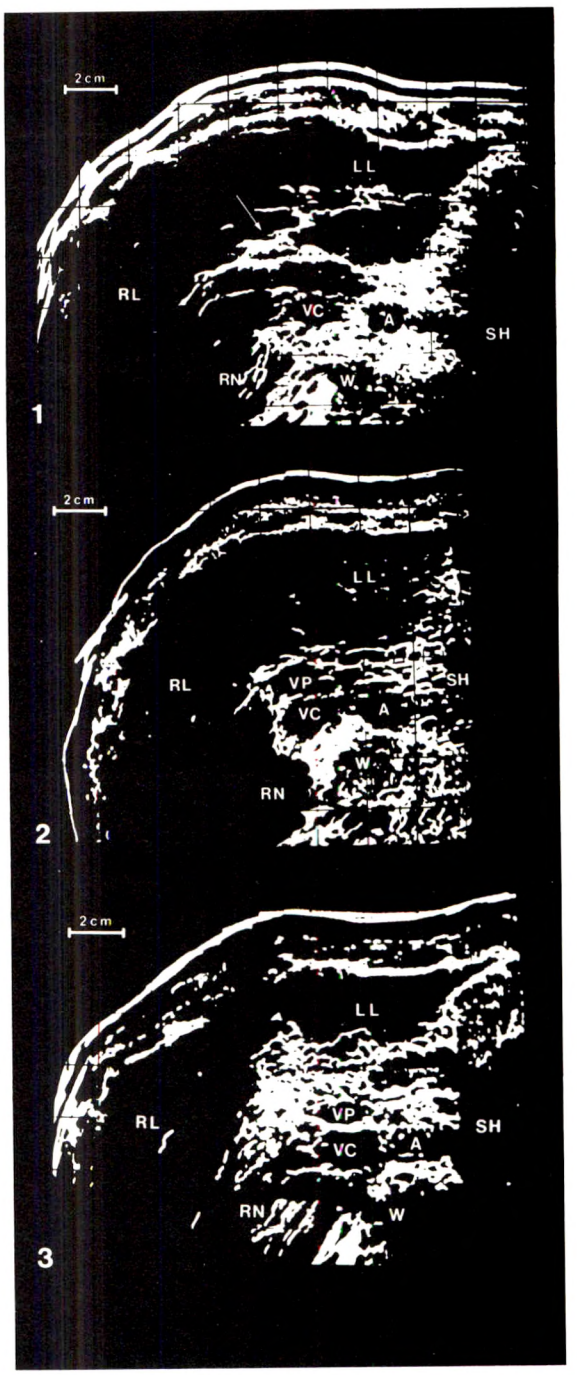


Fig. 3. Topography in serial transverse sections of the upper abdomen under normal conditions: (1) section above the pancreatic region; (2) section through the body of the pancreas; and (3) section through the head of the pancreas.

RL/LL = right/left lobe of the liver; RN = right kidney; VP = portal vein; A = aorta; VC = vena cava; W = vertebral column; SH = shadow zone dorsal to the stomach.

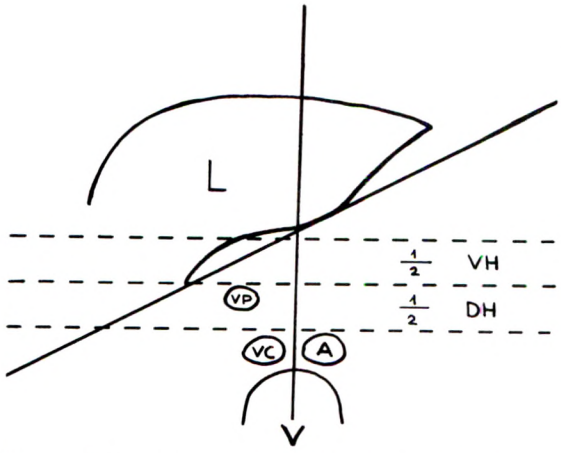


Fig. 4. Schematic drawing of the pancreatic region showing the ventral and dorsal half (VH/DH) of the sonographic compartments discussed in the text.

L = liver; VP = portal vein; A = aorta; VC = vena cava; V = vertebral column.

surface of the liver, the right margin of the stomach shadow, and an imaginary line drawn to the vena cava and the aorta (Fig. 4). This triangle we term the "*sonographic pancreas region*," provided the negative contrast of the portal vein is contained therein. At the level of the *body* of the pancreas the portal vein lies directly ventral to the vena cava or ventrally and to the right of it; at the level of the *head* of the pancreas the portal vein lies more medially, as would be expected by reason of its oblique course (Fig. 2). In the transverse section, slightly cranial to the body of the pancreas, there is no visualization of the trunk of the portal vein, and at this level the section therefore lies outside the sonographic pancreas region (Fig. 3, No. 1). The ramifications of the portal vein in the liver are usually clearly visualized on this section. The *tail* of the pancreas is located, as a rule, in the shadow zone dorsal to the stomach and its outline cannot normally be defined. The triangle of the sonographic pancreas region can be divided into a ventral and a dorsal half, by drawing a transverse line half-way between the liver and the major abdominal vessels in the median line of the body (Fig. 4). A distinction between these 2 compartments is of practical importance, as will be seen later. In the ventral half lies the pancreas and in the dorsal half there are mainly the structures of the root of the mesentery. The posterior surface of the pancreas lies immediately ventral to the portal vein. The head of the pancreas envelops the portal vein on the right almost as far as the vena cava.

The following clinical examples, documented by surgery or necropsy, show the usefulness of the topographic criteria we have formulated for the ultrasonic scanning of the pancreas region.

Dilated pancreatic duct due to a carcinoma of the papilla

In the ventral half of the sonographic pancreas region (Fig. 5) one can see a clear band running from dorsal right to ventral left. This represents a markedly dilated pancreatic duct. The dilatation of this pancreatic duct is due to a carcinoma of the papilla, the negative contrast of which can be found in the right angle of the triangle formed by the sonographic pancreas region. The clear zone of the portal vein cannot be clearly distinguished from the tumor contrast. There are only rudimen-

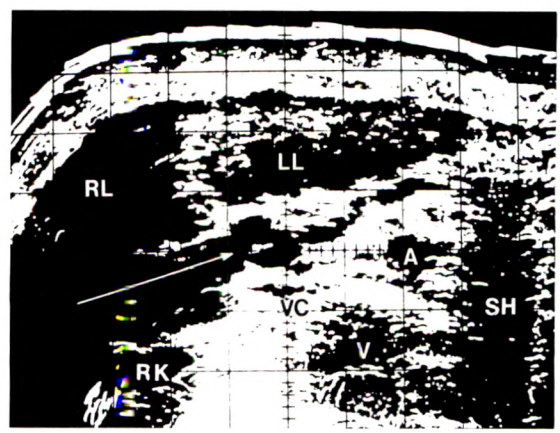


Fig. 5. Dilated pancreatic duct due to a carcinoma of the papilla.

Arrow = indicates the long axis of the pancreas; RL/LL = right/left lobe of the liver; RK = right kidney; A = aorta; VC = vena cava; V = vertebral column; SH = shadow zone dorsal to the stomach.

tary traces indicating the vena cava. The distally obstructed pancreatic duct shows roughly the position of the long axis of the organ.

ACUTE PANCREATITIS

Acute inflammation (Fig. 6) produces a marked dilatation of the sonographic region, particularly in the ventral half. The diffuse mosaic-like echo pattern of the pancreas region appears coarse and more loosely formed owing to edematous infiltration. Figure 6 is a transverse section through the body of the pancreas, for the portal vein is relatively far to the right of the median line.

FIBROSIS, NECROSIS

Fibrotic change or necrotic destruction of pancreatic tissue is acoustically more homogeneous than pancreatic parenchyma which is intact or shows little inflammatory change. Necrotic or fibrous areas thus appear in the sonogram as clearly defined but irregular echo-free zones within the pancreas region. Their figural arrangement forms a negative image of the organ and marks the topographic position of the pancreas in the transverse section.

Figure 7 illustrates an example of acute necrosis of the pancreas. One can see the negative contrast of the necrotic body of the pancreas in the ventral half of the sonographic pancreas region. The abdominal cavity on the left is occupied by a large abscess extending from the liver to the left kidney. A secondary finding in this example is an enlarged gallbladder filled with stones.[2]

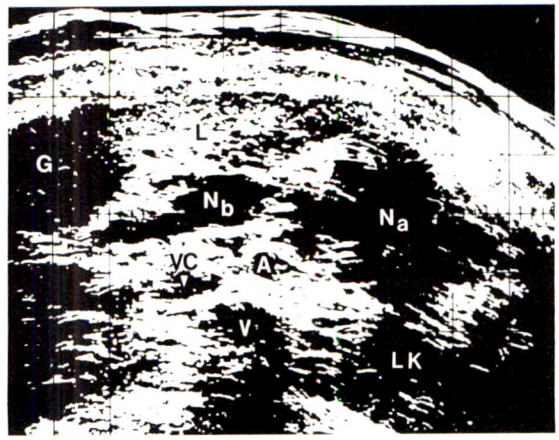


Fig. 7. Acute necrosis of the pancreas.
L = liver; G = gallbladder (filled with stones); LK = left kidney; N_a = abscess in the left abdomen; N_b = necrotic body of the pancreas; A = aorta; VC = vena cava; V = vertebral column.

In Figure 8 is demonstrated a typical scan during an acute inflammatory episode

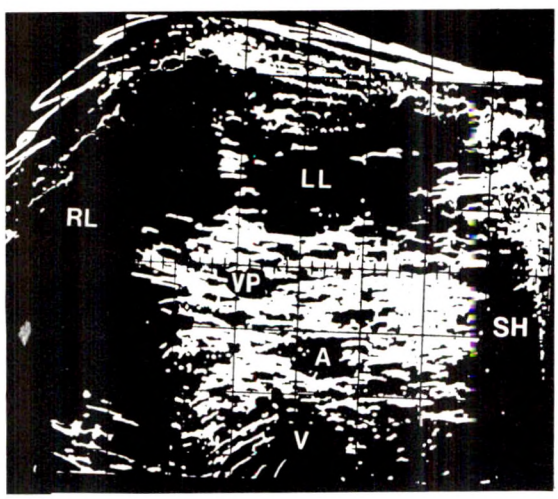


Fig. 6. Acute inflammation of the pancreas.
RL/LL = right/left lobe of the liver; VP = portal vein; A = aorta; V = vertebral column; SH = shadow zone dorsal to the stomach.

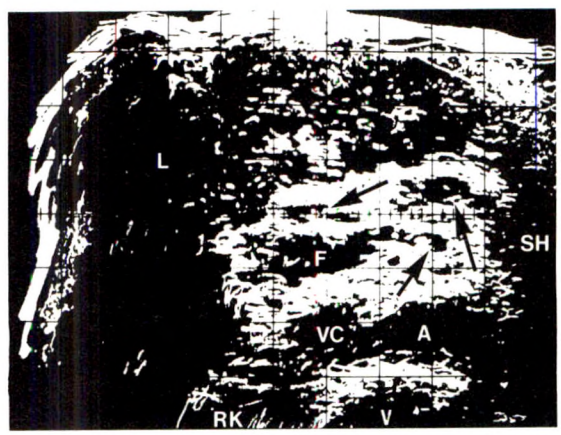


Fig. 8. Acute inflammatory episode in chronic pancreatitis.
L = liver; RK = right kidney; F = fibrotic area in the pancreas; A = aorta; VC = vena cava; V = vertebral column; SH = shadow zone dorsal to the stomach.

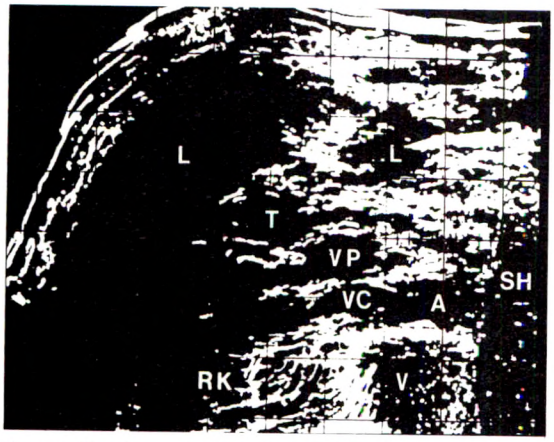


Fig. 9. Carcinoma of the head of the pancreas.
L = liver; RK = right kidney; T = tumor of the head of the pancreas; VP = portal vein; A = aorta; VC = vena cava; V = vertebral column; SH = shadow zone dorsal to the stomach.

in chronic pancreatitis. The fibrotic areas have the appearance of disseminated clear islands of irregular outline within the ventral half of the sonographic pancreas region. This is markedly distended as a result of the acute inflammation. Differential diagnosis from necrosis of the pancreas is based on a follow-up study at intervals of several days during the course of the diseases. In fibrosis of the pancreas the sonogram remains more or less constant, whereas in necrosis of the pancreas the tissue usually undergoes destruction/restitution which increases/decreases from day to day. During an interval free from inflammation in chronic pancreatitis, the pancreas area often seems reduced in size by reason of the atrophied parenchyma.

Tumors of the Head, Body and Tail of the Pancreas

The topographic criteria for evaluation of the pancreas region given at the beginning are also valid, particularly for tumor processes in the pancreas (Fig. 9; 10; 11; and 12). Solid and cystic tumors invariably appear as largely clear areas which are readily distinguishable from the diffuse echo pattern of the pancreas region by their high contrast.[5,6]

A carcinoma of the *head* of the pancreas is illustrated in Figure 9. The negative contrast marking the tumor is located typically in the right angle of the sonographic pancreas region, generally ventral to, or ventral to and on the right of, the portal vein or the vena cava. As is consistent with the clinical law of Courvoisier, a dilated gallbladder is also found sonographically in similar cases with the same implications.[3,5,10] The present example, however, shows the condition after cholecystectomy.

Figure 10 demonstrates the results of the scan when there is a solid tumor in the *body* of the pancreas. The negative contrast of the portal vein is included in the

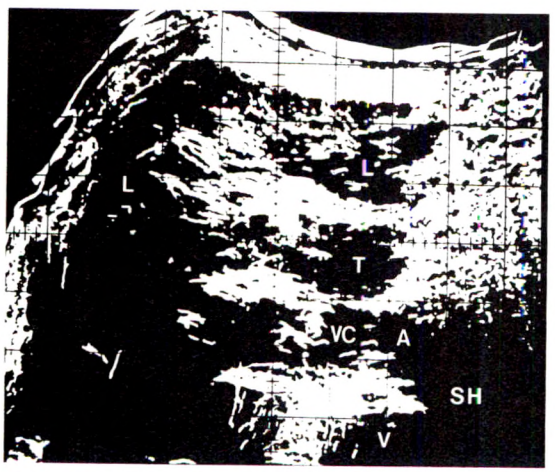


Fig. 10. Solid tumor in the body of the pancreas.
L = liver; T = tumor of the body of the pancreas; A = aorta; VC = vena cava; V = vertebral column; SH = shadow zone dorsal to the stomach.

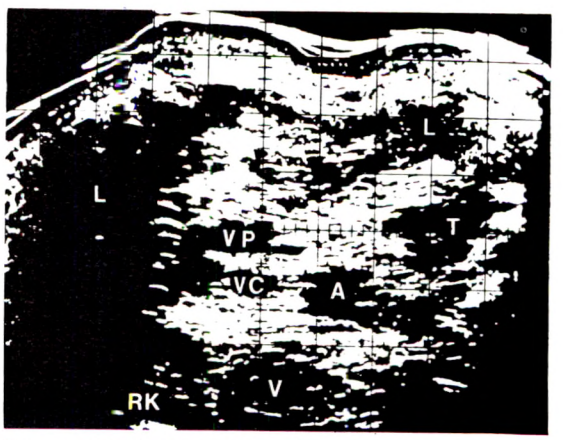


Fig. 11. Carcinoma of the tail of the pancreas.
L = liver; RK = right kidney; T = tumor of the tail of the pancreas; VP = portal vein; A = aorta; VC = vena cava; V = vertebral column.

area of the tumor and is therefore not separately identifiable. The focus of the pathologic process is clearly located in the ventral half of the sonographic pancreas region, it is, therefore, assignable to the pancreas, and is unrelated to extrapancreatic retroperitoneal structures, whose focus would be in the dorsal half.

Figure 11 shows a tumor in the area of the *tail* of the pancreas. Space-occupying lesions in the tail region of the pancreas which result in displacement of the stomach, and thus to an abolition or displacement of its shadow zone, can often be scanned by a ventral approach. From the position of the portal vein shown in Figure 11, the level of the section of the present transverse sonogram is equivalent to a section through the body of the pancreas.

The scan illustrated in Figure 12, No. 2 is the same as in Figure 1*B*—a pseudocyst of the pancreas in the region of its head. It should demonstrate that a precise topographic analysis of the sonogram enables a reliable diagnosis to be made. Study of the complete craniocaudal series of relevant transverse sections (Fig. 12) shows clearly that the pedicle of the cyst definitely runs into the body of the pancreas, and that what is involved here must be a cystic process of the pancreas region.

DISCUSSION

Our experience of the ultrasonic diagnosis of disease of the pancreas can be summarized as follows:

(A) The pancreas, which cannot be visualized by sonographic differentiation, lies in the ventral half of the triangle of what is termed the *sonographic pancreas region* bounded by: (1) the posterior surface of the liver; (2) the right border of the zone formed by the stomach shadow; and (3) an imaginary line drawn to the major abdominal vessels.

(B) Pathologic processes of the pancreas which are marked by a broadening of the pancreas echo pattern or the appearance of abnormal negative contrasts, have their focus in the ventral half of the sonographic pancreas region described.

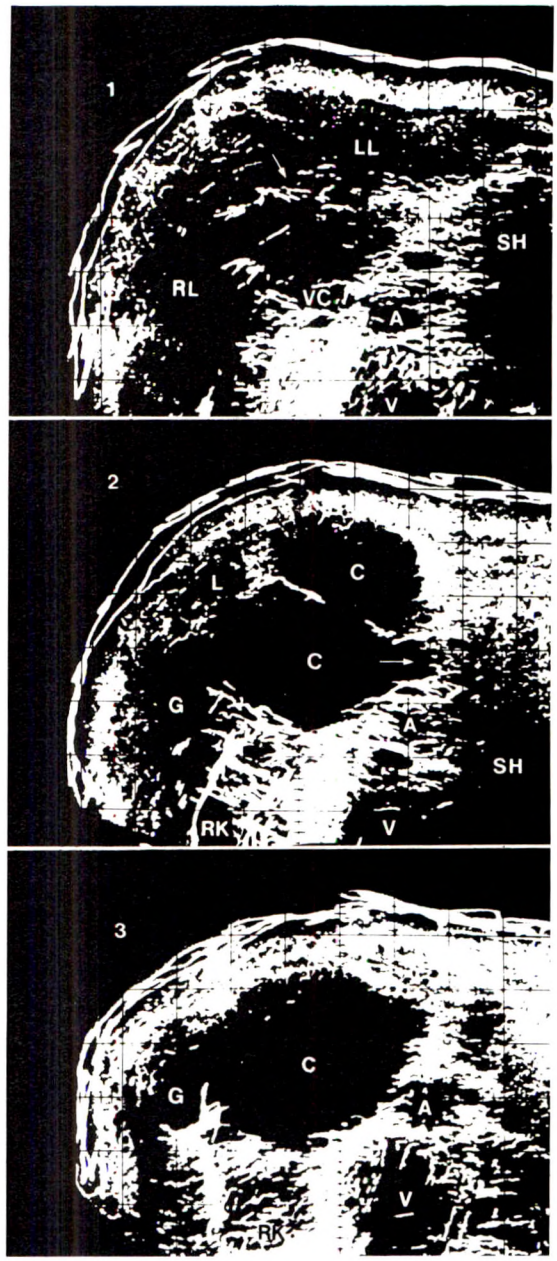


Fig. 12. Pseudocyst of the pancreas. Relevant transverse sections of the complete craniocaudal series. Congestion of the gallbladder. Cholestasis of the liver: (1) transverse section above the pancreatic region. Arrow = normal structures in the liver (ramification of the vena portae); (2) section through the body of the pancreas. Arrow = pedicle of the cyst running in the body of the pancreas; and (3) section through the head of the pancreas.

L = liver; RL/LL = right/left lobe of the liver; G = gallbladder; C = loculated cyst of the pancreas; R = right kidney; A = aorta; VC = vena cava; V = vertebral column; SH = shadow zone dorsal to the stomach.

(C) In some cases sonographic follow-up made during the course of the disease is of crucial importance for diagnostic differentiation.

SUMMARY

Ultrasonic diagnosis readily supplies definite evidence of a pathologic process, but the interpretation of an abnormal ultrasonic scan presents difficulties.

To interpret the scan, knowledge of the topographic criteria in assessing pathologic findings is necessary.

The cited clinical examples demonstrate the usefulness of the topographic criteria devised by us for the sonographic delineation of the pancreas region. They illustrate the typical echo pattern of the following pancreatic diseases: (1) dilated pancreatic duct, carcinoma of the papilla; (2) acute pancreatitis; (3) fibrosis, necrosis; and (4) tumors of the head, body and tail of the pancreas.

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REFERENCES

1. Blauenstein, U. W. Die Ultraschallschnittbilduntersuchung am Oberbauch. *Schweiz. med. Wchnschr.*, 1969, *99*, 985–992.
2. Blauenstein, U. W., and Burger, J. Sonographische Darstellung einer Pankreasnekrose: Beitrag zur Ultraschall-Tomographie der Pankreasregion. *Deutsche med. Wchnschr.*, 1973, *98*, 429–433.
3. Blauenstein, U. W., and Burger, J. Ultranschal diagnostik palpabler Oberbauchtumore. *Med. Welt*, 1973, *24*, 395–401.
4. Blauenstein, U. W., Preiswerk, M., and Müller, H. R. Physik, Technik und Terminologie der Ultraschalldiagnostik. In: Ultraschalltomographie. Edited by H. R. Müller, and U. W. Blauenstein. Benno Schwabe & Co., Basel, 1969.
5. Engelhart, G., and Blauenstein, U. W. Ultrasound in diagnosis of malignant pancreatic tumours. *Gut*, 1970, *11*, 443–449.
6. Engelhart, G. J., and Blauenstein, U. W. Ultraschalldiagnostik von Tumoren der Pankreasregion. *Méd. et Hyg.*, 1971, *29*, 118–120.
7. Engelhart, G. J., and Blauenstein, U. W. Ultraschall-Diagnostik am Oberbauch. F. K Schattauer Verlag, Stuttgart, 1972.
8. Engelhart, G. J., Blauenstein, U. W., and Burger, J. Ultraschall-Tomographie des Oberbauchsitus. *Med. Welt*, 1971, *22*, 766–774.
9. Gordon, D. Ultrasound As a Diagnostic and Surgical Tool. E. & S. Livingstone, Ltd., Edinburgh, 1964.
10. Weill, F., Ricatte, J. P., Prevotat, N., and Bonneville, J. F. Sémiologie tomo-échographique hépato-biliaire élémentaire. *Ann. de radiol.*, 1970, *13*, 567–578.
11. Wells, P. N. T. The Physical Principles of Ultrasonic Diagnosis. Academic Press, Inc., London, 1969.
12. Wells, P. N. T., Ultrasonics in Clinical Diagnosis. J. & A. Churchill, Ltd., London, 1972.

VOL. 122, NO. 2

SECONDARY GASTRIC MURAL ABNORMALITIES SIMULATING PRIMARY DISEASE IN ISOLATED CHRONIC LEFT SUBPHRENIC ABSCESS AND ISOLATED CHRONIC PANCREATITIS*

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EXCEPT for a recent article[1] describing a relative posterior gastric wall silhouette contour change in pancreatitis, there has been no reported study of mural changes of the stomach secondary to left upper quadrant chronic inflammatory disease. There have been, however, numerous reports in the literature regarding pancreatitis, pseudocysts of the lesser sac, and subphrenic abscesses, as they affect the left upper quadrant soft tissue shadows and extrinsically affect stomach and other intraperitoneal and extraperitoneal intestinal relationships.

In this communication, no attempt will be made to review the classic extrinsic manifestations of the inflammatory changes demarcated by the relationships of the anatomic landmarks of the left upper quadrant, as has been described in the literature.[2–11]

BACKGROUND

Obvious laboratory, clinical, or roentgen information may be overlooked until an isolated clinical problem acts as a catalyst for accurate interpretation and application of information which has been present but improperly interpreted or not observed, as is the case with the proverbial, primarily submerged iceberg.

The catalyst in this study was a case that was followed by us for 6 months between July, 1972 and December, 1972 before the actual clinical history was elicited. This history then permitted the correct interpretation of a slowly progressing roentgen picture of hypertrophy of the mural and rugal pattern of the cardia, fundus, and upper one-third of the pars media of the stomach (Fig. 1, *A–C*) in a 31 year old chronically ill woman. Initially, the only available history was that of a partial small intestinal resection for regional enteritis with no clinical or roentgen evidence for recurrence. In spite of moderate weight loss, physical examination and laboratory studies were noncontributory. With the slowly worsening appearance of the rugal and mural pattern of the upper one-third of the stomach, there was obvious concern for the possibility of lymphosarcoma, reticulum cell sarcoma, or even infiltrating adenocarcinoma unrelated to her known transmural enteritis. Endoscopy was normal in spite of the roentgen findings. When the accurate clinical record was finally obtained, the correct interpretation seemed obvious. The previous laparotomy was not uneventful; instead, it resulted in the demonstration of a left subphrenic abscess which responded to medical management postoperatively. Thus, the diagnosis of gastric induration secondary to the left upper quadrant subphrenic abscess was confirmed by laparotomy.

This study was undertaken to determine whether the secondary gastric changes described above were chance findings or whether they are to be anticipated in a substantial number of patients who have had chronic left upper quadrant inflammatory disease as demarcated by the anatomic barriers described in the literature.[5–11]

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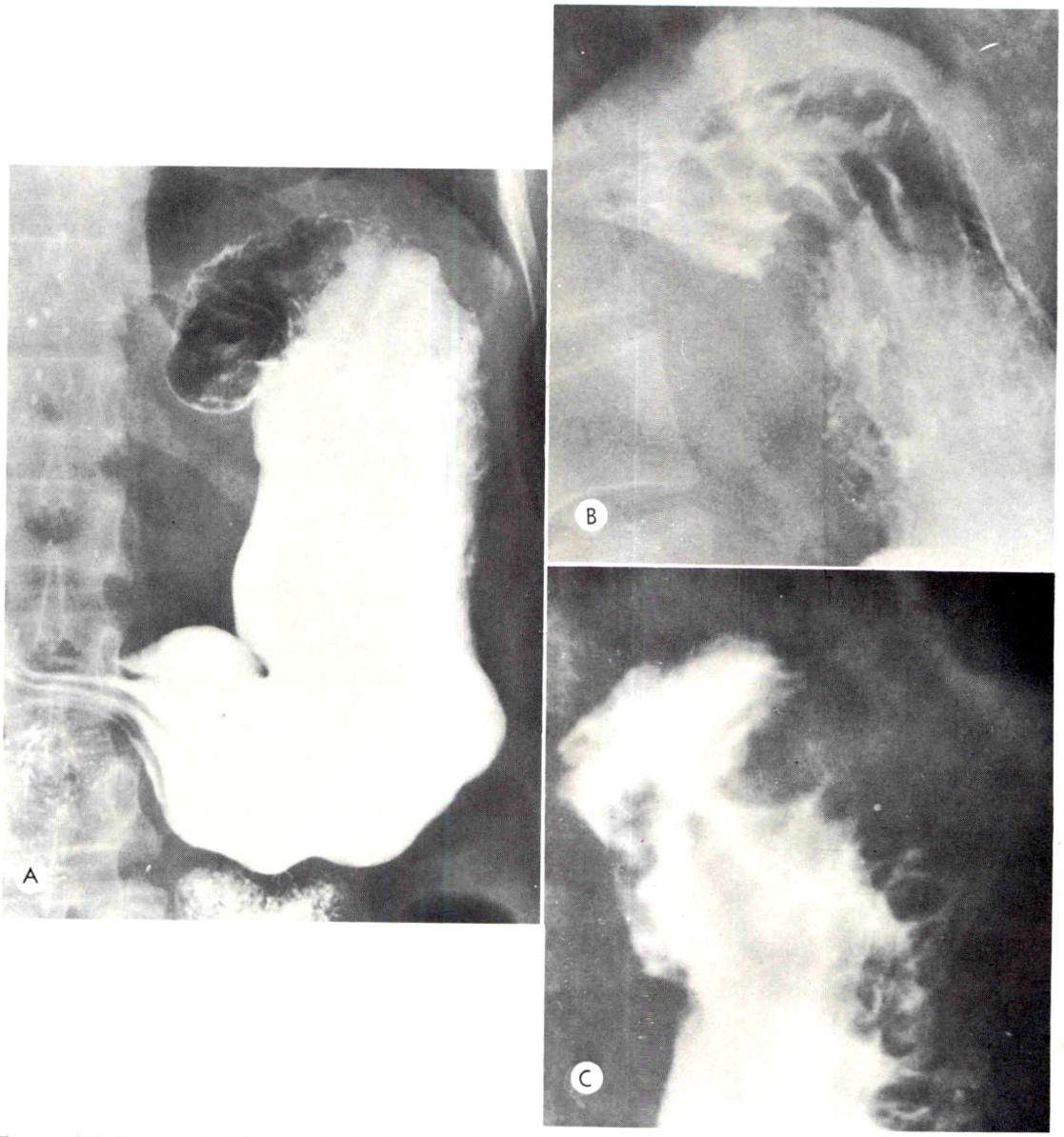


Fig. 1. (*A*) A 31 year old woman who had a laparotomy 18 months earlier for resection of small intestine granulomatous enteritis. Fundal lobular mass is seen through the barium. (*B*) Lateral view of air-filled upper half of stomach taken at same time as *A* showing nodular involvement of the cardia and posterior wall of the pars media. (*C*) Six months later, nodular submucosal and over-all mural thickening has increased to a striking degree but is still limited to the upper half of the stomach.

We reviewed cases of isolated chronic left subphrenic abscesses and isolated left upper quadrant chronic pancreatitis seen at the University of Minnesota Hospitals from 1968 to 1973. Pancreatitis is the more common entity encountered at the University of Minnesota Hospitals.

MATERIAL AND METHOD

There were 16 cases of chronic isolated left subphrenic abscess: 4 did not have roentgen studies performed at this institution; 2 had plain film roentgenograms which did show thickened folds of the stomach as well as other nongastric mani-

festations of subphrenic abscess; 5 had prominently abnormal mural and rugal folds of the upper third to upper one-half of the stomach (Fig. 1, *A–C*; and 2); 4 had minimal to moderate fold changes; and 1 was normal. Thus out of 10 gastrointestinal studies, 50 per cent were strongly positive, 40 per cent were minimally to moderately positive, and 10 per cent were normal (Table I).

Eighty-six confirmed cases of isolated chronic pancreatitis in the left upper quadrant and lesser sac were reviewed. Thirty-eight had no roentgen examinations. Forty-eight cases of the group can be broken down as follows; 36 had routine gastrointestinal contrast medium examinations; 12 had plain film abdominal roentgenography, the latter of which had varying degrees of findings suggesting lesser sac and/or bed abnormality. None of these showed unequivocal gastric wall thickening.

Of the 36 cases who had contrast medium studies, 21 had positive gastric findings (58.6 per cent), and 15 were roentgenographically normal (41.3 per cent) (Table II).

The 36 patients had a total of 52 contrast medium studies performed during the course of their illnesses. Thirty-seven of the 52 examinations were positive. Of these 37 cases, 10 (19.2 per cent) had striking mural thickening in the form of nodular or serpentine folds of the entire upper one-third to one-half of the stomach (Fig. 3). Twelve (23 per cent) had local hypertrophy of the



Fig. 2. Postoperative isolated chronic left subphrenic abscess with associated left pleural effusion. Gastrointestinal examination shows submucosal nodular configuration of cardia, fundus, and contiguous pars media junction.

Table I

16 cases of isolated chronic left subphrenic abscesses: 1968–1973

Strongly Positive Gastrointestinal Findings	Moderately Positive Gastrointestinal Findings	Normal Gastrointestinal Findings	Abdominal Scout Roentgenograms Showing Gastric Wall Thickening	No Roentgen Studies at University Hospital
5	4	1	2	4

Table II

86 cases of isolated lesser sac and left upper quadrant chronic pancreatitis: 1968–1973

Positive Gastrointestinal Findings	Normal Gastrointestinal Findings	Abdominal Scout Roentgenograms Showing No Gastric Wall Thickening	No Roentgen Studies at University Hospital
21	15	12	38

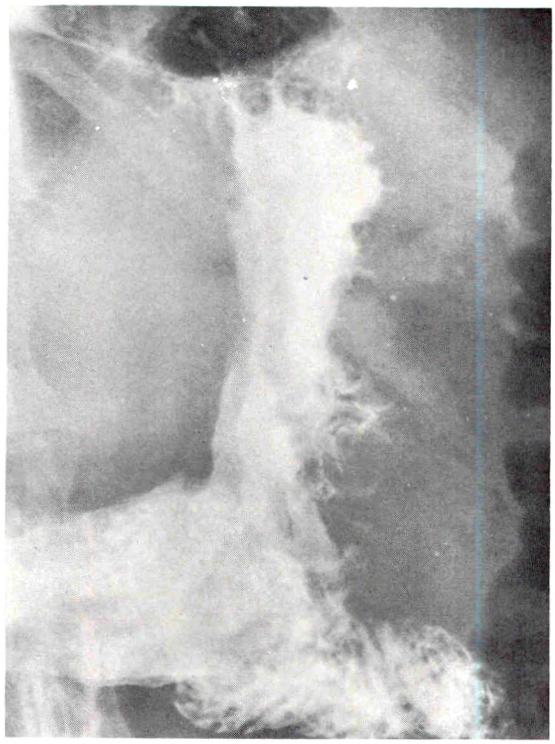


FIG. 3. Typical isolated chronic pancreatitis showing serpentine rugal hypertrophy of the upper half of the pars media and a lesser degree of involvement of the cardia and fundus. The lesser curvature is fixed by lesser sac induration. The greater curvature is fixed by inflammatory involvement of the gastrolienal and phrenicocolic ligaments.

portion of the stomach in contact with the pancreatic bed or lateral left upper quadrant including the gutter. One (1.9 per cent) had simple nonspecific rugal hypertrophy. Fourteen (26.9 per cent) had positive classical changes of pancreatitis such as widening and defacement of the duodenal loop, spasm of duodenitis, pseudocyst gastric displacement, pancreatic necrosis, and pancreatic calculi, but they did not have the mural gastric abnormality described above. Fifteen (28.8 per cent) were normal (Table III).

ROENTGEN FINDINGS

The contrast medium roentgenographic findings can be simply described as being those of submucosal and mural nodular to serpentine gastric wall thickening in the area contiguous to and with the zone of chronic inflammatory disease in the left subphrenic space or the lesser sac, demarcated by the transverse mesocolon below and extending laterally to the gutter as demarcated inferiorly by the phrenicocolic ligament.

One might find differentiation between extensive chronic pancreatitis or subphrenic abscess induced gastric induration difficult, but based on the 10 severe pancreatitis cases and the 5 severe subphrenic abscess cases, we feel that not only the diagnosis of induration is possible, but also that the site of origin of the extragastric inflammation is probably determinable as well.

In the subphrenic abscess cases, the findings were more localized to cardia and fundus (Fig. 1, *A–C*). The mucosa *per se* was not disrupted or ulcerated. The rugae were more nodular (Fig. 2) and the wall was more likely locally thickened in a circumferential fashion. The pars media was less involved and extended primarily no further than the upper third thereof.

In the pancreatitis cases, the gastric involvement seemed more extensive but more prominent along the lesser curvature,

TABLE III

TOTAL OF 52 GASTROINTESTINAL STUDIES IN 36 PATIENTS WITH ISOLATED LESSER SAC AND LEFT UPPER QUADRANT CHRONIC PANCREATITIS

Strongly Positive Pars Media Involvement	Moderately Positive Pars Media Involvement	Classical Pancreatitis Changes with Absent Gastric Mural Involvement	Nonspecific General Rugal Hypertrophy	Normal
10	12	14	1	15

posterior wall, and greater curvature aspect of the stomach (Fig. 3), even though the folds *per se* were less thickened and less abnormal in the cardia and fundus than in the pars media. The fold and mural patterns appeared less nodular and more longitudinally serpentine than in the isolated abscess cases.

DISCUSSION

Although there have been many reports published in the literature on the subject, only Balthazar[1] has recently described an actual gastric silhouette change in pancreatitis. Others[2–4] have confined their observations to the classical extragastric manifestations.

Knowledge of the anatomy of the area is essential in order to understand the limitations of involvement of the proximal stomach as described above. The relationships between the left subphrenic space, left lobe of the liver, spleen, gastroesophageal junction, stomach, lesser sac, transverse colon, transverse mesocolon, and phrenicocolic ligament have been superbly described in many publications[5–11] and will not be repeated here. Nevertheless, as stated, this understanding is essential for the recognition of the fact that an extrinsic chronic inflammatory process confined to the left upper quadrant compartment can be responsible for causing extramucosal mural involvement of the stomach which might be mistaken for tumors such as lymphosarcoma, reticulum cell sarcoma, or carcinoma.

Even though these neoplastic changes can involve the upper confines of the stomach, the limitation to this quarter in an obvious case of inflammation, or in an unknown case as was described earlier in this report, will serve the examiner as a possible clue for inflammation due to the inability to extend below the level of the transverse mesocolon or phrenicocolic ligament as demarcation planes. In generalized neoplastic involvement, extension is probable. Benign giant rugal hypertrophy, Menetrier's disease, extrinsic neoplastic invasion permeating the gastric wall, nonspecific or phlegmonous gastritis, etc., would have to be considered in the differential diagnosis, but the distribution of the zone of involvement would be quite different and more generalized. This is exclusive of the obvious differences in clinical histories for varying entities mentioned above.

CONCLUSION

As based on this study, the authors believe that by combining anatomic knowledge, clinical history, laboratory findings, and roentgen studies, one might be able to avoid the erroneous diagnosis of primary gastric disease when large nodular to serpentine folds are found at the level of the gastric cardia, to and including the fundus and upper pars media as demarcated inferiorly by the transverse mesocolon and phrenicocolic ligament attachments. This distribution should alert one to the possibility of chronic isolated left upper quadrant inflammatory disease, such as subphrenic abscess or chronic pancreatitis limited to the pancreatic bed and left upper quadrant.

In our 5 year University of Minnesota Hospitals study, the 86 cases of chronic pancreatitis whose extension was limited to the left upper quadrant and the 16 cases of isolated left subphrenic abscess confirm this theory.

In the future we hope to analyze secondary intestinal tract effects in other abdominal quadrants associated with chronic infection.

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REFERENCES

1. Balthazar, E., and Henderson, M. Prominent folds on posterior wall and lesser curvature of stomach. *Radiology*, 1974, *110*, 319–321.
2. Clemett, A. R. Examination of pancreas. In: Alimentary Tract Roentgenology. Second edition. Chapter 42. Edited by A. R. Margulis,

and H. J. Burhenne. C. V. Mosby Company, St. Louis, 1973, pp. 1107–1151.

3. Eckert, J. F., Marriott, J. D., and Smith, B. H. Exercise in radiologic-pathologic correlation. *Radiology*, 1968, *91*, 572–581.
4. Mellins, H. Z. Lesser peritoneal sac. In: Alimentary Tract Roentgenology. Second edition. Chapter 45. Edited by A. R. Margulis, and H. J. Burhenne. C. V. Mosby Company, St. Louis, 1973, pp. 1182–1193.
5. Meyers, M. A. Roentgen significance of phrenicocolic ligament. *Radiology*, 1970, *95*, 539–545.
6. Meyers, M. A. Spread and localization of acute intraperitoneal effusions. *Radiology*, 1970, *95*, 547–554.
7. Meyers, M. A., and Whalen, J. P. Radiologic aspects of intra-abdominal abscesses. In: The Diagnosis and Treatment of Abdominal Abscesses. Chapter 5. Edited by I. M. Ariel, and K. Kazarian. Williams & Wilkins Company, Baltimore, 1971, pp. 87–127.
8. Meyers, M. A., Whalen, J. P., Peele, K., and Belne, A. S. Radiologic features of extraperitoneal effusions: anatomic approach. *Radiology*, 1972, *104*, 249–257.
9. Meyers, M. A., and Whalen, J. P. Roentgen significance of duodenocolic relationships: anatomic approach. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 263–274.
10. Whalen, M. P., and Bierny, J. P. Classification of perihepatic abscesses. *Radiology*, 1969, *92*, 1427–1437.
11. Whalen, J. P., Evans, J. A., and Shanser, J. Vector principle in differential diagnosis of abdominal masses: left upper quadrant. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 104–118.

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ANGIOGRAPHY OF SPLENIC ABSCESSES*

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ANGIOGRAPHY has been sparsely applied to the diagnosis of splenic abscess. In 1964, Zatzkin and co-workers[10] reviewed the roentgenologic findings of splenic abscess: angiography was not included among the diagnostic procedures. Rösch,[8] in his 1967 monograph on roentgenology of the spleen and pancreas stated, "The arteriographic appearance of splenic abscess has not yet been described. . . ." Not until 1972 were the angiographic findings of splenic abscess, in 2 cases, initially reported.[2,4,6]

To some extent, the angiographic description is little known because abscess of the spleen is uncommon. Reid and Lang[5] reviewed data from several large series of postmortem examinations from the pre-antibiotic era and found the incidence of splenic abscess to be between 0.2 and 0.7 per cent. Recently, Altemeier *et al.*[1] reviewed their experience with 540 intra-abdominal abscesses and reported none occurring in the spleen. Nevertheless, it is our experience that patients with suspected inflammatory disease of the left upper quadrant of the abdomen are examined angiographically with disproportionate frequency. This is a reflection of the limitations of routine procedures in diagnosing abnormalities of the spleen and of the complicated milieu in which abscesses arise. In the last 2 years, we have studied 4 patients angiographically in whom splenic abscesses were subsequently proved surgically.

The purpose of this paper is to demonstrate thoroughly the angiographic appearance of splenic abscesses and to promote the use of angiography in preoperative evaluation.

REPORT OF CASES

CASE I. A 20 year old man sustained a gunshot wound to the abdomen resulting in multiple fistulae and abscesses. Despite several operations and intensive antibiotic therapy, his fever persisted. Physical examination and routine roentgenographic studies indicated a left subphrenic abscess. Abdominal angiography was performed for further evaluation of the left upper quadrant prior to surgical treatment.

A splenic arteriogram (Fig. 1*A*) demonstrated splenomegaly with displacement and stretching of splenic arterial branches around a 6.0 cm., avascular mass in the lower pole of the spleen. In the parenchymogram phase (Fig. 1*B*), the avascular space showed irregular margins. The venous phase was normal. No subphrenic space was seen. Splenectomy disclosed a 461 gm. spleen with a thick-walled abscess in the lower pole, 6.5 cm. in diameter.

CASE II. A 55 year old woman with a history of rheumatoid arthritis, Sjögren's syndrome, and Felty's syndrome was evaluated for persistent fever. She had taken steroids for 10 years. A Tc^{99m} sulfur colloid scintiphotogram of the liver and spleen showed splenomegaly and multiple focal splenic defects. The excretory urogram demonstrated a mass in the upper pole of the left kidney. The patient was thought to have widespread lymphoma.

Renal angiography revealed a pseudotumor of the left kidney. A splenic angiogram (Fig. 2*A*) showed massive splenomegaly with elongated, intrasplenic arteries. Two avascular zones measuring 2 to 4 cm. in diameter were also noted. The parenchymogram (Fig. 2*B*) showed the indistinct margins of the 2 masses. The venous phase was normal.

Splenectomy disclosed a 1,077 gm. spleen with 3 abscesses, 2 of which corresponded to the avascular zones demonstrated angiographically. Not seen angiographically was a 1 cm. abscess in the lower pole. Several small periph-

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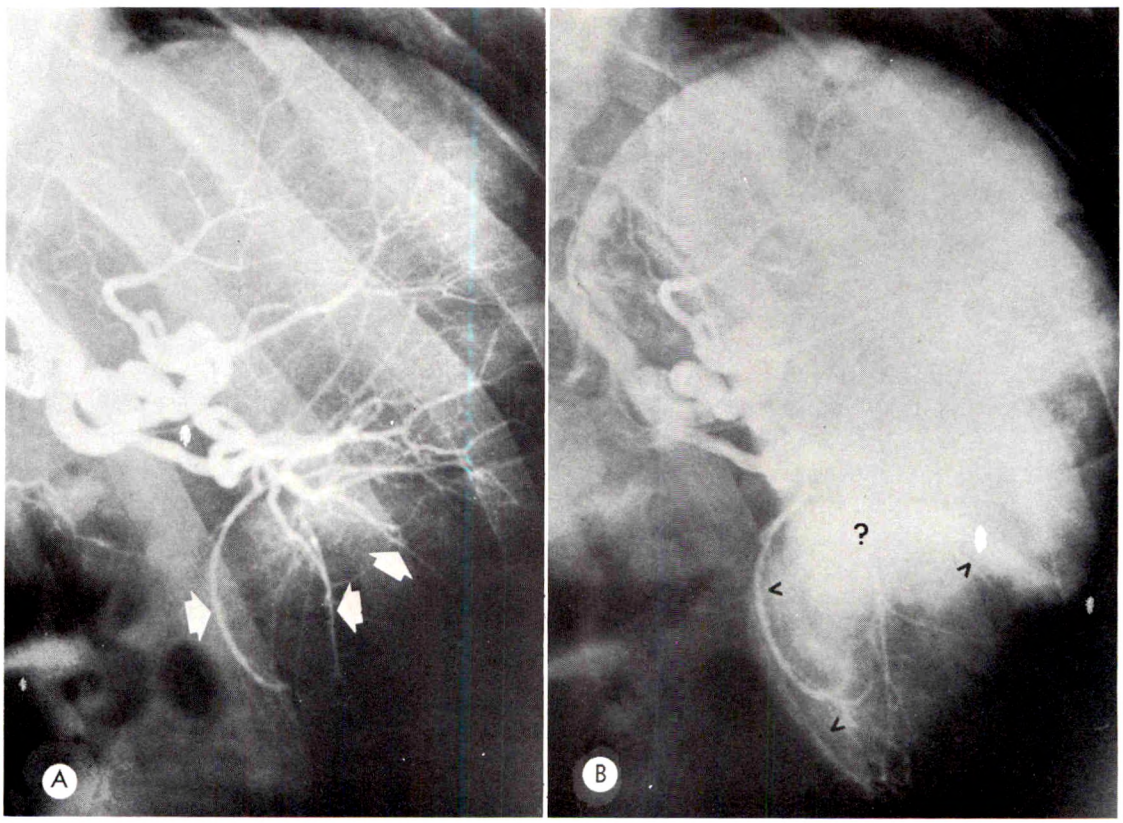


Fig. 1. Case 1. (*A*) Arterial phase of splenic arteriography showing stretched splenic branches in lower pole (arrows). (*B*) Late arterial early capillary phase showing avascular mass in lower pole with vague margins (arrowheads and question mark).

eral infarcts and diffuse extramedullary hematopoiesis were present.

Case III. A 32 year old male heroin addict was experiencing abdominal pain and fever. Physical examination and routine roentgenographic procedures indicated ascites, left pleural effusion, splenomegaly, and gastroesophageal varices. Paracentesis and thoracocentesis yielded sanguinous fluid, which was sterile. A Tc^{99m} sulfur colloid scintiphotogram of the liver and spleen confirmed the splenomegaly and revealed a splenic defect of the upper pole.

On the fifteenth hospital day, the patient developed high fever and acute pain in the left upper abdominal quadrant. For further evaluation of the left upper quadrant, angiography was performed (Fig. 3*A*), which confirmed splenomegaly and demonstrated a 7×10 mm. aneurysm of a splenic arterial branch of the upper pole. An avascular space (Fig. 3*B*) surrounded the aneurysm and nearby arteries were displaced. Splenic veins were intrinsically normal but drained to multiple collateral veins, reflecting the patient's known portal hypertension and hepatofugal blood flow.

A 589 gm. spleen was surgically removed. A 4×5 cm. necrotic cavity containing a mycotic aneurysm and several small abscesses replaced the upper pole.

Case IV. A 23 year old male heroin addict with a previous history of acute bacterial endocarditis was admitted to the hospital with shaking chills and pain in the left upper quadrant of the abdomen. A midabdominal mass was palpated. Blood cultures were positive for Group D streptococcus and *Streptococcus viridans*. A Tc^{99m} sulfur colloid scintiphotogram of the liver and spleen showed a marked decrease in spleen size compared with a similar study done 9 months before. Focal defects noted previously were no longer present. Routine roentgenograms of the abdomen revealed a

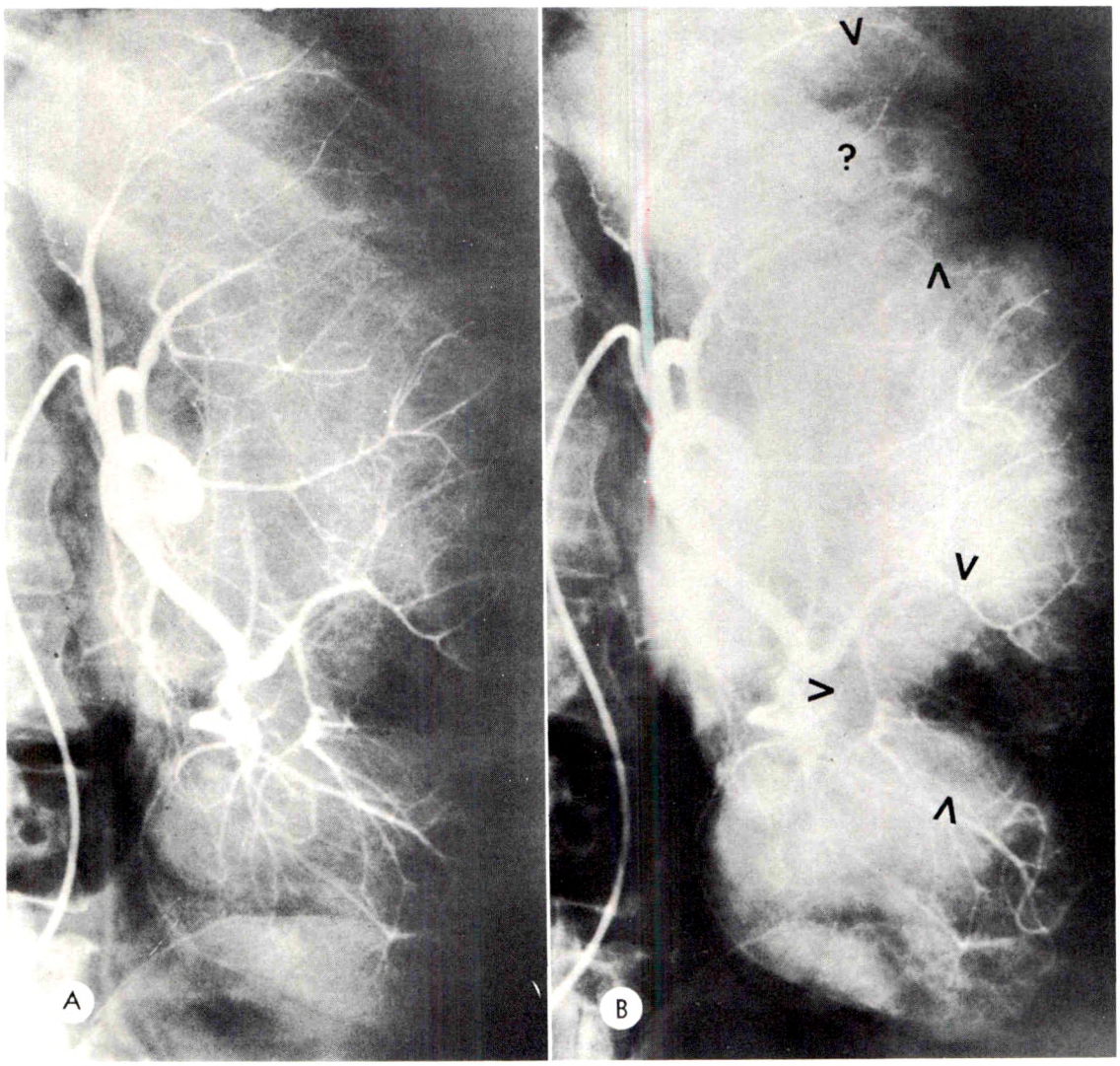


Fig. 2. Case 11. (*A*) Arterial phase of splenic arteriography showing splenomegaly (note relation to lumbar vertebrae and left iliac crest) and stretched branches throughout the spleen. (*B*) Late arterial, early venous phase shows 2 avascular zones with indistinct margins (arrowheads and question mark).

mass in the left upper quadrant.

Angiographic evaluation of the left upper quadrant mass showed the celiac axis to be compressed by the median arcuate ligament; an aneurysm of the superior mesenteric artery was also seen. Splenic arteriography (Fig. 4, *A* and *B*) demonstrated splenomegaly and a large, central, avascular, splenic mass with shaggy walls displacing intrasplenic branches. The venous phase was normal.

Subsequent laparotomy confirmed a mycotic aneurysm of the superior mesenteric artery. A large spleen was removed, which contained a central necrotic mass consisting of 700 ml. of pus.

DISCUSSION

The clinical findings are summarized in Table 1. All 4 patients had underlying abnormalities contributing to infection. None had evidence of splenic trauma, although splenic injury in Case 1 could not be absolutely excluded. None had evidence of extension of infection from contiguous structures (stomach, colon, left kidney).

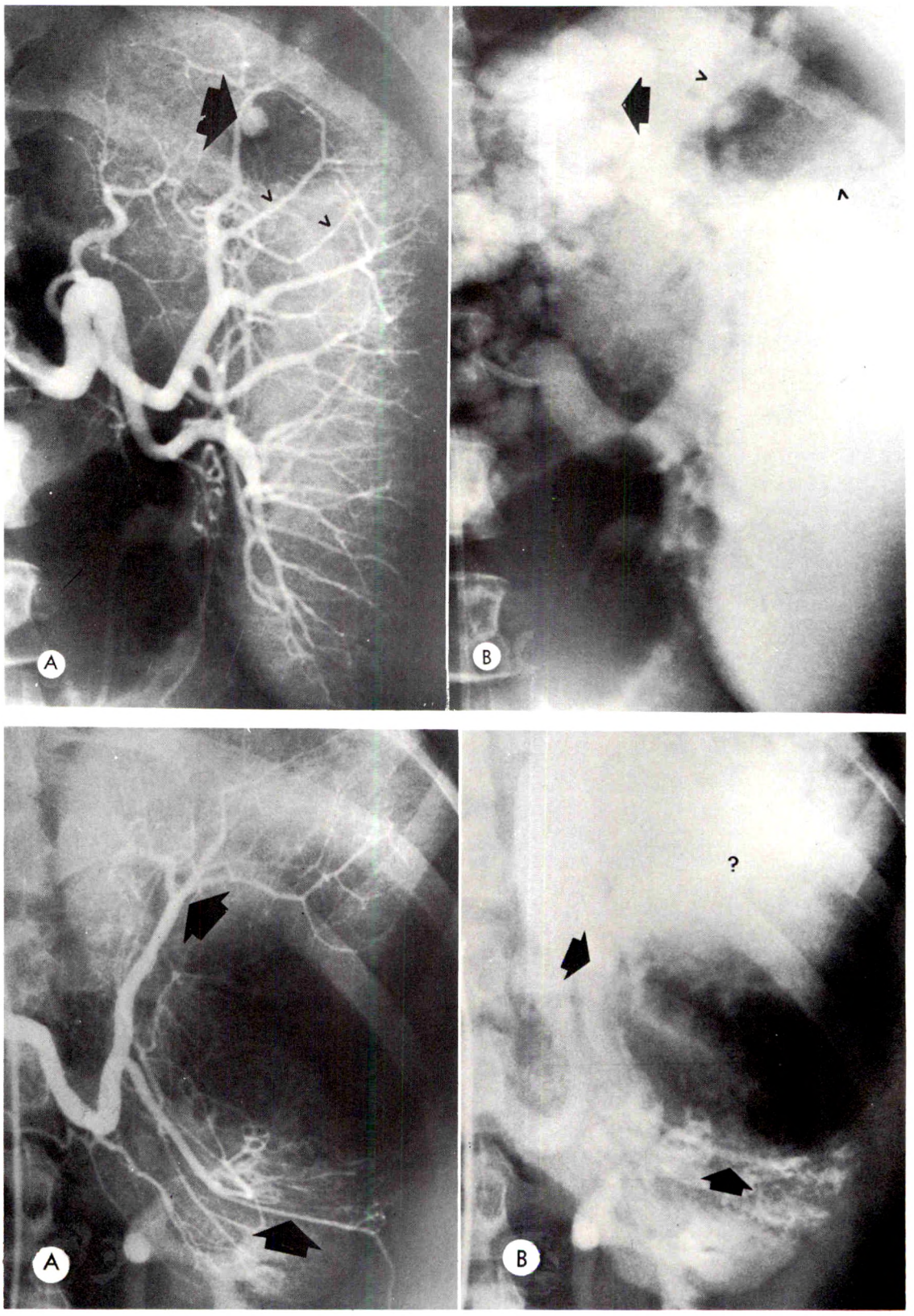

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Fig. 3. Case III. (*A*) Arterial phase of splenic angiography showing splenomegaly (note relative size of spleen to vertebral bodies), mycotic aneurysm (large arrow), and stretched branches (arrowheads). (*B*) Capillary, early venous phase showing avascular zone in upper pole (arrowheads) and large collateral venous channels (large arrow).

Table I

SUMMARY OF CLINICAL FINDINGS

Case	Age, Sex (yr.)	Predisposing Abnormalities	Major Signs and Symptoms	Clinical Diagnosis before Angiography	Organisms in Splenic Abscess
I	20, M	Gunshot wound of abdomen with fistulae and abscesses	Persistent fever of occult source	Left, subphrenic abscess	*Escherichia coli*, other undefined gram negative rods
II	55, F	Rheumatoid arthritis, Sjögren's syndrome, and Felty's syndrome—patient receiving corticosteroids	Persistent fever, splenomegaly	Lymphoma, lymphosarcoma	*Escherichia coli*, species of Aspergillus (seen microscopically but not cultured)
III	32, M	Heroin addict	Fever, bloody ascites, splenomegaly	Cirrhosis with portal hypertension, (?) splenic infarction	No growth (patient receiving antibiotics)
IV	23, F	Heroin addict, subacute bacterial endocarditis	Fever, abdominal mass	Mesenteric abscess, splenic abscess or infarction	*Streptococcus viridans* and Group D streptococcus

Hence, hematogeneous dissemination of bacteria to the spleen is believed to be the source of the splenic abscesses in our series.

The angiographic findings in our patients were remarkably similar (Table II). Splenomegaly and an irregular avascular mass within the spleen were invariably present; the borders of the mass were shaggy. Stretched splenic branches reflected the expansile nature of the splenic abscess. There was no rim of increased staining around the abscesses or hypervascularity.[6,7] No arterial encasement or pooling of contrast material was noted. Splenic veins were normal.

Although these findings are not diagnostic of splenic abscess, several deserve special attention. The irregular margins of the defects were evidence against the diagnosis of simple splenic cyst.[9] Hydatid cysts of the spleen have a rim of increased density during the parenchymal and venous phases of the angiogram; this was not seen in our reported abscesses.[7] We could not, however, distinguish the abscesses from large, acute hemorrhagic infarctions or avascular metastases to the spleen.[8]

SUMMARY

The criteria outlined and summarized in Table II show no single observation diagnostic of splenic abscess.

Fig. 4. Case IV. (*A*) Arterial phase of splenic angiography showing branches displaced and stretched (arrows). (*B*) Capillary phase showing avascular space with ill-defined (question mark) margins (arrows).

TABLE II

ANGIOGRAPHIC FINDINGS IN SPLENIC ABSCESSES

Case	Splenomegaly	Avascular Intrasplenic Masses	Abscess Margins	Abnormalities of Venous Phase	Accumulations of Contrast Medium or Abnormal Stain	Mycotic Aneurysm
I	Moderate (461 gm.)	One (6.0 cm. in diameter)	Ill-defined, shaggy	None	None	None
II	Severe (1,077 gm.)	Two (2–4 cm. in diameter)	Vague, irregular	None	None	None
III	Moderate (589 gm.)	One (4.5 cm. in diameter)	Vague, irregular	None	None	One, splenic (8×10 mm.)
IV	Severe (approximately 1,000 gm.)	One (9 cm. in diameter)	Vague, irregular	None	None	One (superior mesenteric artery)

This constellation of findings, however, in the context of a known abdominal inflammatory process, is quite helpful in differentiating a splenic abscess from other avascular intrasplenic masses. If the patient has an avascular, expansile, intrasplenic mass with irregular margins and documented bacterial seeding of the blood stream or a mycotic aneurysm, a splenic abscess should be the primary diagnostic consideration.

Moreover, it is apparent from our cases that angiography may be pivotal in the preoperative evaluation of such patients.

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REFERENCES

1. ALTEMEIER, W. A., CULBERTSON, W. R., FULLEN, W. D., and SHOOK, C. D. Intra-abdominal abscesses. *Am. J. Surg.*, 1973, *125*, 70–78.
2. BALTAXE, H. A., WATSON, R. C., and LEVIN, D. C. Angiographic appearance of splenic masses. *Angiology*, 1972, *23*, 316–328.
3. GOULD, H. R., CLEMETT, A. R., and ROSSI, P. Radiologic diagnosis of splenic metastasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 755–760.
4. LEPAGE, J. R., PRATT, A. D., JR., MIALE, A., and CALVERT, W. P. Diagnosis of splenic abscess by radionuclide scanning and selective arteriography. *J. Nuclear Med.*, 1972, *13*, 331–332.
5. REID, S. E., and LANG, S. J. Abscess of spleen. *Am. J. Surg.*, 1954, *88*, 912–917.
6. REUTER, S. R., and REDMAN, H. C. Gastrointestinal Angiography. W. B. Saunders Company, Philadelphia, 1972.
7. RIZK, G. K., TAYYARAH, K. A., and GHANDUR-MNAYMNEH, L. Angiographic changes in hydatid cysts of liver and spleen. *Radiology*, 1971, *99*, 303–309.
8. RÖSCH, J. Roentgenology of the Spleen and Pancreas. Charles C Thomas, Publisher, Springfield, Ill., 1967.
9. SHANSER, J. D., MOSS, A. A., CLARK, R. E., and PALUBINSKAS, A. J. Angiographic evaluation of cystic lesions of spleen. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *119*, 166–174.
10. ZATZKIN, H. R., DRAZAN, A. D., and IRWIN, G. A. Roentgenographic diagnosis of splenic abscess. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 896–899.

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EDITORIAL

IMMUNOLOGY AND RADIOLOGY

LIKE radiology, immunology had its beginnings in empirical procedures, and also like radiology, it has blossomed into a field which encompasses almost all of medicine. This efflorescence waited from the time of Jenner until means of identifying and quantitating the protein and glycoprotein fractions of the blood became possible and practical. In addition to the sophisticated physical, chemical, and isotope procedures used in investigation and diagnosis, other methods, such as electron microscopy, have become indispensable for evaluation of some of the diseases due to disturbance in the immune globulin fractions of the body fluids. For example, demonstration of lesions in the basement membrane of the renal glomerulus may be only adumbrated by light microscopy, but is clearly shown with an electron microscope. The significance of the whole field has been emphasized by the recent award of a Nobel prize in 1972 to Edelman and Porter for elucidating the peptide structure of the immunoglobulins.

Immunity is mediated by cellular or humoral mechanisms, and both are closely associated with circulating lymphocytes. Cellular immunity is produced through the effect of lymphocytes which have been processed by the thymus, T-lymphocytes; humoral immunity is produced by the effect of immune globulins, some of which are bound to the surface of lymphocytes which have not been so processed, B-lymphocytes, from bone marrow, and of the plasma cell series. Each lymphocyte stains for only one immunoglobulin class, and 5 different immunoglobulins have been isolated: IgG, IgA, IgM, IgD, and IgE. Each of these immunoglobulins is elaborated by a single clone of lymphoid cells, or of the plasma cells into which it has differentiated.

Each immunoglobulin molecule is made up of a large number of amino acids, arranged in 4 specific peptide chains, 2 quite large, called heavy chains, and 2 much shorter, called light chains. The heavy chains are principally responsible for the specific chemical, physical and immunologic properties of the immunoglobulin. There are thus 5 types of heavy chain, called gamma, alpha, mu, delta, and epsilon, appropriately and respectively. There are only 2 types of light chain. In multiple myeloma the 4 chains of the immunoglobulin molecule occasionally dissociate: the light chains are excreted through the kidneys, form casts in the tubules, and appear in the urine as the unusual substance recognized as Bence Jones protein since 1848.

The total amount of immunoglobulin in normal circulating blood varies considerably according to the type of globulin. About 10–20 per cent of circulating lymphocytes normally carry surface bound immunoglobulins, and of these, about two-thirds carry IgM, and one third IgG. Of the immunoglobulin in the serum, 80 per cent is IgG. IgA, on the other hand, is the dominant type in secretions, gastrointestinal, respiratory, and renal. Thus, different diseases lead to the appearance of abnormal proteins in different body fluids. Since the immunoglobulins are elaborated by cells of the plasma cell series, many of the disturbances in immunoglobulins are associated with abnormal numbers of these cells in the circulating blood or marrow. These abnormalities may be due to: deficiency; specific or monoclonal increase, involving only 1 type of Ig; or polyclonal, nonspecific

increase, involving several classes of Ig; and the increase may include normal or abnormal proteins.

Diseases associated with immunoglobulin deficiency are primary or secondary and the classification of the various forms is difficult and incomplete. In this issue of the JOURNAL, Dr. Marshak and his co-authors[1] discuss these deficiency problems in relation to the gastrointestinal tract.

The classical immunoglobulin disturbance is multiple myeloma, of the polyclonal type, with increase in IgG and IgA, usually.[2] While the proliferation of the plasma cell series leads to the characteristic bone changes and the anemia, it is the immunoglobulin itself which leads to the renal problems referred to. In addition to the proteinuria, there are more fundamental insults to the kidney. The antigen-antibody complex and associated complement fractions are precipitated in the basement membrane of the renal glomerulus,[3] interfering significantly with function. The extent of the precipitation cannot be determined by light microscopy, but requires electron microscopy for evaluation.[4] The danger of intravenous urography in the presence of multiple myeloma is well known, and was the subject of a previous editorial in these pages.[5]

Closely related to multiple myeloma is Waldenström's macroglobulinemia, in which there are abnormal amounts of IgM in the blood, produced by activated lymphocytes, immature forms, and plasmacytid lymphocytes. Some of the significant manifestations of the disease are due to the hyperviscosity of the blood due to the presence of the globulin. The hyperviscosity leads to hemostatic abnormalities: epistaxis, retinal hemorrhages, and gastrointestinal bleeding. Cellular proliferation leads to lymphoid organomegaly which is also a feature of this monoclonal abnormality.

Some immunoglobulins precipitate out of solution at temperatures below normal body temperature and are called cryoglobulins. This precipitation results in Raynaud's phenomenon in some patients, or in dependent purpura, or vasculitis, in others. Abnormal values of circulating immunoglobulins have been found in many diseases previously thought to be unrelated. Some of these are systemic lupus erythematosus, rheumatoid arthritis, Goodpasture's syndrome,[6] Sjögren's disease,[7] amyloidosis, sarcoidosis,[8] the Henoch-Schönlein syndrome,[9] and certain neoplasms, such as those of the colon, prostate, breast, and lung.[10] In many of these situations, it is only one of the classes of immunoglobulins which is involved.

Recently, since quantitative studies have become possible, and since detection of various subclasses of the 5 main classes have been identified, it appears that some diseases are manifestations of the presence of only fractions of the immunoglobulins, usually the heavy chain fraction. The presence of specific fractions is associated with various different clinical abnormalities. Thus, heavy chain disease of the gamma class is accompanied by lymphoproliferа-

[1] MARSHAK, R. H., HAZZI, C., LINDNER, A. E., and MAKLONSKY, D. Small bowel in immunoglobulin deficiency syndromes, AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1974, *122*, 227–240.

[2] OSSERMAN, E. F. Multiple Myeloma and Related Plasma Cell Dyscrasias in Immunologic Diseases. Edited by MAX SAMTER. Second edition. Little, Brown & Company, Boston, 1971, pp. 520–547.

[3] FEDERMAN, D. D. In case records of the Massachusetts General Hospital. *New England J. Med.*, 1974, *291*, 242–249.

[4] GUTMAN, R. A., STRIKER, G. E., GILLILAND, B. C., and CUTLER, R. E. The immune complex glomerulonephritis of bacterial endocarditis. *Medicine*, 1972, *51*, 1–25.

[5] LEUCUTIA, T. Multiple myeloma and intravenous pyelography. Editorial. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *85*, 187–189.

[6] SCHEER, R. L., and GROSSMAN, M. A. Immune aspects of the glomerulonephritis associated with pulmonary hemorrhage. *Ann. Int. Med.*, 1964, *60*, 1009–1021.

[7] VAN BOXEL, J. A., HARDIN, J. A., GREEN, I., and PAUL, W. E. Multiple heavy-chain determinants on individual B lymphocytes in the peripheral blood of patients with Sjögren's syndrome. *New England J. Med.*, 1973, *289*, 823–827.

[8] VAGAČ, M., BADALÍK, L., VIRSIK, K., ROVENSKÝ, J., and ŽITŇAN, D. Die Niveaus der Immunoglobuline bei Sarkoidose. *Wien. klin. Wchnschr.*, 1974, *86*, 333–335.

[9] EVANS, D. J., WILLIAMS, D. G., PETERS, D. K., SISSONS, J. G. P., BOULTON-JONES, J. M., OGG, C. S., CAMERON, J. S., and HOFEBRAND, B. I. Glomerular deposition of properidin in Henoch-Schönlein syndrome and idiopathic focal glomerulonephritis. *Brit. M. J.*, 1973, *3*, 326–328.

[10] SOLOMON, A., and MCLAUGHLIN, C. L. Immunoglobulin disturbances and their clinical significance. *Med. Clin. North America*, 1973, *57*, 499–516.

tive disorders, with enlargement of the spleen, liver, and lymph nodes, and with edema of the uvula and palate. Alpha heavy chain disease is characterized by malabsorption, chronic diarrhea, steatorrhea, weight loss, and invasion of the small intestine with mature plasma cells and lymphocytes. The disease may go on to involve the respiratory tract similarly. This constellation is no doubt associated with the overwhelming presence of IgA (and its component alpha heavy chains). This type of globulin is found in the gastrointestinal and respiratory tracts, rather than in serum. One patient with this clinical picture, including diffuse lymphomatosis of the gastrointestinal tract, has been found to produce immunoglobulin principally a subclass of IgG, rather than IgA.[11] In chronic lymphatic leukemia there is an excess of the mu heavy chains. Staining of the cells has shown that some of the immunoglobulin is surface bound to the lymphocytes in chronic lymphatic leukemia, while in acute lymphoblastic leukemia no surface bound immunoglobulin has been found.[12] This dissimilarity suggests that chronic leukemia occurs among the B-cell lymphocyte population and acute leukemia occurs in the T-cell population.

Further investigation of immunoglobulins will probably have additional practical importance from an entirely different point of view. In some patients with abnormal circulating immunoglobulins, who had been followed over a period of many years, multiple myeloma developed only after an interval as long as 13 years.[12] Furthermore, close monitoring of the Ig blood levels in patients with known multiple myeloma has led to the ability to predict relapses in some cases.[10] Since elevation of the abnormal globulin level precedes clinical evidence of disease by many days, a relapse can be prevented by appropriate treatment given on the basis of the Ig blood levels before there are any other indications of the imminent relapse. Also, it has been noted that in those malignant lymphomas in which the patient presents an excess of gamma heavy chains, bone involvement does not occur as a complication,[13] eliminating the need to search for this danger.

Work of this type is being carried out in many centers, in many countries. Recently, the burgeonning information has required standardization, because of its quantity, and a uniform terminology is being developed through the World Health Organization. The vital significance of this field in the question of organ transplantation has not even been touched on here, but its significance is obvious. The immune mechanism is a most sensitive means for the body to recognize environment, as exemplified in foreign protein, and to differentiate this from native protein. Recently Sir Peter Medawar aptly characterized the whole field of immunology as the "biology of self recognition."[14] We anticipate that from this field there will be more and more information which will become of significance for the radiologist.

E. Frederick Lang, M.D.

[11] Kopeć, M., Świerczyńska, Z., Pazdur, J., Luft, S., Placheka, M., Dabska, M., Glinska, D., and Wozniczko-Orlowska, G. Diffuse lymphoma of the intestines with a monoclonal gammopathy of the IgG, Kappa type. *Am. J. Med.*, 1974, *56*, 381–385.

[12] Natvig, J. R., and Kunkel, H. G. Human Immunoglobulins: Classes, Subclasses, Genetic Variants, and Idiotypes. In: Advances in Immunology. Edited by F. J. Dixon, and H. G. Kunkel. Academic Press, New York, 1973, pp. 1–59.

[13] Ellman, L. L., and Bloch, K. J. Heavy chain disease. *New England J. Med.*, 1968, *278*, 1195–1201.

[14] Symposium. Clinical Immunology and the Physician. *Proc. Roy. Soc. Med.*, 1974, *67*, 525–580.

NEWS ITEMS

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS (ASTR)

The 16th Annual Meeting of the American Society of Therapeutic Radiologists will be held at Sonesta Beach Hotel, Key Biscayne, Florida from Thursday, October 31, to Sunday, November 3, 1974.

A very elaborate program has been arranged. This includes: Residents Radiation Biology Breakfast Seminars on Thursday, Friday and Saturday, and ASTR Members Breakfast Business Meetings on Thursday, Friday, and Saturday.

There will be 2 Panel Discussions on: (1) "Carcinoma of the Ovary," Thursday; and (2) "Radiation Effects in Critical Dose-Limiting Tissues," Sunday; and 2 Summary Lectures on; (1) Ongoing Clinical Trials Involving Radiation Therapy, Friday; and (2) The Pathology of Tumors of the Central Nervous System, Saturday.

Twenty-four proffered papers have been scheduled for Thursday and Friday forenoon. On Saturday, 2 parallel Sessions will be held: a Session of 14 proffered papers on "Radiation Therapy and Allied Fields;" and a second Session of 15 proffered papers on "Clinical and Research Radiation Biology." The Sunday morning Session will be devoted to 7 proffered papers on Radiation Therapy of more recent interest and at the end to 2 papers of "Summary of Saturday's Session on Clinical and Research Radiation Biology."

A most attractive Social Program has been arranged for the Ladies. This consists of: 1. Visit to Villa Viscaya and Matinée Musicale on Thursday morning, and Poolside Cocktail Party in the evening; 2. Biscayne Bay By Boat, on Friday morning; 3. Seaquarium Tour on Saturday morning and the President's Cocktail party and a splendid banquet followed by dancing on Saturday evening.

Technical Workshops will be held Thursday, Friday and Saturday afternoons from 3:00 P.M.–5:00 P.M. The discussions will include: simulators; computers; dosimetry and quality control; linear accelerators; and treatment aids, comprising 26 items altogether.

Arrangements have also been completed for the 1975 meeting which will be held at the Hyatt Regency, San Francisco, California, in the Embarcadero Center, October 8–12. The 1976 Meeting is scheduled to be held at Hyatt House in Atlanta, Georgia.

TRENDS IN CHILDHOOD CANCER

The Division of Oncology of the Children's Hospital of Philadelphia, and the Department of Radiation Therapy of the American Oncologic Hospital of the Fox Chase Cancer Center will hold a symposium titled "Trends in Childhood Cancer" on November 15 and 16, 1974.

The course is approved by the A.M.A. for the Physician's Recognition Award.

For information, please contact H. Gunter Seydel, M.D., M.S., Chief, Department of Radiation Therapy, American Oncologic Hospital, Central and Shelmire Avenues, Philadelphia, Pennsylvania 19111.

18th ANNUAL POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

University of California School of Medicine, San Francisco, California

The Department of Radiology and Extended Programs in Medical Education will hold this Course at the Fairmont Hotel, San Francisco, February 17–21, 1975.

The course is designed to provide a survey of selected subdivisions of diagnostic radiology for the radiologist in clinical practice. The faculty of the Course is comprised of the Faculty of the Department of Radiology, University of California School of Medicine, San Francisco and an international Guest Faculty.

All registrations must be made in ad-

vance as attendance is limited. No daily registration is available.

Detailed information regarding the daily speakers and schedules will be available at a later date.

Please mail application to: Extended Programs in Medical Education, Room 575-U, University of California, San Francisco, California 94143.

WEST OF SCOTLAND COMMITTEE FOR POSTGRADUATE MEDICAL EDUCATION AND GLASGOW INSTITUTE OF RADIOTHERAPEUTICS

An intensive 5 day course suitable for candidates preparing for the F.F.R. Part II Examination will be held in the Glasgow Institute of Radiotherapeutics (Western Infirmary and Belvidere Hospital), March 3–7, 1975.

The program will include tutorial sessions, case presentations, and discussions of topics related to recent advances. Meetings will be held daily from 9:15 A.M. to 4:30 P.M., beginning at 9:15 A.M. on Monday March 3rd in the Radiotherapy Department Lecture Theatre, Western Infirmary.

Applications should be made to the Dean of Postgraduate Medicine, The University of Glasgow, Glasgow G12 8QQ, Scotland.

RADIOLOGY OF THE GASTROINTESTINAL TRACT

Tutorial Postgraduate Course

The Department of Radiology, Duke University Medical Center, will hold a Tutorial Postgraduate Course at the Governors Inn, Research Triangle Park, N.C.

This course is awarded 28 hours A.M.A. Category 1 accreditation.

The course is designed for radiologists, but open to other physicians whether in training or practice. The emphasis will be on personalized, tutorial type teaching of basic subjects on the gastrointestinal tract by recognized authorities using their original roentgenograms with ample opportunity for discussion. The subject matter will cover all facets of gastrointestinal disease including comprehensive coverage of diagnostic techniques. A detailed abstract book with references will be provided.

The Program Chairman is Richard G. Lester, M.D., Professor and Chairman of Radiology, Duke University Medical Center.

For further information please contact, Robert McLelland, M.D., Department of Radiology, Box 2808, Duke University Medical Center, Durham, North Carolina 27710.

THIRD INTERNATIONAL SYMPOSIUM ON DETECTION AND PREVENTION OF CANCER

This is the first announcement of the Third International Symposium on Detection and Prevention of Cancer. The Second Symposium was held in Bologna, Italy, April, 1973. The Third Symposium is now scheduled in New York City, at the Americana Hotel, April 26 to May 1, 1976.

The format of the Symposium includes 12 Conferences on general topics related to cancer detection and prevention and 18 Panels on specific and organ-oriented subjects. Approximately 22 Symposia will be arranged by groupings of proffered papers. There will be 14 Short Courses and 20 Practical Workshops. Also, daily consultation sessions with experts will be offered. Provisions are made for ample discussion at all sessions.

Further information may be obtained from Herbert E. Nieburgs, M.D., Secretary General, Mount Sinai School of Medicine of the City University of New York, Fifth Avenue and 100th Street, New York, New York 10029.

BOOK REVIEWS

ANGIOGRAPHY IN INFANTS AND CHILDREN. Edited by Michael T. Gyepes, M.D. Cloth. Pp. 378, with many illustrations. Price, $24.75. Grune & Stratton, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1974.

Dr. Gyepes and his co-contributors have produced a well balanced résumé of what is available in the field of pediatric angiography. In addition, angiography is projected in proper perspective and is not oversold. Over-all, balance of presentation is reasonably good, although certain sections are better than others. Because the volume is not a textbook, there is a lack of in-depth coverage of some of the diseases illustrated, but I am sure that such in-depth coverage was not the prime objective in this work.

The illustrations are generally of good caliber and case material selection is excellent. If one were to do nothing more than look at the illustrations and read the legends, one would gain considerable benefit from this volume. A few of the illustrations are underlabeled, and for those who might not be well versed in angiographic anatomy this does pose a minor problem.

The technical aspects of performing angiography are amply considered in the various chapters, and for anyone desiring to perform angiography in the pediatric age group, considerable helpful information regarding technique and complications is available. In addition, a number of practical tips derived from experience only come through in the various sections.

For general radiologists, and especially for residents in training, this book will provide an atlas-like review of angiographically demonstrable pathology in the pediatric age group, and at the same time will help to project the entire field of pediatric angiography into proper focus.

LEONARD E. SWISCHUK, M.D.

CLINICAL CARDIAC RADIOLOGY. By Keith Jefferson, M.A., F.R.C.P., F.F.R., Consultant Radiologist, National Heart Hospital and St. George's Hospital, London; Lecturer, Cardiothoracic Institute, University of London; and Simon Reese, M.A., M.R.C.P., F.F.R., Consultant Radiologist, National Heart Hospital and St. Bartholomew's Hospital, London; Lecturer, Cardiothoracic Institute, University of London, England. Cloth. Pp. 314, with many illustrations. Price, $57.75. Butterworths, 2265 Midland Avenue, Scarborough, Ontario, Canada M1P 4S1, 1973.

This is a book devoted to the radiology of acquired and congenital heart disease. The authors undertook an ambitious task resulting in a work of 314 pages, 30 chapters, and 377 illustrations.

The book is divided into 2 major parts. The first discusses in general terms the radiology of the heart, describing the anatomy and some physiology. The second part is designed to arrive at a more specific diagnosis. There are 4 sections in this latter part which are: shunts without cyanosis; shunts with cyanosis; obstruction and regurgitation; myocardial and pericardial disease.

The introduction to the 30 chapters is preceded by a concise table, which indicates how to use the book, and which will help the reader to interpret a roentgenogram of a patient with a cardiac problem. In this table are described all the salient radiographic and clinical features of the most important lesions.

At the end of the book the authors give information about the radiographic techniques used in their institution and a complete description of their clinical material.

Finally, they produce a computer analysis of the clinical material in which they correlated the radiographic findings with catheterization data. This information is useful and adds weight to the value of the text.

The authors achieved a fine task in presenting in a succinct form a very vast topic. They stressed the plain film roentgenographic analysis over the angiographic findings; yet surprisingly they have completely omitted the role of oblique roentgenograms. Some of the chapters are unique: thus, the section devoted to "the other systems in heart disease" (the bony thorax, inherited and allied syndromes with heart disease—general disease and the heart) is outstanding.

The authors discuss all the congenital heart lesions with authority and based upon their own vast experience. The organization of the book and the style are quite excellent.

On the negative side, it is unfortunate that in the first part, chapters dealing specifically with the anatomy, the physiology and the embryology were omitted. Throughout the text it is assumed that the reader has command of this basic knowledge. Terms and concepts are introduced, the explanations of which cannot be found in this book (example: endocardial cushions). No attempt was made to introduce the reader to angiocardiography, since as stated before, the purpose of the book is to describe plain film roentgenographic findings. The discussions of the lesions are simply descriptive, without enough stress on the physiologic reasons behind the findings. Some clinical entities such as relapsing polychondritis and its associated cardiac lesions, aortic left ventricular tunnel, and others, have been completely omitted. Furthermore, it is obvious that the authors have much greater experience in congenital, than in acquired heart disease and the sections dealing with these acquired lesions suffer from it. The bibliography is far from being complete.

In spite of the above objections, this reviewer wishes to congratulate the authors for a fine accomplishment. It is obvious, when one reads this textbook, that Drs. Jefferson and Reese did not copy someone else's work, but related their personal experience. Certainly, at any stage of development, a reader can benefit from such an experience.

HAROLD A. BALTAXE, M.D.

BOOKS RECEIVED

ATLAS OF VERTEBRAL ANGIOGRAPHY. By Mutsumasa Takahashi, M.D., Professor and Director, Department of Radiology, Akita University School of Medicine, Akita, Japan. Cloth. Pp. 384, with many illustrations. Price, $65.00. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1974.

ROENTGEN EXAMINATIONS IN ACUTE ABDOMINAL DISEASES. Third edition. By J. Frimann-Dahl, M.D., Ph.D., Professor of Radiology (Emeritus), Ullevål Hospital, Oslo, Norway. Cloth. Pp. 617, with many illustrations. Price, $29.50. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

PATIENT CARE AND SPECIAL PROCEDURES IN RADIOLOGIC TECHNOLOGY. Fourth edition. By John C. Watson, R.T., Director of Technology, the University of Connecticut Medical Center; formerly, Radiologic Administrator, University of California Medical Center, San Francisco; and Technical Consultant to the Yale University School of Medicine. Cloth. Pp. 200, with 114 illustrations. Price, $10.50. C. V. Mosby Company, 3301 Washington Boulevard, St. Louis, Mo. 63103, 1974.

NUCLEAR HEPATOLOGY: CLINICAL AND PHYSIOLOGICAL ASPECTS OF LIVER DISEASE BY RADIOISOTOPES. By Masahiro Iio, M.D., Director, Department of Nuclear Medicine and Radiological Science, Tokyo Metropolitan Geriatric Medical Center, Tokyo; Instructor, Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Tokyo; Hideo Yamada, M.D., Chief, 1st Division, Department of Nuclear Medicine and Radiological Science, Tokyo Metropolitan Geriatric Medical Center, Tokyo; Kenichi Kitani, M.D., Chief, 1st Division of Clinical Physiology, Department of Physiology, Tokyo Metropolitan Institute of Gerontology, Tokyo; and Yasuhito Sasaki, M.D., Chief, Section of Nuclear Medicine, 2nd Department of Internal Medicine, Faculty of Medicine, University of Tokyo, Tokyo, Japan. Cloth. Pp. 164, with many figures. Price, $23.50. Igaku Shoin Ltd., Tokyo. Exclusive U. S. agents, The Williams & Wilkins Company, 428 East Preston Street, Baltimore, Md. 21202, 1973.

GASTRIC IRRADIATION IN PEPTIC ULCER. Edited by Walter L. Palmer. Cloth. Pp. 161, with some figures. Price, $7.50. University of Chicago Press, 5801 Ellis Avenue, Chicago, Ill. 60637, 1974.

HUMAN MELANIN. By Eugene Follmann, Director of Investigative Laboratory of Clinic of Dermatology, University of Buenos Aires, Argentina. Cloth. Pp. 250, with 103 illustrations. Price, $12.00. Prof. Dr. Eugene Follmann, Lacarra 1513, Buenos Aires, Sec. 7, Rep. Argentina, 1973.

LUPUS ERYTHEMATOSUS: A REVIEW OF THE CURRENT STATUS OF DISCOID AND SYSTEMIC LUPUS ERYTHEMATOSUS AND THEIR VARIANTS. Second edition. Edited by Edmund L. Dubois, M.D., Associate Clinical Professor of Medicine, Clinical Immunology and Rheumatic Disease Section, Department of Medicine, University of Southern California School of Medicine; Director of the "Collagen Disease" Clinic, Los Angeles County/University of Southern California Medical Center, Los Angeles, Calif. Cloth. Pp. 798, with many illustrations. Price, $36.00 plus $1.50 shipping in the U.S. University of Southern California Press, Los Angeles, Calif. 90007, 1974.

HYSTEROSALPINGOGRAPHY. Second edition. By Alvin M. Siegler, M.D., D.Sc., Clinical Professor of Obstetrics and Gynecology, State University of New York Downstate Medical Center, Brooklyn, N. Y. Cloth. Pp. 218, with many illustrations. Price, $25.00. Medcom, Inc., 2 Hammarskjöld Plaza, New York, N. Y. 10017, 1974.

Computers in Radiotherapy—Physical Aspects. By Raymond G. Wood, M.Sc., F.Inst.P., C.Eng., M.I.E.E., Lately Chief Physicist, South Wales and Monmouthshire Radiotherapy Service and University Hospital of Wales (Cardiff). Paper. Pp. 112, with some figures. Price, £2.45. Computers in Medicine Series, General Editor, D. W. Hill. The Butterworth Group, 88 Kingsway, London, WC2B 6AB, England, 1974.

Gonadal Shielding in Diagnostic Radiology. DHEW Publication (FDA) 74-8028. Paper. Pp. 18, with a few illustrations. Prepared by the Radiological Health Sciences Education Project, University of California, San Francisco, under contract FDA 72-13 with the Bureau of Radiological Health, Food and Drug Administration, Rockville, Md. 20852, 1974.

Technetium Atlas. Produced in co-operation with the Isotope Laboratory, Faculty of Medicine, State University of Groningen, Holland; Director, Prof. dr. M. G. Woldring. Cloth. Pp. 46, with many illustrations. Published by Philips-Duphar B.V. Cyclotron and Isotope Laboratories, Petten, Holland, 1973.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Hilton Hotel, San Francisco, Calif., September 24–27, 1974.

American Radium Society
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Atlantic City, N. J., June 15–19, 1975.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following city during this year: Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Written examinations are scheduled in 13 large centers in June 1975.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1975 was September 30, 1974.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

American Association of Physicists in Medicine
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D.C. 20010. Annual Meeting.

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 31–Nov. 3, 1974.

American Nuclear Society
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545. Winter Meeting: Washington, D.C., Oct. 27–Nov. 1, 1974.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil in 1977.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Univ. of South Florida, Tampa, Fla.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

Third Congress of the European Association of Radiology
Secretary General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland, Meeting: June 22–27, 1975.

Second Asian and Oceanian Congress of Radiology
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters 2161 Taft Ave., Manila, Philippines.
Meeting: Manila, Philippines, Nov. 10–14, 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Osteopathic College of Radiology
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Mark M. Mishkin. The Graduate Hosp., Univ. of Pennsylvania, Philadelphia, Pa. 23rd Annual Meeting: San Diego, Calif., May 4–6, 1975.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Philip R. Shalen, Maj. MC, Departm. of the Army, Departm. of Radiology, US Army General Hosp., Landstuhl, APO New York 09180.

Blockley Radiological Society
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Albert A. Franco, 45 Alexander St., Lockport, N. Y. 14094. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, Grant Hosp., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. George N. Weston, 1515 Ygnacio Valley Rd., Walnut Creek, Calif. 94598. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Rodger W. Lambie, 701 E. 63rd St., Ste. 108, Kansas City, Mo. 64110.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Robert F. Conti, Holy Cross Hosp., Fort Lauderdale, Fla. 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, 716 Carrolton Blvd., West Lafayette, Ind. 47906.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce Saxe, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Isaac Sanders, 1429 Valley View Rd. 13, Glendale, Calif. 91202. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bdg. Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 16, 1975.

NEW ENGLAND SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

NORTH AMERICAN SOCIETY OF CARDIAC RADIOLOGY
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Pablo Ramos, Box 1618, Bismarck, N. D. 58501. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 5301 F Street, Sacramento, Calif. 95819. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 175 S. Merkle Rd., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 p.m., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1975.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 p.m.

San Diego Radiological Society
President, Dr. Stanley G. Seat, 476 Prospect St., LaJolla, Calif. 92037. Meets the first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 p.m.

Section of Radiology, National Medical Association
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 p.m., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Hilton Hotel, San Francisco, Calif., September 22–23, 1974.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting: St. Francis Hotel, San Francisco, Calif., May 28–31, 1975.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. James O. Richardson, Jr., 280 Mamaronek Ave., White Plains, N. Y. 10605. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociacón de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 335, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1 Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St.

Catherine St. West, Montreal 107, Que., Canada. Annual Meeting: Toronto, Ont., May 11–16, 1975.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincoln's Inn Fields, London WC2A 3PN, England.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting: Madrid, Apr. 24–26, 1975.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14–1151, Teheran, Iran. The Society meets on the second Saturday of each month. 5th Iranian Congress of Radiology, Apr. 12–17, 1975. Deadline for acceptance of papers, Jan. 20, 1975.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel. Nineteenth National Radiology Congress: Tel-Aviv, Israel, Nov. 6–8, 1974.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
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Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Neck and Chest

Salisachs, L. Gubern, Estrany, M. Carbonell, and Pi, L. Gubern. Las hernias pulmonares. (Pulmonary hernias.) *Anales de Med.*, Dec., 1973, *59*, 1239–1251. (From: Servicio de Neumatología y Pediatría, Servicio de Traumatología, Ortopedia y Cirugía Infantil, Instituto Corachán, Barcelona, Spain.)

The authors report 3 cases of pulmonary herniation in pediatric patients (ages 1½ months, 6 years, and 10 years).

Hernia of the lung through the chest wall is a rare condition. The lung can protrude through "weak" areas in the muscles or fascia or through true abnormal defects in the chest wall (chondrocostal aplasia).

The herniated lung presents as a chest wall mass, that increases in size during coughing or straining, and can be reduced with gentle pressure. The usual sites are the supraclavicular area, the parasternal area, and the paravertebral area.

If the hernia is related to a large chest wall defect, it can be accompanied by paradoxical movements during respiration, as well as a mediastinal shift and thus produce respiratory distress.

This condition can be associated with other congenital malformations (myelomeningocele, heart defects, diaphragmatic eventration, etc.).

The roentgenographic study will be useful in showing the rib anomalies, and possible associated malformations (a tangential view of the area of interest could conceivably demonstrate the herniated lung itself).

Treatment is surgical and should be done as soon as the diagnosis is established, particularly if respiratory distress is present.—*Julian B. Alvarez, M.D.*

Abdomen

Birnholz, Jason C., Ferrucci, Joseph T., Jr., and Wyman, Stanley M. Roentgen features of dysphagia aortica. *Radiology*, April, 1974, *111*, 93–96. (Address: Joseph T. Ferrucci, Jr., M.D., Department of Radiology, Massachusetts General Hospital, Boston, Mass. 02114.)

An aortic impression upon the distal esophagus on barium examination is a frequent finding in elderly patients and is generally considered an incidental senescent variation; however, 30 elderly patients with dysphagia were found to have extrinsic compression of the distal esophagus by the descending thoracic aorta when studied at the Massachusetts General Hospital. Each patient was unable to swallow solid foods without a feeling of substernal fullness, sticking, and regurgitation. The average duration of dysphagia was 14 months (range 7 months to 6 years). All but 1 patient admitted a weight loss, generally about 9 kilograms (20 pounds).

Constant roentgenographic features included: (1) ectasia of the descending portion of the aorta; (2) partial distal esophageal obstruction; (3) a smooth, flattened contour along the left lateral margin of the esophageal barium column; and (4) pulsatile movement of the barium bolus synchronous with aortic pulsation.

The study concluded that a sclerotic distal aorta is a frequent, although relatively unrecognized, cause of dysphagia in the older age groups. Women were affected predominantly more than men. Female predilection is attributed to the greater likelihood that dorsal kyphosis will develop in postmenopausal women, allowing the mid-esophagus to fall backward into the posterior thorax and causing the distal esophagus to approach the diaphragmatic hiatus obliquely. Since the lower esophagus is fixed from behind by the rigid aorta, it becomes sharply angulated as it traverses the hiatus. In most instances, the fluoroscopic appearance during esophagography will exclude the possibility of neoplastic disease and permit differentiation from achalasia, although submucosal infiltration by a malignant lesion arising in the gastric fundus could mimic this appearance. The so-called "dysphagia-aortica" can be managed conservatively since the degree of esophageal compromise progresses slowly. Forced dilatation of the lower esophagus is frequently useless and may result in significant morbidity.—*David E. Marley, Jr., M.D.*

Cubillos, Lorenzo, Croxatto, Hector, Pizarro, Ismael, and Geisse, Guillermo. Ulcera duodenal postbulbar. (Postbulbar duodenal ulcer.) *Prensa méd. argent.*, June, 1973, *60*, 606–619. (From: Unidad de Gastroenterología, Hospital Clínico de la Universidad Católica de Chile.)

The postapical portion of the duodenum is that area between the apex of the duodenal bulb and the ampulla of Vater.

Ulcerations in this area are not common, but probably they occur more frequently than believed. Usually they are located in the postero-infero-medial aspect, closely related to the head of the pancreas, and gastroduodenal artery (thus explaining the frequency of severe bleeding). They occur more frequently in males over 50 years old, and they can be single or multiple. They are difficult to demonstrate roentgenologically, although constant awareness of it would improve the number of positive examinations. Treatment is surgical, preferably electively, rather than during an acute hemorrhagic episode.

The authors review 54 cases: 5 discovered at autopsy (death unrelated to the ulcer); 9 cases proven at surgery (6 had preoperative roentgenologic study); and in only 1 case was the postbulbar ulcer demonstrated.—*Julian B. Alvarez, M.D.*

Joffe, Norman. Medial cecal defect associated with metastatic pancreatic carcinoma. *Radiology*, May, 1974, *111*, 297–300. (Address: Department of Radiology, Beth Israel Hospital, 330 Brookline Avenue, Boston, Mass. 02215.)

Involvement of the bowel by metastatic carcinoma is not uncommon, especially in patients with a primary malignancy in the breast, lungs, pancreas, stomach, ovary or uterus. Spread is usually by direct invasion from a contiguous primary neoplasm, or from a remote lesion by way of the mesenteric reflections, intraperitoneal seeding, lymphatic permeation, or true hematogeneous emboli. The author discusses a relatively common sign secondary to metastatic pancreatic carcinoma, which may be the initial clue or may represent the only roentgen evidence of spread from a pancreatic neoplasm. This sign consists of a relatively discrete extrinsic pressure defect on the medial aspect of the cecum.

The roentgenologic findings in 7 patients with this cecal deformity associated with metastatic pancreatic carcinoma are presented. Barium studies performed on 6 of these patients all showed a similar roentgenologic abnormality consisting of a localized defect on the medial aspect of the cecum below the ileocecal junction. The mucosa of the cecum was intact and the appearances were essentially those of an extrinsic pressure defect, or less commonly, an extramucosal intramural lesion. Barium studies were not performed on the 7th patient who presented with symptoms of acute intestinal obstruction, but the surgical findings indicated that a similar deformity of the cecum would almost certainly have been demonstrated. The surgical or autopsy findings in all 7 of these cases confirmed the presence of a primary carcinoma of the pancreas with a prominent metastatic mass in the right lower quadrant adherent to the medial portion of the cecum. The mode of spread is thought to occur either by way of the mesenteric reflections or by means of intraperitoneal seeding with the metastatic focus found in these patients to be located in the region of the inferior ileocecal fossa which is situated adjacent to the angle of junction of the ileum and cecum.

The differential diagnosis of a mass in this location includes: appendiceal abscess; mucocele; carcinoma; intussusception; Crohn's disease involving the terminal ileum; carcinoid; lipoma; and endometriosis. Other primary malignancies which may metastasize to this region include the ovary, colon, and stomach. —*Donald N. Dysart, M.D.*

Fani, C., and Puntillo, F. Addome acuto da tumori solidi benigni del mesentere. (Acute abdomen from benign solid tumors of the mesentery.) *Ann. ital. chir.*, 1974, *47*, 235–250. (From: Pio Istituto di S. Spirito ed Ospedali Riuniti di Roma, Italy.)

Two cases are reported in which a fibroma of the ileocecal region and a lipoma of the mesentery of the mid-ileum presented with a symptomatology of acute abdomen.

Roentgen studies were performed only in the patient with the lipoma, in whom an abdominal survey demonstrated a typical pattern of small bowel obstruction and an empty space in the mid-abdomen surrounded by the distended loops of small intestine. This particular roentgen finding known also in the French literature as the "vide radiographique" would suggest the presence of a benign cystic or tumoral lesion of the mesentery.

Reviewed and discussed are the pathologic, clinical and roentgen findings of the benign solid tumors of the mesentery.—*A. F. Govoni, M.D.*

Meyers, Morton A. Radiological features of the spread and localization of extraperitoneal gas and their relationship to its source: an anatomical approach. *Radiology*, April, 1974, *111*, 17–26. (Address: Department of Radiology, New York Hospital-Cornell University Medical Center, 525 E. 68th Street, New York, N. Y. 10021.)

Extraperitoneal gas is usually produced by perforation of bowel secondary to inflammatory or ulcerative disease, blunt or penetrating trauma, a foreign body, iatrogenic manipulation, or gas-producing infection originating in extraperitoneal organs. Extraperitoneal gas displays a characteristic spread and localization depending upon its source and differs from peritoneal gas primarily in that its spread is guided by the fixed fascial planes and paths of least tissue resistance.

The author describes the anatomy of the extraperitoneal fascial planes and compartments, based on careful study of body sections, postmortem injections, and retroperitoneal pneumography. The extraperitoneal region extends from the posterior parietal peritoneum in front to the fascia transversalis posteriorly and is demarcated into 3 distinct compartments by fascial layers:

1. The anterior pararenal space is located between the posterior parietal peritoneum and the anterior renal fascia and includes the ascending and descending colon, duodenal loop, and the pancreas.
2. The perirenal compartments are located between the anterior and posterior renal fascia which fuses medially with the psoas fascia and the great vessels.
3. The posterior pararenal space extends between the posterior renal fascia and the fascia transversalis and includes a thin layer of adipose tissue, blood vessels, and lymphatics but no major organs.

The hallmark of gas in the extraperitoneal tissue is

mottled or linear radiolucencies which often lie along established fascial planes. On erect or decubitus roentgenograms, these gaseous collections remain fixed, or show only limited mobility, a reliable distinction from intraperitoneal gas. Observation of 2 further characteristics on the upright roentgenograms is helpful in differentiating small amounts of extraperitoneal gas from free intraperitoneal air:

1. Free intraperitoneal air conforms to the highest curvature of the dome of the diaphragm and may have a flat lower border. Subphrenic extraperitoneal gas often parallels a lower plane of the diaphragmatic curvature, medial or lateral to its apex, and invariably demonstrates a crescentic outline.
2. Intraperitoneal subdiaphragmatic air increases on inspiration and decreases on expiration in contrast to extraperitoneal gas which appears to increase on expiration and decrease on inspiration.

The author describes the localization and spread of extraperitoneal gas from perforation of the descending duodenum, gas producing infections of the pancreas, perforated sigmoid diverticulitis, rectal perforation, perineal gas producing infections and extraperitoneal gas of subdiaphragmatic origin. Roentgen localization of extraperitoneal gas to a specific compartment and an understanding of its route of spread, governed by the fascial boundaries, is of practical significance in determining the most likely source of the gas.—*J. L. Montgomery, M.D.*

Genitourinary System

Olier, Ch., Benassayag, E., Galian, Ph., and Franc, B. La pyonéphrose xanthogranulomateuse de l'adulte est-elle une affection rare? A propos de 71 observations anatomocliniques. (Is xanthogranulomatous pyelonephritis a rare lesion in adults? A review of 71 anatomo-clinical cases.) *Presse méd.*, Jan., 1974, *3*, 133–137. (Address: Dr. Ch. Olier, Hôpital Cochin, 27, rue du Faubourg Saint-Jacques, F. 75674 Paris Cedex 14, France.)

The diagnosis of xanthogranulomatous pyelonephritis (XGP) rests upon precise macroscopic and histologic criteria. It is not a specific pathologic entity, but rather a particular evolutive stage of a suppurative pyelonephritis, resulting from poor drainage of necrotic material complicated with increased cellular destruction and liberation of lipids.

Out of a total of 155 cases of pyelonephritis operated on in the authors' hospital between 1960 and 1972, 71 cases were classified as xanthogranulomatous. Such a high incidence goes against the generally recognized rate of this process.

In their study, the authors report a female predominance: 47 females against 24 males. Among the males, a peak incidence is noticed between 60 and 70 years of age, whereas in the females XGP occurred uniformly throughout the various decades. Bilateral involvement has not been encountered; the right kidney was diseased 29 times and the left 42 times. The authors' practice being restricted to adults, no children are included in their study.

Clinical findings. The duration of symptoms extended from 2 months to 16 years. Most of the patients were or had been under antibiotic treatment prior to their hospital admission. Symptoms were nonspecific: lumbar pains, at time mimicking renal colics; poor general state (62 cases); fever, often oscillating and above 38° C. (47 cases). A palpable abdominal or lumbar mass was present in 34 cases. Urinary infection was detected in all the cases and was identified in 67 cases. Positive gram germs were responsible in 6 cases; amicrobial pyuria was encountered in 7 cases, including 2 cases of renal tuberculosis. Negative gram germs were demonstrated in 51 cases and identified in 34 cases as follows: *Proteus* in 23 cases, out of which 17 cases showed concomitant lithiasis; *E. coli* bacillus in 7 cases; *Pyocyanic bacillus* in 2 cases; and *Klebsiella* and *Aerobacter* in 1 case, respectively.

Radiologic findings. The radiologic investigation was found adequate in 56 cases. Renal mensurations were sometimes greatly increased, but small kidneys were also observed. Intravenous pyelography revealed a nonfunctional kidney in 77 per cent of the cases. Cavity opacification was performed either by percutaneous puncture of the renal pelvis or, in most cases, by preoperative retrograde catheterization. Lithiasis, present in 43 cases, could be radiologically localized in 41 cases; coralliform calculi were present in 24 cases; pyelic lithiasis was noted in 10 other cases; calyceal lithiasis in 5 others, and ureteral lithiasis in 4 cases. Chemical analysis, performed in 32 of these cases, yielded a phosphatic content in 19 cases, an oxalic content in 7, and uric content in 6. In the 15 remaining cases radiologically investigated, no lithiasis was demonstrated. A tumoral appearance was observed in 4 of them without obstruction. Arteriography, which was performed in 3 of them, demonstrated elongation and thinning of the arteries around a dense avascular mass, without anomalies of the peripheral vessels or of the venous return. In 1 of the cases malakoplakia was associated with the xanthogranuloma.

The comparison of this radiologic series with one of nonxanthogranulomatous pyelonephritis did not show a significant difference in the lithiasis incidence or the radiologic appearance. Two particular aspects deserve mention: (1) cyst formations unmodified by compression were observed in 4 cases (such a finding is attributable to the intense sclerotic reaction); (2) "geographical map" margins of the opacified pyelocalyceal cavities which are explained by pyofibrinous deposits.

Surgical treatment. Total nephrectomy was per-

formed in 65 cases, completed by preventive ureterectomy in 3 cases. Splenectomy was necessitated in 2 cases due to accidental tearing during the operation. When the lesions were circumscribed, a more conservative treatment was applied: partial nephrectomy was resorted to in 2 cases; enucleation of the granuloma in 1 case; and simple surgical drainage of the abscessed granuloma in 3 cases.

Surgical complications included: prolonged operative scar suppuration in 6 cases; persistent lumbar fistula in 1 case; and postoperative death supervened in 2 cases, one from massive hemorrhage following surgical laceration of vena cava and the other from cachexia and amyloidosis.—*Henri-Paul Lévesque, M.D.*

Goldin, Alan R., Naude, Johan H., and Thatcher, Geoffrey N. Therapeutic percutaneous renal infarction. *Brit. J. Urol.*, April, 1974, *46*, 133–135. (From: Departments of Diagnostic Radiology, Urology, and Medicine, Groote Schuur Hospital and the University of Cape Town, South Africa.)

A sterile barium suspension was injected *via* selective renal catheter in order to induce therapeutic infarction in a hypertensive patient.

The circumstances of this case were as follows: a 21 year old woman developed renal failure on the basis of rapidly progressive glomerulonephritis. One kidney was resected at the time of a cadaver renal transplantation; however, the graft was rejected. She was subsequently managed by hemodialysis, although despite adequate medical therapy her hypertension had been uncontrollable. Nephrectomy had been considered, but was felt to carry an excessive risk in this instance and for that reason she was referred for percutaneous infarction. Preliminary abdominal aortography *via* percutaneous femoral catheter confirmed the presence of a single right renal artery which was then catheterized. The selective renal arteriogram demonstrated a contracted kidney with a thin rim of parenchyma and also showed that there was no adrenal artery arising from the renal artery. At this point, with the patient slightly rotated to the right to lower the kidney, 5 ml. of barium suspension was carefully injected with fluoroscopic monitoring. The renal circulation was filled as far proximal as the main divisions of the renal artery, and barium was not permitted to reflux proximal to the catheter tip. The patient remained in the right oblique position for 20 minutes following the procedure. Right flank pain developed 1 hour after infarction and was moderately severe for 3 days. The white blood cell count rose, but then returned to normal after 10 days. The blood pressure gradually dropped to 150 over 90 ml. of mercury and remained at that level. An abdominal roentgenogram 6 weeks following the procedure showed that the barium filled vessels had decreased in size.

The authors point out that the safety of percutaneous renal infarction depends entirely on meticulous injection of the barium suspension, with great care being exercised to prevent reflux into the aorta with risk of paraplegia. This control was readily achieved by means of fluoroscopic monitoring of multiple small volume barium injections.—*R. H. Troupin, M.D.*

Cendron, J., Gubler, J. P., Valayer, J., and Kiriakos, S. Dysplasie multikystique du rein chez l'enfant: à propos de 45 observations. (Multicystic dysplasia of the kidney in children: report of 45 cases.) *J. urol. et néphrol.*, Oct.-Nov., 1973, *79*, 773–799.

Multicystic renal dysplasia also has been called multicystic kidney (MCK) by Schwartz in 1936. It is not an exceptional renal malformation in children; it is more common than polycystic disease or multilocular cyst. It is an acquired, nonhereditary congenital lesion, usually without familial history. About 150 cases have been recorded in the literature under varied denominations.

Three main criteria distinguish the MCK from the other cystic renal disorders: the cystic malformation is unilateral; the dysplasia involves the whole kidney; and its nature is fetal, not congenital.

The authors have collected 45 cases over a period of 12 years. Sex distribution was practically equal and the ages ranged from 0 to 10 years: 80 per cent before the age of 2; and 60 per cent before 16 months. In 28 cases, the right kidney was involved; in 17 cases, it was the left kidney. In 2 cases afflicted with a double kidney, the lesion was observed in the upper one. The lesion was also present in 2 cases with horseshoe kidney and in 1 case with crossed renal ectopia. Five of the cases were diagnosed at autopsy.

The malformation presents 2 different anatomoclinical forms: (1) the pseudotumoral MCK, where the size and weight exceed those of a normal kidney with a contralateral kidney usually normal; (2) the hypoplastic MCK associated in 50 per cent of the cases with contralateral nephrourinary lesions. In 20 cases, where a tumoral mass was clinically discovered, the mass corresponded to a pseudotumoral MCK in 17 cases and in 3 cases to a contra-lateral dilated kidney, the other kidney being a small hypoplastic MCK. In 22 cases where the MCK was revealed by nephrourologic symptoms, the hypoplastic type was present except in 1 case. Clinical symptoms are not due to the hypoplastic MCK, but to contralateral renal lesions.

Radiologic findings. The plain film roentgenogram of the abdomen is useful only in newborns and for the pseudotumoral MCK, revealing the retroperitoneal location of the mass by the compression of the small and large bowels. The barium enema study might demonstrate compression or lowering of the colon. Intravenous urography is an essential examination

and was performed in 38 of the cases. The MCK is not functional and the contralateral kidney was normal in 19 cases, but abnormal in 19 others. A pyeloureteral junction syndrome was present in 5 cases, and in 2 other cases the pyelocalyceal dilatation was attributable to compression by a large MCK. Ureteral stenosis was demonstrated in 1 case, ureteral reflux in 4 cases, lithiasis in 1 case, hypoplasia in 2 cases and unobstructive pyelocalyceal anomaly in 1 case. Cystography will rarely opacify the blind ureter but is useful, when a contralateral lesion is suspected, to demonstrate ureterocele and detect ureteral reflux. Cystoscopy was performed 28 times, revealing an absent ureteral meatus in 5 cases, a gaping meatus in 1 case and ureterocele in 4 cases. An obliterated ureter was found in 2 cases and was partially catheterized. Retrograde pyelography was performed when the ureteral orifice could be catheterized. When a blind ureter is demonstrated with reflux of the contrast medium in the bladder, these changes favor a MCK.

The differential clinical diagnosis is different according the age of the patient and the pseudotumoral or hypoplastic type of the MCK. In the tumoral form, MCK is highly suggestive with the symptomatic triad: lumbar tumor; nonfunctional kidney; and blind ureter. In the forms revealed by urinary symptomatology, the symptoms are related to contralateral lesions, the MCK being nonfunctional and hypoplastic. The urologic investigation has 2 goals: (1) the identification of the silent kidney (if it is associated with a blind ureter, the diagnosis is certain, if the ureteral meatus is absent, MCK is to be considered along with renal agenesis which is often associated with homolateral malformation of the gonad); (2) identification of the contralateral lesion responsible for the symptoms.

Pathologic anatomy. The MCK is characterized by 3 essential elements: a polycavitary mass without a kidney-shape; more or less extensive atresia of the ureter; and filiform blood vessels. Macroscopically, the MCK is formed by multiple cystic cavities surrounding a dense central core, where the blood vessels and the ureter, if present, converge. In the tumoral form, the involved kidney is larger than the contralateral kidney, the cysts are pressed one against another. The cysts are translucid and, after opaque medium injection, intercommunication between 2 or 3 of them may be demonstrated. There is no communication between the cysts and the ureter or the hypoplastic excretory cavities that might be present in the central core.

In the hypoplastic form, the same macroscopic aspect is observed except that the peripheral cysts are smaller, less numerous, and less crowded.

The central core of the MCK is composed of a fibrous structure intermingled with cystic microcavities reminiscent of aborted calyces or pelvis.

The blood supply of the MCK is rarely secured by an individual pedicle, but usually by many tiny arterioles originating from the aorta. The perirenal network is abundant.

The excretory system is practically always abnormal: it was totally absent in 13 cases, although there was in 1 case a vesical meatus which could not be catheterized, or it might be present as a vague fibrous tract. In most cases the ureter is present with atresia of its upper part.

Histologically, the dysplasia in the MCK is associated with a complete architectural disorganization and immature structures. The contralateral anomalies of the kidney are seen nearly exclusively in the hypoplastic type of MCK.

In the differential histopathologic diagnosis, the authors discuss the infantile and adult type of polycystic disease, multilocular cyst, solitary cyst, urinary cysts, diffuse cortical cystosis and medullary sponge kidney.

At the end, they consider the hypotheses concerning the pathogenesis of MCK related to the metanephros and the ureteral bud.—*Henri-Paul Lévesque, M.D.*

Skeletal System

Borghi, A., and D'Ettorre, A. L'osteoblastoma benigno: considerazioni radiologiche su 43 casi. (The benign osteoblastoma: radiologic considerations on 43 cases.) *Riv. radiol.*, 1973, *13*, 47–66. (Address: A. Borghi, Servizio di Radiologia, Istituto Ortopedico Rizzoli, Bologna, Italy.)

The osteoblastoma, which is also known under the following synonyms: osteogenic fibroma (Golding and Sisson, 1954); osteoblastocytoma (Reitano and Lanza, 1965); and giant osteoid osteoma (Dahlin and Johnson, 1954), is a rare benign neoplasm of the bone arising from the mesenchymal elements, the osteoblasts, and characterized by the proliferation of these cells with new formation of osteoid tissue.

Jaffe (1956) and Lichtenstein (1956) first recognized the pathologic changes which are the characteristics of the osteoblastoma, while the largest series which brought further light on this type of osseous neoplasm were by Goidanich and Battaglia (1958) and Lichtenstein and Sawier (1964).

From an analysis of the literature and of their own series the authors point out that the tumor is more frequent in the second and third decade, that patients of the male sex are more often affected, and that the tumor involves mainly the long bones and the spine. Rare are other localizations and exceptional is the involvement of a phalanx (Appicciutoli *et al.*, 1971).

Briefly reviewed are the gross pathologic and the histologic findings. Discussed in detail are the roentgen characteristics which should lead to a correct roentgenologic conclusion, and the differential diagnostic criteria with other bone lesions, such as:

formed in 65 cases, completed by preventive ureterectomy in 3 cases. Splenectomy was necessitated in 2 cases due to accidental tearing during the operation. When the lesions were circumscribed, a more conservative treatment was applied: partial nephrectomy was resorted to in 2 cases; enucleation of the granuloma in 1 case; and simple surgical drainage of the abscessed granuloma in 3 cases.

Surgical complications included: prolonged operative scar suppuration in 6 cases; persistent lumbar fistula in 1 case; and postoperative death supervened in 2 cases, one from massive hemorrhage following surgical laceration of vena cava and the other from cachexia and amyloidosis.—*Henri-Paul Lévesque, M.D.*

Goldin, Alan R., Naude, Johan H., and Thatcher, Geoffrey N. Therapeutic percutaneous renal infarction. *Brit. J. Urol.*, April, 1974, *46*, 133–135. (From: Departments of Diagnostic Radiology, Urology, and Medicine, Groote Schuur Hospital and the University of Cape Town, South Africa.)

A sterile barium suspension was injected *via* selective renal catheter in order to induce therapeutic infarction in a hypertensive patient.

The circumstances of this case were as follows: a 21 year old woman developed renal failure on the basis of rapidly progressive glomerulonephritis. One kidney was resected at the time of a cadaver renal transplantation; however, the graft was rejected. She was subsequently managed by hemodialysis, although despite adequate medical therapy her hypertension had been uncontrollable. Nephrectomy had been considered, but was felt to carry an excessive risk in this instance and for that reason she was referred for percutaneous infarction. Preliminary abdominal aortography *via* percutaneous femoral catheter confirmed the presence of a single right renal artery which was then catheterized. The selective renal arteriogram demonstrated a contracted kidney with a thin rim of parenchyma and also showed that there was no adrenal artery arising from the renal artery. At this point, with the patient slightly rotated to the right to lower the kidney, 5 ml. of barium suspension was carefully injected with fluoroscopic monitoring. The renal circulation was filled as far proximal as the main divisions of the renal artery, and barium was not permitted to reflux proximal to the catheter tip. The patient remained in the right oblique position for 20 minutes following the procedure. Right flank pain developed 1 hour after infarction and was moderately severe for 3 days. The white blood cell count rose, but then returned to normal after 10 days. The blood pressure gradually dropped to 150 over 90 ml. of mercury and remained at that level. An abdominal roentgenogram 6 weeks following the procedure showed that the barium filled vessels had decreased in size.

The authors point out that the safety of percutaneous renal infarction depends entirely on meticulous injection of the barium suspension, with great care being exercised to prevent reflux into the aorta with risk of paraplegia. This control was readily achieved by means of fluoroscopic monitoring of multiple small volume barium injections.—*R. H. Troupin, M.D.*

Cendron, J., Gubler, J. P., Valayer, J., and Kiriakos, S. Dysplasie multikystique du rein chez l'enfant: à propos de 45 observations. (Multicystic dysplasia of the kidney in children: report of 45 cases.) *J. urol. et néphrol.*, Oct.-Nov., 1973, *79*, 773–799.

Multicystic renal dysplasia also has been called multicystic kidney (MCK) by Schwartz in 1936. It is not an exceptional renal malformation in children; it is more common than polycystic disease or multilocular cyst. It is an acquired, nonhereditary congenital lesion, usually without familial history. About 150 cases have been recorded in the literature under varied denominations.

Three main criteria distinguish the MCK from the other cystic renal disorders: the cystic malformation is unilateral; the dysplasia involves the whole kidney; and its nature is fetal, not congenital.

The authors have collected 45 cases over a period of 12 years. Sex distribution was practically equal and the ages ranged from 0 to 10 years: 80 per cent before the age of 2; and 60 per cent before 16 months. In 28 cases, the right kidney was involved; in 17 cases, it was the left kidney. In 2 cases afflicted with a double kidney, the lesion was observed in the upper one. The lesion was also present in 2 cases with horseshoe kidney and in 1 case with crossed renal ectopia. Five of the cases were diagnosed at autopsy.

The malformation presents 2 different anatomoclinical forms: (1) the pseudotumoral MCK, where the size and weight exceed those of a normal kidney with a contralateral kidney usually normal; (2) the hypoplastic MCK associated in 50 per cent of the cases with contralateral nephrourinary lesions. In 20 cases, where a tumoral mass was clinically discovered, the mass corresponded to a pseudotumoral MCK in 17 cases and in 3 cases to a contra-lateral dilated kidney, the other kidney being a small hypoplastic MCK. In 22 cases where the MCK was revealed by nephrourologic symptoms, the hypoplastic type was present except in 1 case. Clinical symptoms are not due to the hypoplastic MCK, but to contralateral renal lesions.

Radiologic findings. The plain film roentgenogram of the abdomen is useful only in newborns and for the pseudotumoral MCK, revealing the retroperitoneal location of the mass by the compression of the small and large bowels. The barium enema study might demonstrate compression or lowering of the colon. Intravenous urography is an essential examination

and was performed in 38 of the cases. The MCK is not functional and the contralateral kidney was normal in 19 cases, but abnormal in 19 others. A pyeloureteral junction syndrome was present in 5 cases, and in 2 other cases the pyelocalyceal dilatation was attributable to compression by a large MCK. Ureteral stenosis was demonstrated in 1 case, ureteral reflux in 4 cases, lithiasis in 1 case, hypoplasia in 2 cases and unobstructive pyelocalyceal anomaly in 1 case. Cystography will rarely opacify the blind ureter but is useful, when a contralateral lesion is suspected, to demonstrate ureterocele and detect ureteral reflux. Cystoscopy was performed 28 times, revealing an absent ureteral meatus in 5 cases, a gaping meatus in 1 case and ureterocele in 4 cases. An obliterated ureter was found in 2 cases and was partially catheterized. Retrograde pyelography was performed when the ureteral orifice could be catheterized. When a blind ureter is demonstrated with reflux of the contrast medium in the bladder, these changes favor a MCK.

The differential clinical diagnosis is different according the age of the patient and the pseudotumoral or hypoplastic type of the MCK. In the tumoral form, MCK is highly suggestive with the symptomatic triad: lumbar tumor; nonfunctional kidney; and blind ureter. In the forms revealed by urinary symptomatology, the symptoms are related to contralateral lesions, the MCK being nonfunctional and hypoplastic. The urologic investigation has 2 goals: (1) the identification of the silent kidney (if it is associated with a blind ureter, the diagnosis is certain, if the ureteral meatus is absent, MCK is to be considered along with renal agenesis which is often associated with homolateral malformation of the gonad); (2) identification of the contralateral lesion responsible for the symptoms.

Pathologic anatomy. The MCK is characterized by 3 essential elements: a polycavitary mass without a kidney-shape; more or less extensive atresia of the ureter; and filiform blood vessels. Macroscopically, the MCK is formed by multiple cystic cavities surrounding a dense central core, where the blood vessels and the ureter, if present, converge. In the tumoral form, the involved kidney is larger than the contralateral kidney, the cysts are pressed one against another. The cysts are translucid and, after opaque medium injection, intercommunication between 2 or 3 of them may be demonstrated. There is no communication between the cysts and the ureter or the hypoplastic excretory cavities that might be present in the central core.

In the hypoplastic form, the same macroscopic aspect is observed except that the peripheral cysts are smaller, less numerous, and less crowded.

The central core of the MCK is composed of a fibrous structure intermingled with cystic microcavities reminiscent of aborted calyces or pelvis.

The blood supply of the MCK is rarely secured by an individual pedicle, but usually by many tiny arterioles originating from the aorta. The perirenal network is abundant.

The excretory system is practically always abnormal: it was totally absent in 13 cases, although there was in 1 case a vesical meatus which could not be catheterized, or it might be present as a vague fibrous tract. In most cases the ureter is present with atresia of its upper part.

Histologically, the dysplasia in the MCK is associated with a complete architectural disorganization and immature structures. The contralateral anomalies of the kidney are seen nearly exclusively in the hypoplastic type of MCK.

In the differential histopathologic diagnosis, the authors discuss the infantile and adult type of polycystic disease, multilocular cyst, solitary cyst, urinary cysts, diffuse cortical cystosis and medullary sponge kidney.

At the end, they consider the hypotheses concerning the pathogenesis of MCK related to the metanephros and the ureteral bud.—*Henri-Paul Lévesque, M.D.*

Skeletal System

Borghi, A., and D'Ettorre, A. L'osteoblastoma benigno: considerazioni radiologiche su 43 casi. (The benign osteoblastoma: radiologic considerations on 43 cases.) *Riv. radiol.*, 1973, *13*, 47–66. (Address: A. Borghi, Servizio di Radiologia, Istituto Ortopedico Rizzoli, Bologna, Italy.)

The osteoblastoma, which is also known under the following synonyms: osteogenic fibroma (Golding and Sisson, 1954); osteoblastocytoma (Reitano and Lanza, 1962); and giant osteoid osteoma (Dahlin and Johnson, 1954), is a rare benign neoplasm of the bone arising from the mesenchymal elements, the osteoblasts, and characterized by the proliferation of these cells with new formation of osteoid tissue.

Jaffe (1956) and Lichtenstein (1956) first recognized the pathologic changes which are the characteristics of the osteoblastoma, while the largest series which brought further light on this type of osseous neoplasm were by Goidanich and Battaglia (1958) and Lichtenstein and Sawier (1964).

From an analysis of the literature and of their own series the authors point out that the tumor is more frequent in the second and third decade, that patients of the male sex are more often affected, and that the tumor involves mainly the long bones and the spine. Rare are other localizations and exceptional is the involvement of a phalanx (Appicciutoli *et al.*, 1971).

Briefly reviewed are the gross pathologic and the histologic findings. Discussed in detail are the roentgen characteristics which should lead to a correct roentgenologic conclusion, and the differential diagnostic criteria with other bone lesions, such as:

Brodie's abscess, osteogenic fibroma, chondroblastoma, and osteoid osteoma.

The authors have reviewed the case histories of the Archives of the Istituto Ortopedico Rizzoli and found 43 cases of benign osteoblastoma, of which 14 were previously reported by Goidanich and Battaglia.

The roentgen findings were grouped as follows: (1) area of osteolysis without an associated osteosclerotic reaction (5 cases); (2) area of osteolysis with perifocal osteosclerotic reaction (16 cases); (3) expansion of the involved bone with central calcifications (10 cases); (4) multicameral aspect (8 cases); and (5) osteosclerosis and hyperostosis (4 cases).

Several reproductions demonstrate the roentgen findings described by the authors.—*A. F. Govoni, M.D.*

Palmia, C., and Volterrani, F. Lo studio delle para-osteoartropatie nei paraplegici. (The study of the para-osteoarthropathies in paraplegics.) *Nuntius Radiol.*, Sept., 1969, *35*, 831–841. (From: Reparto radiologico del Centro Traumatologico Ortopedico di Milano, Italy.)

The authors, after a brief review of the pathologic changes and of the etiopathogenesis of the heterotopic ossifications observed in patients with traumatic spinal cord injuries, describe the related roentgen findings and discuss them also from the differential point of view.

This analysis is based on a review of 380 para- or tetraplegic patients, of whom 152 presented paraarticular heterotopic processes of ossification. The appearance of these new bone formations was noted between the seventh and tenth week from the trauma, reaching the maximal formation of bone within 2 years.

The heterotopic ossifications were noted in the hip in 78 cases, in the knee in 36, in the femur in 25, in the elbow in 9, and in the shoulder in 4 cases.

The spinal cord lesion was localized in the cervical spine in 17 cases, between T1 and T9 in 52, between T10 and T12 in 23, and in the lumbar segment in 60 patients.

Several demonstrative illustrations accompany the article. The references are limited only to the French and Italian medical literature.—*A. F. Govoni, M.D.*

Rémy, J., Béguery, P., Binot, A., and Ponté, C. La dysplasie spondylo-épiphysaire tardive: à propos de trois observations nouvelles. (Spondylo-epiphyseal dysplasia tarda; report of 3 new cases.) *J. de radiol., d'électrol. et de méd. nucléaire*, Nov., 1973, *54*, 805–812. (Address: Pr agr. J. Rémy, Service central de Radiologie, Hôpital A.-Calmette, F. 59000 Lille, France.)

Spondylo-epiphyseal dysplasia tarda (SEDT) is a rare hereditary disease with a recessive X-linked transmission. Only 88 cases have been recorded in the literature, but its frequency is probably underestimated: the entity is poorly known and its minor forms are unrecognized.

The authors present the history and the radiologic manifestations of the disease in 3 brothers, aged respectively 18, 17 and 12 years. In their discussion, they proceeded to a general review of the disease.

SEDT is usually discovered past the age of 10, on account of a short stature or premature osteoarthrosis, or at the occasion of a family study. *Clinically*, the dysmorphism is rather typical: there is a mild dwarfism due to a trunk shortening; the extremities are normally developed. The pubis-vertex distance is shorter than the pubis-ground distance. Dorsal xyphosis and lumbar hyperlordosis are frequently present. The narrow pelvic rim contrasts with the normal scapular rim. The neck is short, but the face and skull are normal.

Radiologic changes. There is a generalized vertebral platyspondyly. The lesions predominate in the lower dorsal and the lumbar area with a characteristic aspect. In the anteroposterior view, the vertebral body is flattened, its end plates are irregular with a median biconvex bulging. In the lateral view the anterior third of the vertebral body is flat and the biconvex bulging involves the posterior two-third. In the cervical region and the upper half of the dorsal spine, the vertebral bodies are generally flattened with their end plates irregular, slightly undulated but remaining grossly parallel. In some cases, the odontoid process is hypoplastic and cone-shaped. In adults, early spondylarthrosis developed: the intervertebral disk is thinned, sometimes calcified, and often shows the vacuum sign in patients under 40 years of age.

The epiphyseal lesions are inconstant, moderate and predominate in the proximal extremities. Their shape is more altered than trabecular structures. The articular surfaces are irregular, flattened and flared.

The *differential diagnosis* of SEDT is easy when its features are known. In the poly-epiphyseal dysplasia with vertebral involvement, there is no platyspondyly and the end plates are irregular with intraspongious herniations reminiscent of Scheuermann's disease. In brachylomy, there is also truncular dwarfism and generalized platyspondyly, but the end plates of the vertebral bodies remain parallel, irregular, sometimes biconcave. The epiphyseal lesions are usually restricted to the hips. In spondylometaphyseal dysplasia, platyspondyly is generalized, but the vertebral lesions are more severe and the changes of the pelvis are diagnostic. In congenital spondyloepiphyseal dysplasia, the disease is present from birth and the pelvic changes are again diagnostic. Morquio's disease manifests itself earlier, dwarfism is more pronounced, platyspondyly is marked with a typical tongue-like projection on the anterior wall

of the vertebral body. On the other hand, the epiphyseal and metaphyseal lesions are generalized.—*Henri-Paul Lévesque, M.D.*

Feldman, Frieda, Johnson, Austin M., and Walter, Joseph F. Acute axial neuroarthropathy. *Radiology*, April, 1974, *111*, 1–16. (Address: Frieda Feldman, M.D., Department of Radiology, Columbia-Presbyterian Medical Center, 622 W. 168th Street, New York, N. Y. 10032.)

Although neuroarthropathies were once linked with varied spinal cord and peripheral nerve lesions, they are most commonly referred to as "Charcot's joints," associated with tabes, and described as disorganized, unstable, and painless.

Various theories of pathogenesis have evolved through the years. The consensus of opinion currently favors involvement of insensitive joints which are actively misused or abused as motor power remains intact. Varied etiologies and clinical presentations suggest additional contributory factors, such as local infection and vasomotor, trophic, and metabolic disturbances; however, these have not been substantiated.

Diabetic arthropathy has been particularly problematic, being regarded alternately as neuropathic, infectious, or ischemic in origin. Productive bone changes have been linked with central spinal cord lesions such as trauma, tumor, congenital malformations, and diseases which commonly spare the sympathetic nervous system, including tabes, (posterior tracts) and syringomyelia (central gray matter). Destructive bone manifestations have been associated more often with peripheral nerve injuries and related to trauma, alcoholism, and diabetes, which presumably affects postganglionic nerve segments carrying sympathetic vasoconstrictive as well as sensory and motor fibers.

It has been postulated that neuroarthropathy may be related to a neurally initiated vascular reflex leading to increased osteoclastic bone resorption in an area supplied by a particular vascular bed. Thus, loss of vasoconstrictive impulses presumably leads to osteolysis due to hyperemia and bone resorption; however, local fragmentation may mechanically compromise vascular supply, leading to local necrosis. Thus, the pathogenesis of neuroarthropathy has not been entirely resolved, with current clinical concepts being based predominantly on peripheral manifestations.

Mention of a "Charcot joint" has invariably conjured up a classical clinical and roentgenographic complex involving swelling, hypermobility, malalignment, and osteosclerosis without pain in a peripheral joint, typically appearing in a patient with a protracted course and positive serology and usually following minor trauma.

The authors review their experience with 14 cases which indicates that these prerequisites are often not satisfied, particularly when applied to the vertebral column. Their cases emphasize that the spine deserves more frequent consideration as a primary presenting site of neuropathic disease and document its acute or rapidly progressive development in the spine. Several less familiar clinical and roentgenographic features are illustrated.

Because of the problems that exist in a pathologic, serologic, and clinical diagnosis of neuropathic disease, the pivotal role of the roentgenologist in suggesting or confirming the diagnosis is emphasized.—*Jon F. Bergstrom, M.D.*

Meema, H. Erik, Oreopoulos, Dimitrios G., Rabinovich, Sol, Husdan, Harry, and Rapoport, Abraham. Periosteal new bone formation (periosteal neostosis) in renal osteodystrophy: relationship to osteosclerosis, osteitis fibrosa, and osteoid excess. *Radiology*, March, 1974, *110*, 513–522. (Address: H. Erik Meema, M.D., The Toronto Western Hospital, 399 Bathurst Street, Toronto, Ontario M5T 2R8, Canada.)

Periosteal new bone formation may be either centrifugal (or appositional), in which lamellar new bone is slowly added to the external bone surfaces, or centripetal, in which new bone forms along an elevated periosteum at some distance from the bone. To distinguish this latter form, the term periosteal neostosis (PN) is suggested. An example of this type of reaction is found in hypertrophic osteoarthropathy. Periosteal neostosis in renal osteodystrophy has been previously described in only 11 patients.

In a review of 117 patients with chronic renal disease, the authors found 10 patients with PN. All their patients were studied with comprehensive skeletal surveys, microradioscopic examination of the hands for subperiosteal and intracortical resorption, grading of osteosclerosis and arterial calcifications, and densitometric evaluation of bone mineral content. Iliac crest biopsies were performed when possible to evaluate for osteitis fibrosa, osteomalacia, and osteosclerosis.

Of the 10 patients with PN, 6 had been treated with hemodialysis or peritoneal dialysis. Four had complained of some bone pain during the period of observation of the PN. In the patients with PN, serum calcium levels were variable, serum phosphorus levels were elevated in all but 1 (who had had a renal transplant) and alkaline phosphatase levels were elevated in all. In the patients without PN, alkaline phosphatase was elevated in less than half. PN was observed in a variety of long bones, most commonly in the metatarsals, and also in the pelvis. Subperiosteal resorption was seen in all patients with PN. Osteosclerosis was observed roentgenographically in 9 or 10. There was no significant difference in occurrence of pathologic fractures or

soft tissue calcifications in patients with or without PN. Iliac crest biopsies were performed in 8 of the 10 patients. Of the 8, all showed osteoid excess (osteomalacia), 7 showed osteitis fibrosa, and 3 or 4 showed osteosclerosis. Periosteal calcifications or new bone formations were seen histologically in 7 of the cases upon review of the material. Histologic observation in this series suggests that the primary factor in pathogenesis of PN is osteitis fibrosa rather than osteomalacia, as has been previously suggested.

PN is apparently one of the hyperostotic manifestations of secondary hyperparathyroidism occurring in chronic renal disease and is usually associated with osteosclerotic skeletal changes seen elsewhere in the body.—*J. M. Stoebner, M.D.*

Blood and Lymph System

Sokoloff, Joel, Wickbom, Ingmar, McDonald, Daniel, Brahme, Folke, Goergen, Thomas G., and Goldberger, Lawrence E. Therapeutic percutaneous embolization in intractable epistaxis. *Radiology*, May, 1974, *111*, 285–287. (Address: Joel Sokoloff, M.D., Department of Radiology, University of California, San Diego, Calif.)

Two cases of intractable epistaxis with angiographic localization of the site of hemorrhage and successful percutaneous therapeutic embolization are reported. Both patients had profuse hemorrhage which did not respond to posterior nasal packs and required transfusion in both cases. Selective external carotid arteriography localized the active bleeding sites from the sphenopalatine artery. Selective embolization of the maxillary artery was accomplished using Gelfoam particles cut to 1–3 mm. in size suspended in normal saline. There was prompt occlusion of the offending vessel and immediate cessation of bleeding in both cases

These cases illustrate that angiography can not only localize the site of bleeding in instances where surgical intervention is needed, but can be utilized as a therapeutic procedure. The only morbidity was a transient right infraorbital hypoesthesia in the second reported case.

An addendum to the article relates the experience with an additional patient with successful control of the epistaxis. Aspiration secondary to profuse hemorrhage occurred and the authors recommend the use of endotracheal intubation in instances of severe bleeding.—*James A. Chapman, M.D.*

Ekelund, Leif, and Lunderquist, Anders. Pharmacoangiography with angiotensin. *Radiology*, March, 1974, *110*, 533–540. (Address: Leif Ekelund, M.D., Department of Diagnostic Radiology, University Hospital, Lund, Sweden.)

Eighty patients from 10 to 79 years of age received selective catheterization of various vessels. Fifteen to 20 minutes after routine angiography, angiotensin was employed as a pharmacoangiographic agent. In one-half of the patients the hepatic artery was injected. The remainder was among the celiac, superior mesenteric, inferior mesenteric, iliac, femoral, subclavian, and other arteries. No untoward reactions were encountered.

Methods of injection and recommended dosages of angiotensin are given. The diagnostic value of angiography improved in 70 per cent of the cases. In 5 cases there was reduction of diagnostic value. In 3 patients (1 with liver metastasis, 1 with fibrosarcoma in the soft tissue of the arm, and 1 with adenocarcinoma of the cecum) tumor vessels could be seen only following angiotensin injection. The diagnostic improvement reflected a higher concentration of contrast material in the smaller arteries due to reduced blood flow. Also, there was visualization of a larger number of tumor vessels and improved outlining of tumors.—*A. Kent Teaford, M.D.*

Radiation Therapy

Jacquillat, Cl., Belpomme, D., Weil, M., Auclerc, G., Teillet, F., Weisgerber, C., Tanzer, J., Boiron, M., and Bernard, Jean. Les néoplasies simultanées et successives: à propos de 18 observations d'affections malignes compliquant l'évolution de la maladie de Hodgkin. (The simultaneous and successive neoplasms: report of 18 observations of malignancy complicating the evolution of Hodgkin's disease.) *La nouvelle Presse méd.*, Dec., 1973, *2*, 3089–3092. (Address: Pr agr. Cl. Jacquillat, Hôpital Saint-Louis, 2, place du Docteur-Fournier, F. 75475 Paris Cedex 10, France.)

The authors review the pertinent literature and note that the risk of developing cancer is 3 to 5 times greater among patients having Hodgkin's disease as compared to normals. This risk is 29 times greater for those who have been treated extensively with radiation and intensive chemotherapy.

The authors' study of 18 patients treated extensively for Hodgkin's disease revealed development of Kaposi's sarcoma in 2 cases, acute myeloblastic leukemia in 6 cases and visceral cancer of various origin in 10 cases.

No relationship could be noted between these cancerous complications and the initial characteristics, or histology or stage of the disease. The authors, however, note the importance of prior repeated and extensive radiotherapy and intensive treatments with all the derivatives of nitrogen mustard.

In spite of this augmented risk, they conclude that such vigorous treatments are not contraindicated, for they constitute the best guarantee for the control of Hodgkin's disease.—*Jirair N. Sarian, M.D.*

Green, Nathan, Iba, Gerald, and Shirey, John K. The clinical experience of patients with carcinoma of the lung and chronic pulmonary disease treated by radiotherapy. *Radiology*, April, 1974, *111*, 189–192. (Address: Nathan Green, M.D., Division of Radiation Therapy, Hospital of the Good Samaritan, 1212 Shatto Street, Los Angeles, Calif. 90017.)

The indications for radiotherapy in the management of lung cancer patients, who also have a chronic pulmonary disease, have not been substantiated. Forty-one such patients received irradiation to their primary lung cancer. Thirty patients had tumor spread to the hilus and/or mediastinum, 9 had peripheral tumors, and 2 had postoperative recurrence.

Opposed treatment portals, varying in size from 100 sq. cm. to 400 sq. cm., were used. Doses less than 4,000 rads were employed in 10 patients, and 31 patients received 4,000 rads or greater.

Thirty-one patients had objective tumor regression assessed by comparing roentgenograms.

Twenty patients experienced subjective improvement of symptoms. Seven patients developed radiation pneumonitis, 1 developed tuberculosis, and another died near completion of treatment from cardiorespiratory shock.

Neither field size nor severity of chronic pulmonary disease could be correlated with the incidence of radiation pneumonitis.

Patients with lung cancer and chronic pulmonary disease had a shorter median survival than expected. Radiotherapy was of benefit in alleviating hemoptysis, chest pain and superior vena caval syndrome. Peripheral cancers and asymptomatic individuals received little benefit from radiotherapy.—*Odis D. Skinner, M.D.*

Papillon, Jean. Endocavitary irradiation of early rectal cancers for cure: a series of 123 cases. *Proc. Roy. Soc. Med.*, Dec., 1973, *66*, 1179–1181. (From: Radiotherapy Department, Centre Leon-Berard, Lyons, France.)

Endocavitary irradiation is an effective method of treatment of early, small rectal cancers in highly selective cases. There is no mortality and there are few complications. Seventy-two per cent of 123 cases treated are alive and well after more than 5 years. Eighteen patients died from rectal cancer (8 from metastasis, 9 from local-regional spread, and 1 postoperative death).

Radiation therapy is given by contact x ray, and supplemented by interstitial curie therapy to the residual lesion or to the bed of the tumor.

A Philips machine was used for contact therapy and had the following characteristics: voltage 50 kv.; focal distance 4 cm.; output 1,500–2,000 r/min. in air; important absorption in the first 1 cm.; and a narrow light tube capable of introduction into a special rectoscope. The field of irradiation was circular with a diameter of 3 cm. Two overlapping fields may be used.

Three to 5 applications of 2–4 minutes each were made during an over-all treatment time of 4–6 weeks. A dose of 2,500–4,000 r was given at each session in the outpatient department. The tumor was evaluated prior to each treatment, and the technique was adjusted according to the speed of shrinkage of the tumor.

Radium implantation was made in the knee-chest position without general anesthesia. Needles 4 cm. in length were kept in place against the rectal wall by a rubber drain which was pushed through the anus and fixed to the skin. A dose of 2,000–3,000 rads was delivered in 2–3 days.

There is usually a mild proctitis for 2–3 weeks. There are no significant subsequent rectal problems. Occasionally, there is slight bleeding due to telangiectasis.

Criteria for patient selection are included in the article.—*Paul M. Dassel, M.D.*

Server, G., Rodríguez-Bermejo, M., and Monzonis, M. C. Carcinoma pleomorfico osificante de la vejiga. (Pleomorphic carcinoma of the urinary bladder containing osteoid tissue.) *Arch. españ. urol.*, Jan.-Feb., 1947, *17*, 85–92. (From: Residencia Sanitaria de la S. S. "Virgen de la Arrixaca," Servicios de Urología y Anatomía Patológica, Murcia, Spain.)

The authors report the second recorded case in the medical literature of an epithelial bladder tumor containing osteoid tissue, giving it a histologic appearance similar to that of a giant cell bone tumor. Most probably the presence of osteoid tissue is due to the fact that epithelial cells rich in alkaline phosphatase can differentiate into osteoid-producing cells.

The osteoid was not calcified; therefore the tumor was not detectable on the preliminary intravenous pyelogram. After the bladder was outlined with contrast medium the tumor showed as a filling defect.

The patient was a 74 year old male, who presented with recurrent gross hematuria.—*Julian B. Alvarez, M.D.*

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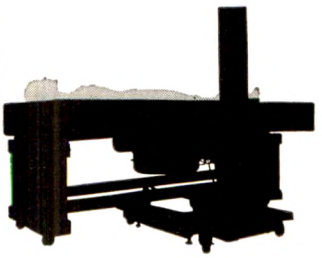
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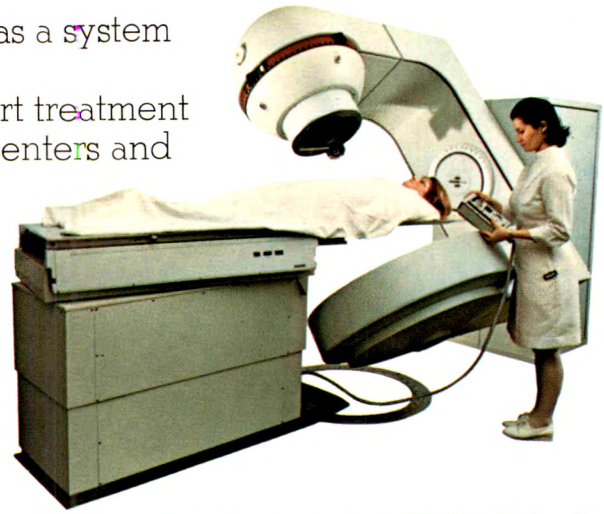
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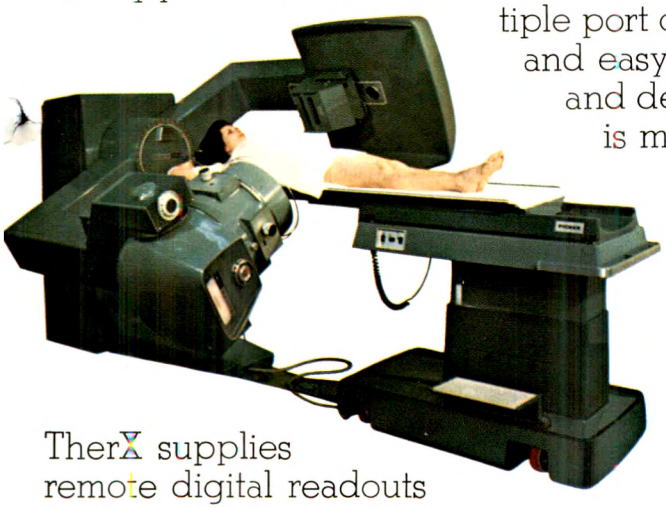


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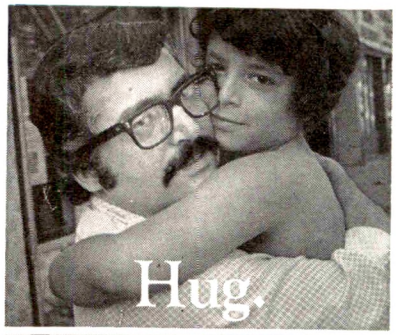
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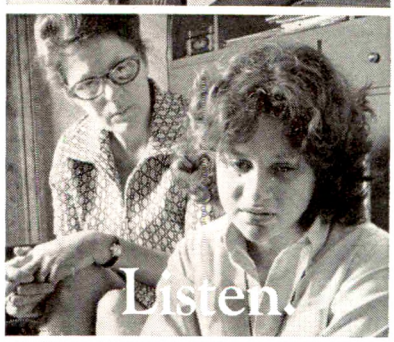
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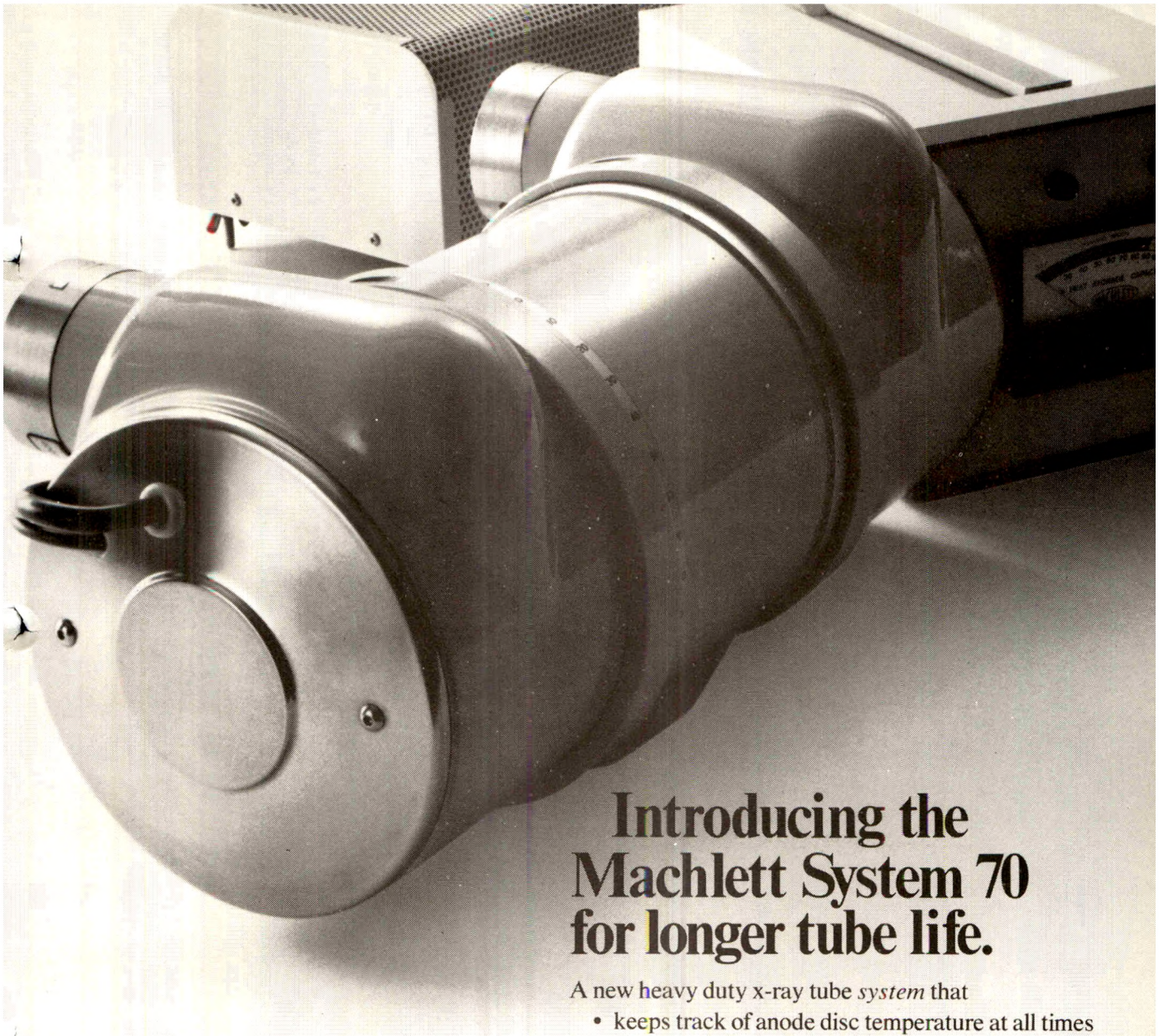




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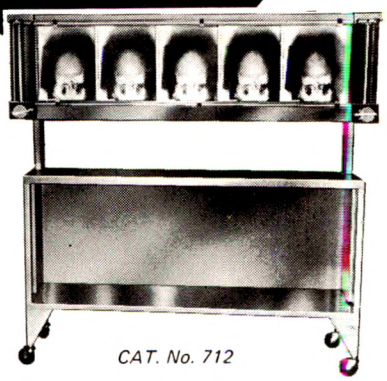
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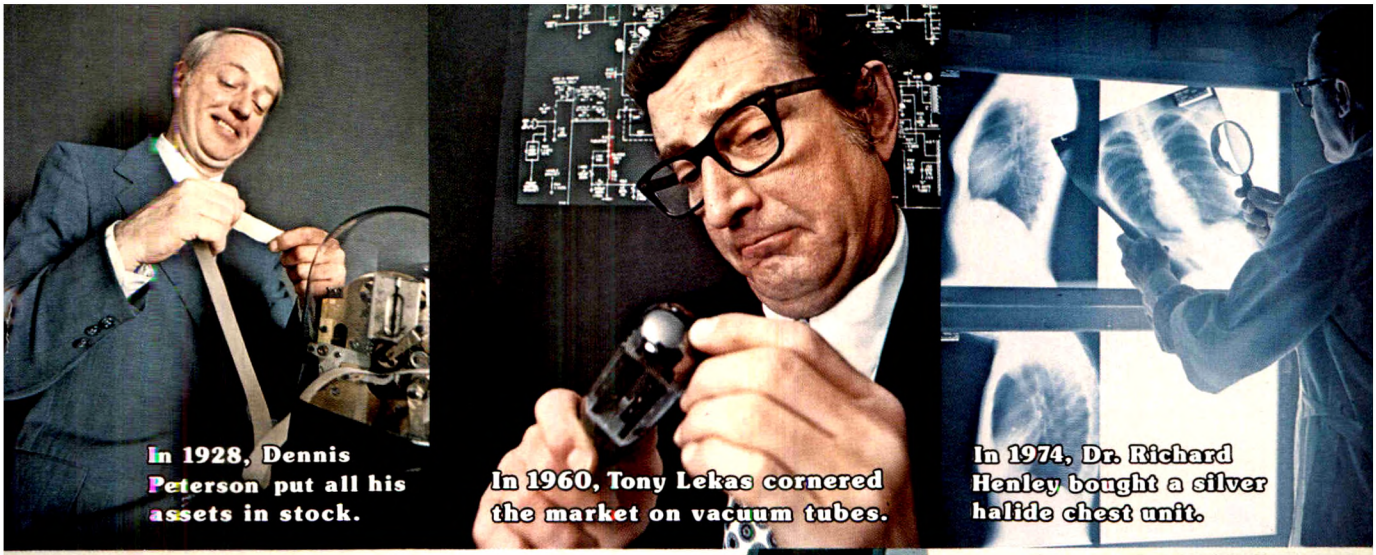
Handbook for RADIOLOGIC TECHNOLOGISTS AND SPECIAL PROCEDURES NURSES IN RADIOLOGY *by* **Nieta Whitman Powell,** *formerly at Oklahoma Univ., Oklahoma City. Foreword by* **Bob G. Eaton.** This book is written for special procedures nurses, technologists and physicians who are given the responsibility of establishing a special procedures laboratory. Covered are such areas as the team concept, adult and pediatric laboratories, procedural techniques, sterile techniques, surgical prep and draping areas, instrument preparation, cardiac catheterization, general radiographic procedures, cardiopulmonary resuscitation and electrical safety. Many illustrations and drawings are included to increase the book's communicative value. *'74, 104 pp., 30 il., $8.75*

INDICATIONS AND ALTERNATIVES IN X-RAY DIAGNOSIS: A Guide to the Effective Employment of Roentgenologic Studies in the Solution of Diagnostic Problems (2nd Ed.) *by* **Melvyn H. Schrieber,** *Univ. of Texas, Galveston.* Chapters are based upon organ systems illustrating common abnormalities. The reader is provided with a thorough background in the uses to which the ordinary and special x-ray studies may be put, emphasizing alternatives available when the indicated examination cannot be performed or when more than one kind of examination is available. Indications and contraindications are carefully defined. *'74, 192 pp., 98 il., $11.50, paper*

XERORADIOGRAPHY OF THE BREAST (2nd Ptg.) *by* **John N. Wolfe,** *Hutzel Hospital, Detroit, Michigan. Foreword by* **Wendell G. Scott.** Written primarily for radiologists but of interest to others working in the field of breast cancer, this text presents a new and significant method for early detection of breast cancer. The mechanics and principles involved in this procedure are clearly spelled out. Breast anatomy and normal breasts are discussed, providing a thorough understanding essential to achieving the proper technique and correct interpretation of the xeromammogram. The xeroradiographs included are accurately reproduced in their original blue color for consistency of detail. *'74, 194 pp. (8 1/2 x 11), 242 il. (92 in color), $23.50*

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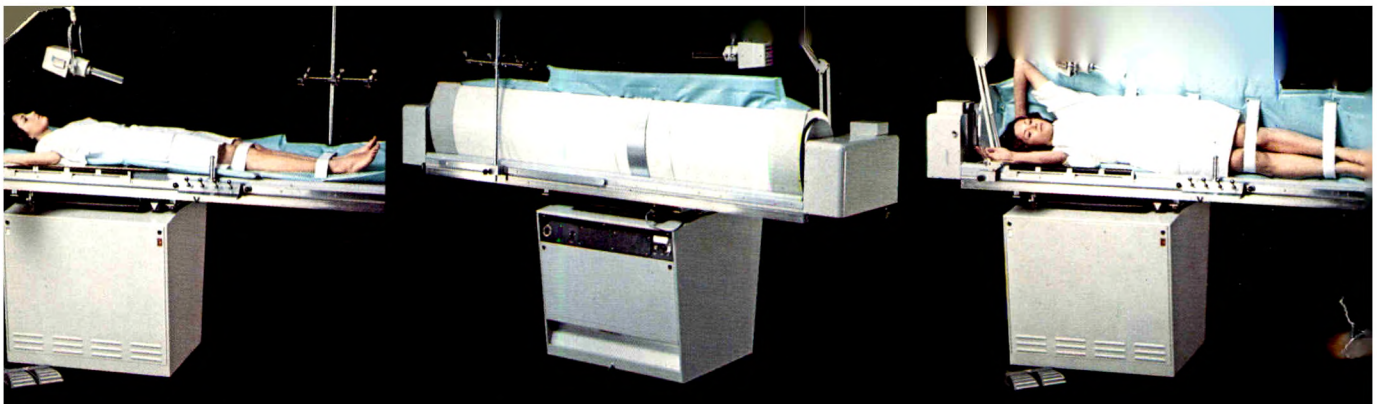
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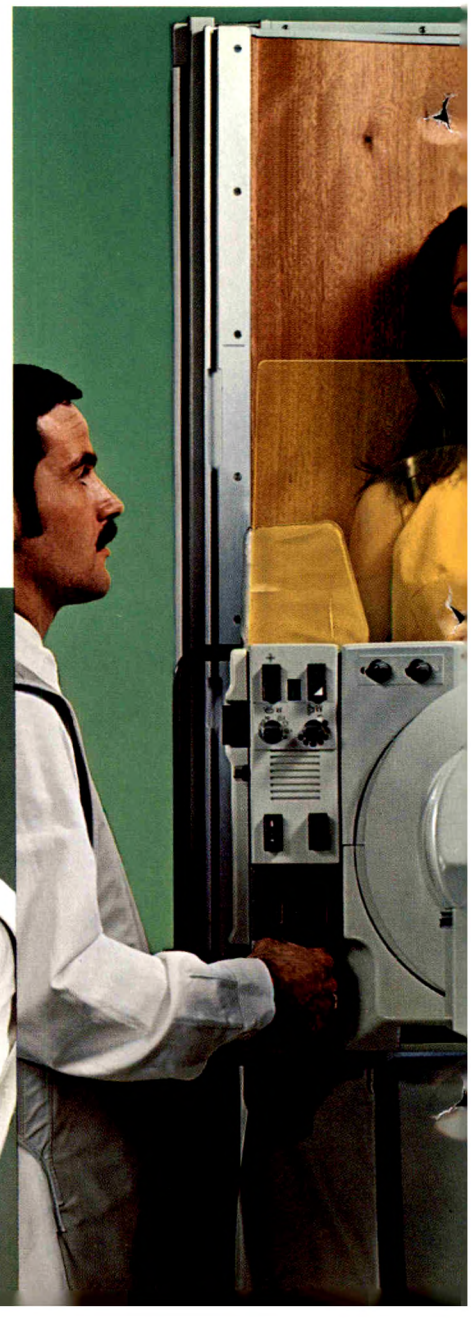
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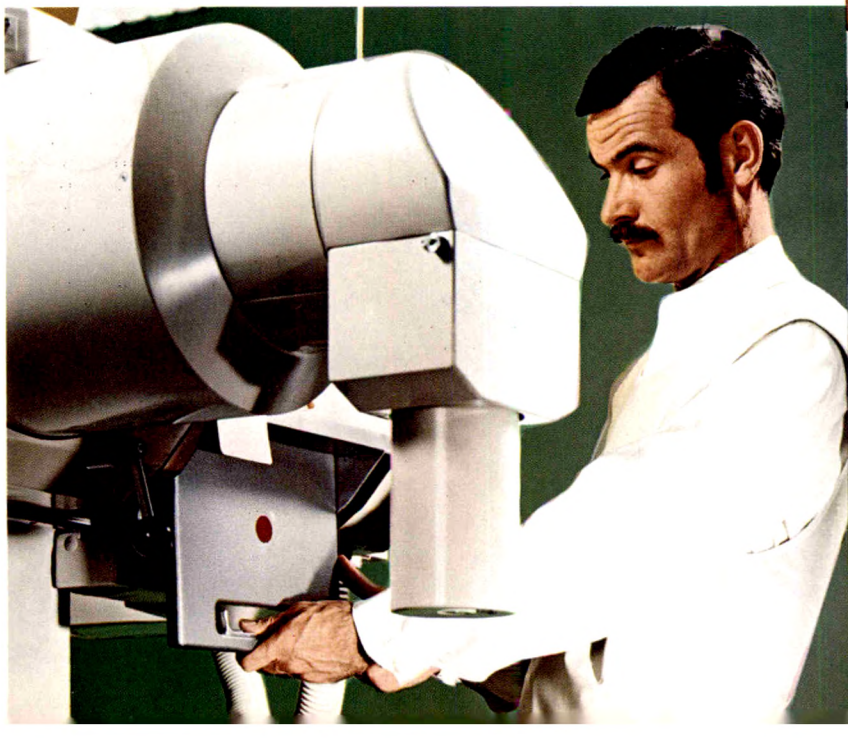
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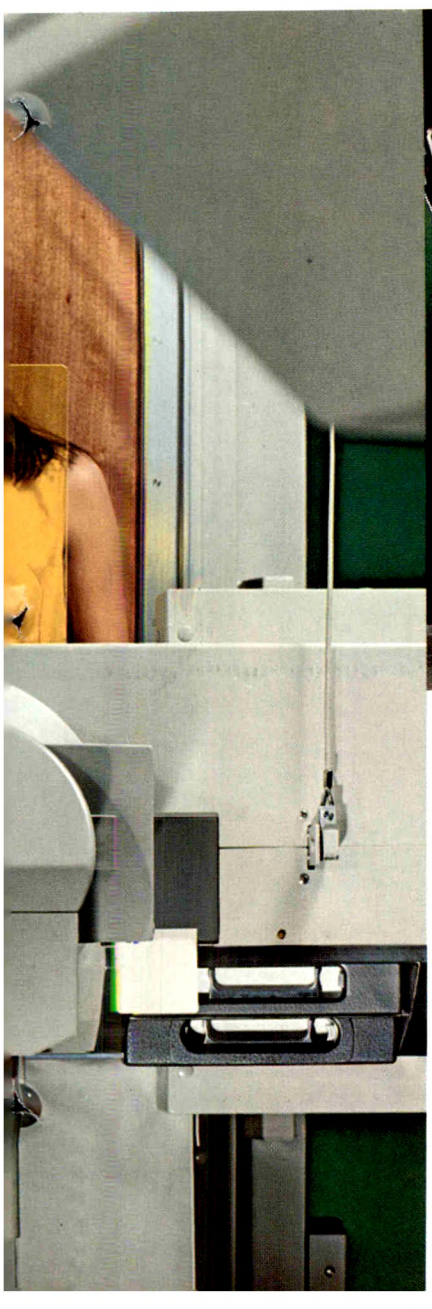
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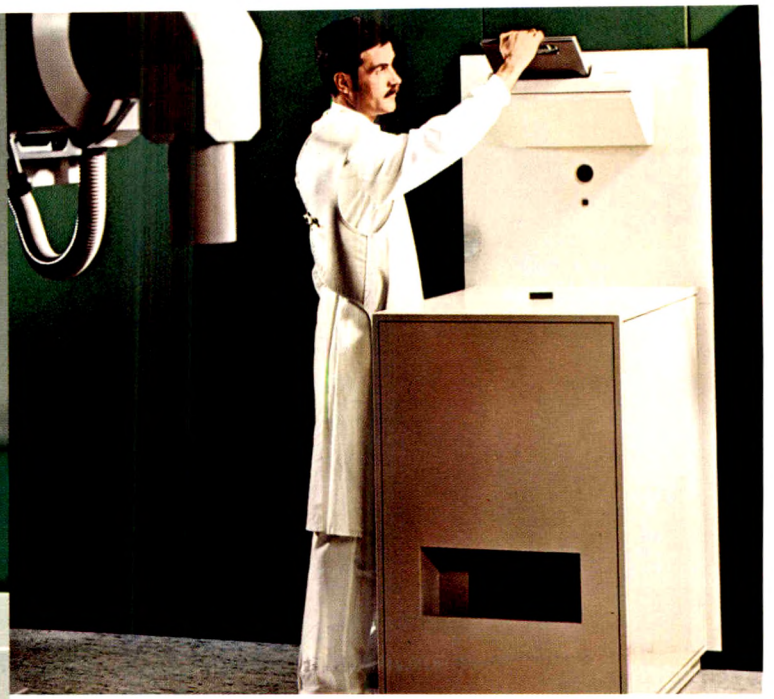
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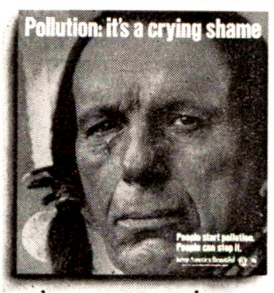
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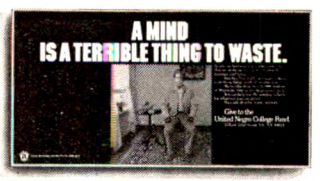




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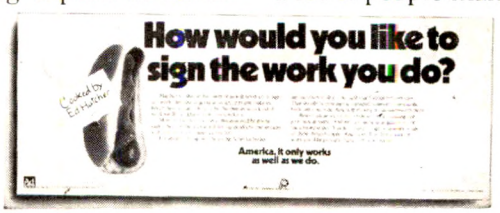
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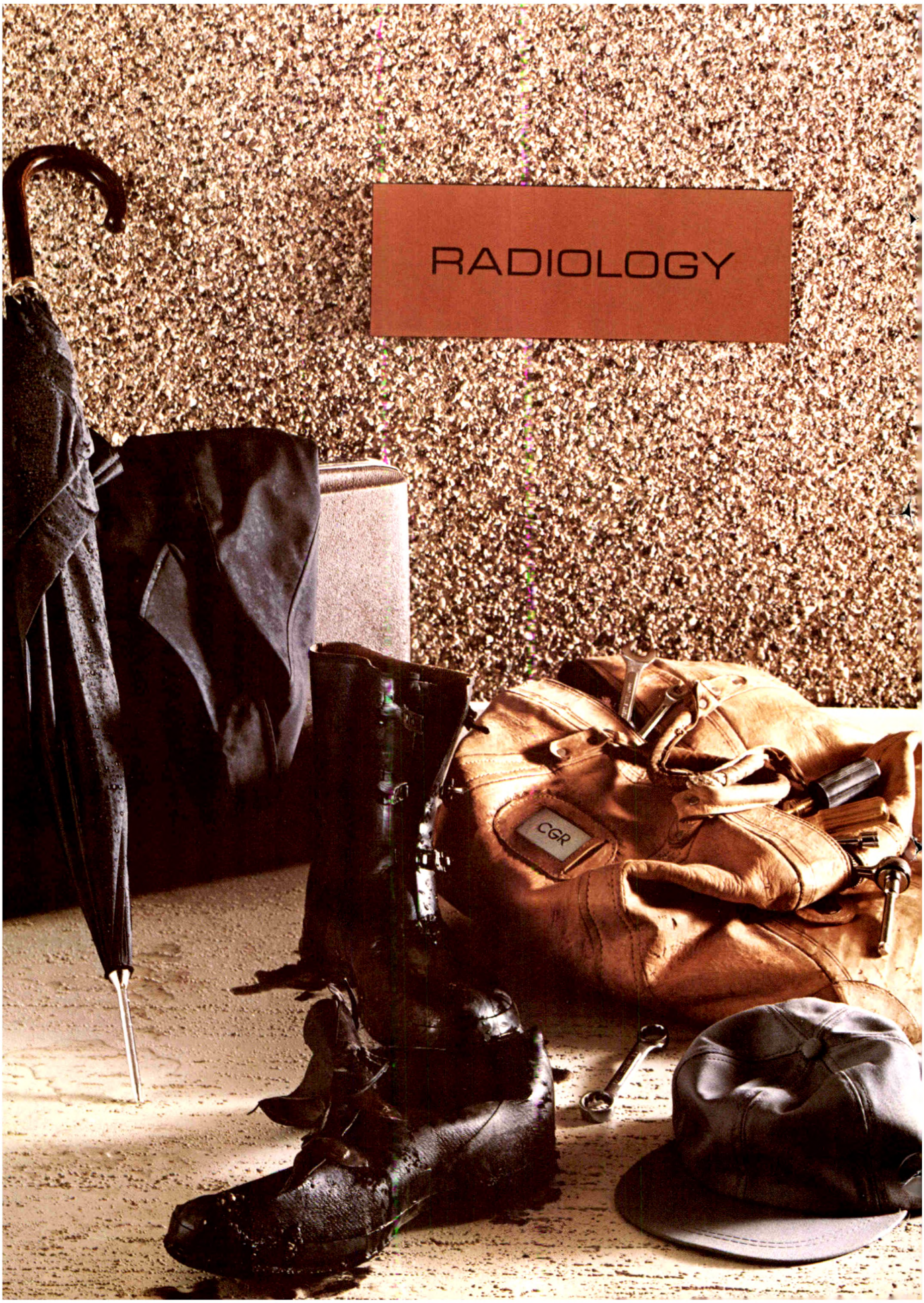
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THE FUNDAMENTALS OF RADIOLOGICAL SCIENCE *by* **John Hale**, *Hospital of the Univ. of Pennsylvania, Philadelphia.* The four basic fields of applied radiological science are radiotherapy, clinical nuclear medicine, diagnostic radiology and radiation safety physics. It is the aim of this textbook to present the basic body of scientific knowledge which is common to the specialty fields of applied radiological science. A student needs exposure to two different texts—one dealing with basic radiological physics and another more advanced book dealing with one of the four specialized areas. He must have this basic understanding of fundamental principles in order to continue to learn to assimilate new ideas and methods throughout his professional life. Directed toward first-year residents in radiology, this book will also be useful as a beginning text for physicists and engineers who are interested in radiological or health physics. After a review of the basic mathematics and physics essential in the study of radiological sciences has been presented a careful study is made of atomic and nuclear physics, the radiation field and the interaction of radiation with matter. Finally, the fundamentals of dosimetry are discussed. *'74, 356 pp., 172 il., 3 tables, $14.75*

THE BIOLOGICAL AND CLINICAL BASIS OF RADIOSENSITIVITY. *Edited by* **Milton Friedman**, *Instituto Regina Elena, Rome, Italy. (35 Contributors)* Radiosensitivity, a factor which must be considered in the treatment of every tumor and in most radiobiological experiments, encompasses disruptive changes that occur at many levels. The wide variety of factors under study has resulted in a communication gap between the laboratory and clinical researchers. In order to reduce this gap expert investigators in different fields were asked to adapt their observations to the concepts of radiosensitivity and chemosensitivity. Questions considered are: At which biological level do major effects occur? Which are the most important factors influencing radiosensitivity? What are the differences between radiosensitivity and chemosensitivity? What clinically useful information can be derived from cell survival curves? Can the variation in sensitivity during each phase of the cell cycle be used to improve the ultimate radiation effect? Should its possible usefulness be explored further, or should it be abandoned? At the preclinical level, the lethal dose for each tumor is depicted from several viewpoints. Combined radiation and chemotherapy is evaluated not only for potential benefits, but also for possible disruption of efficient irradiation. The neglected field of dynamic radiation pathology, studied by examination with light and electron microscopy, of serial biopsy specimens is surveyed. Finally, a detailed investigation is described, wherein the validity of a number of radiobiological laws are analyzed during the treatment of squamous cell carcinoma. A comprehensive and detailed index renders a vast amount of available information. *'74, about 589 pp. (7 x 10), 373 il., 67 tables*

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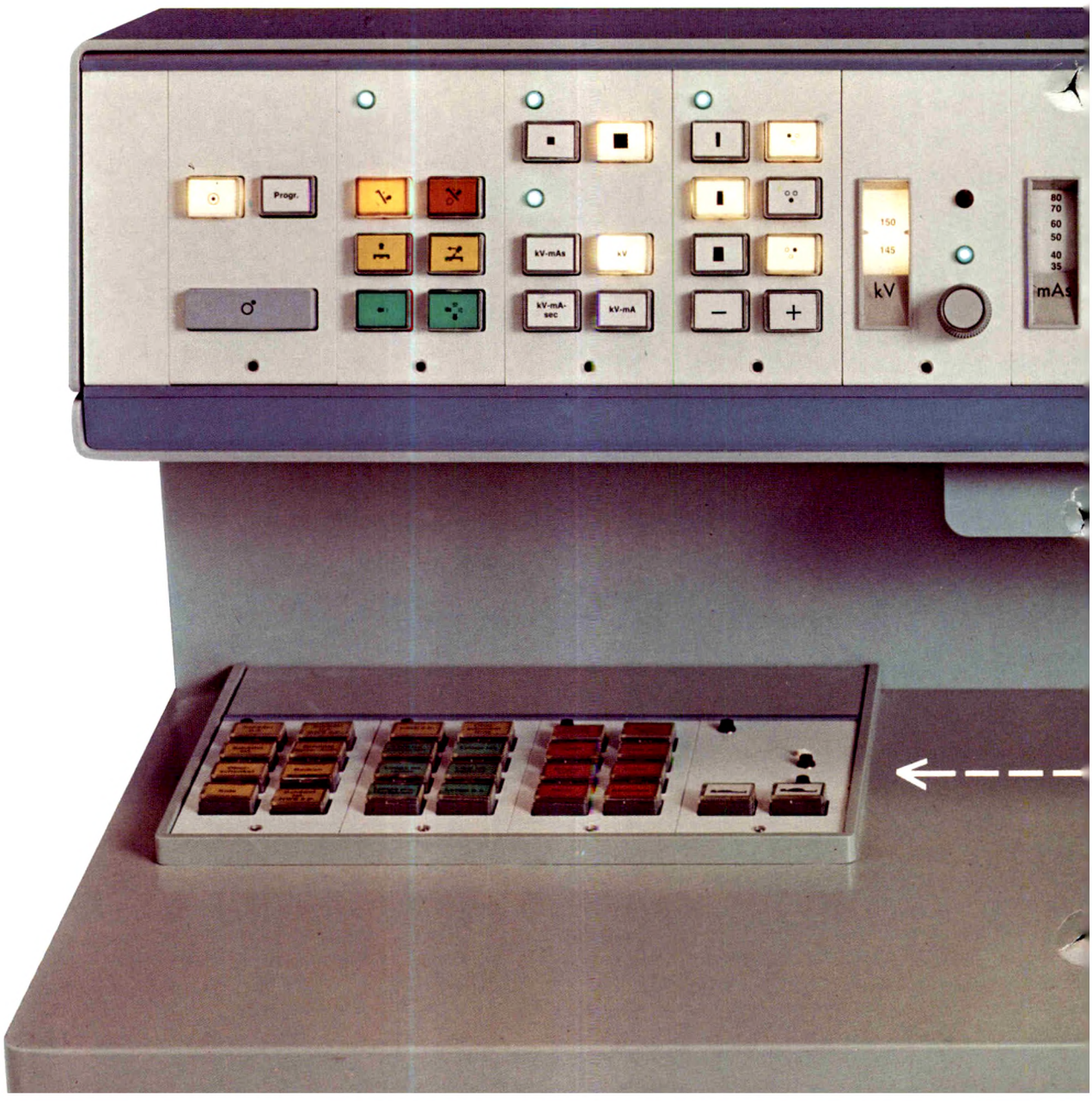
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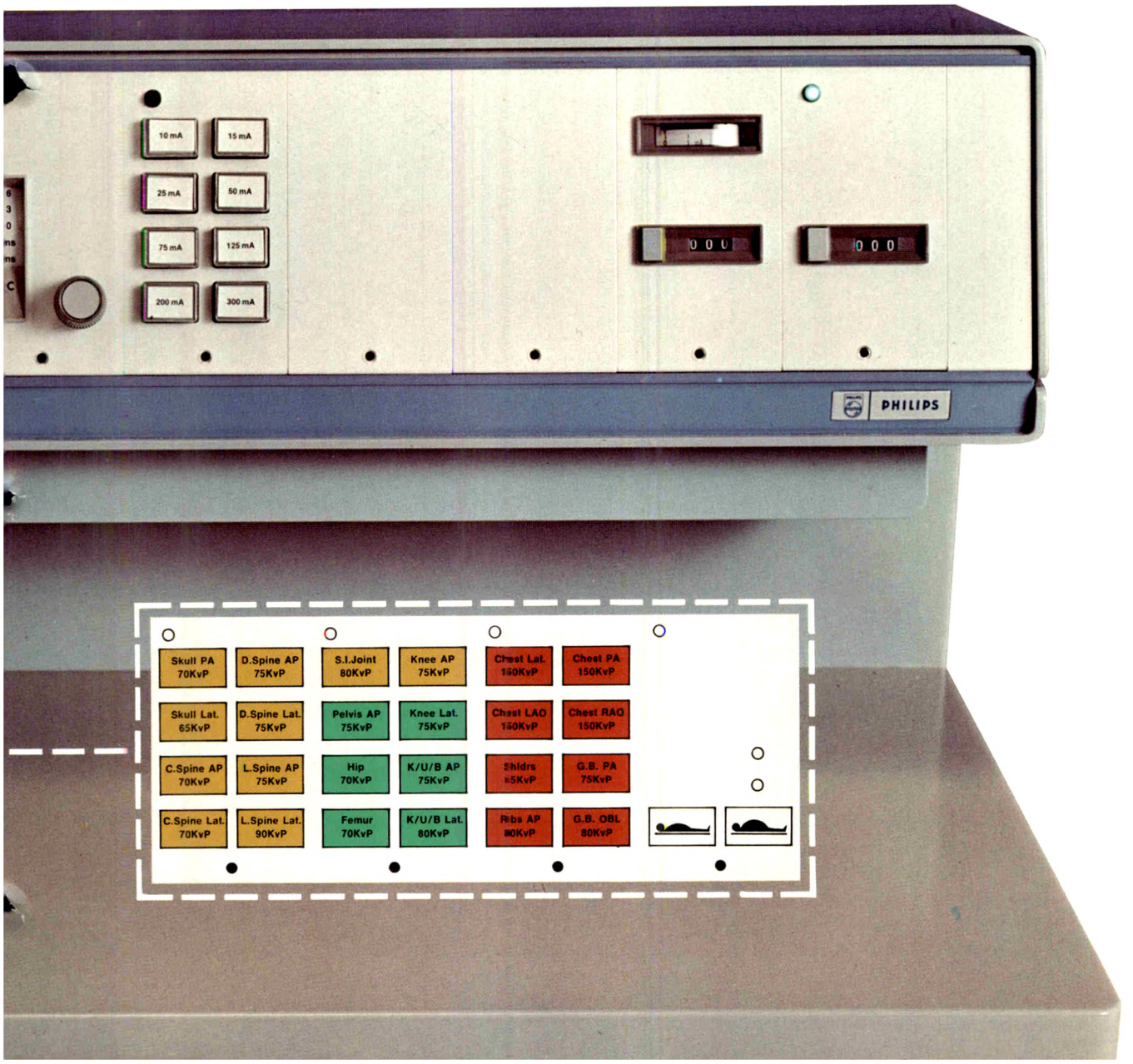
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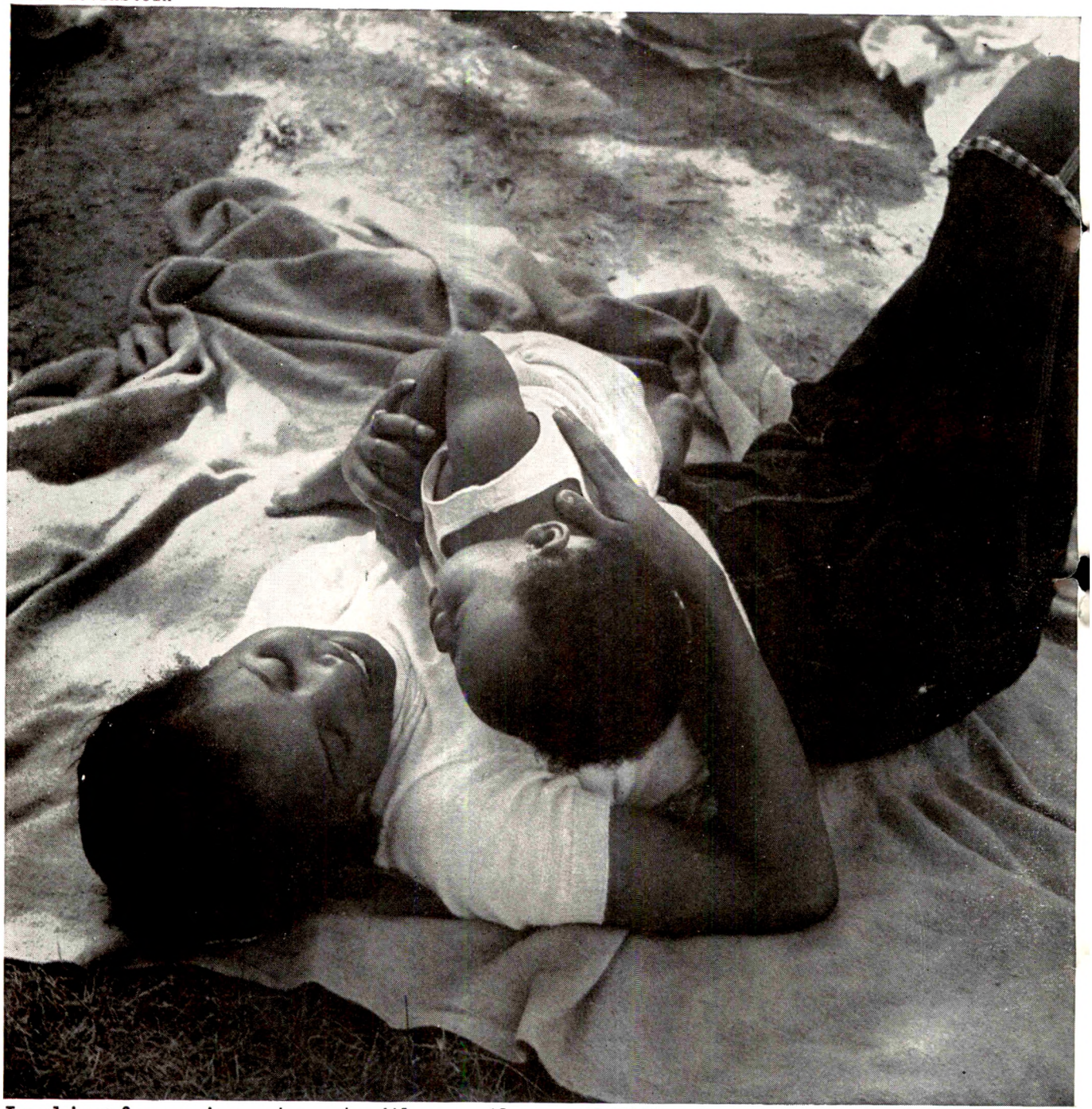
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Smith, John Med. Rec. # 3211498

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Dr. N. Hall Day # Mon 777 EMER
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UPPER G.I. SERIES
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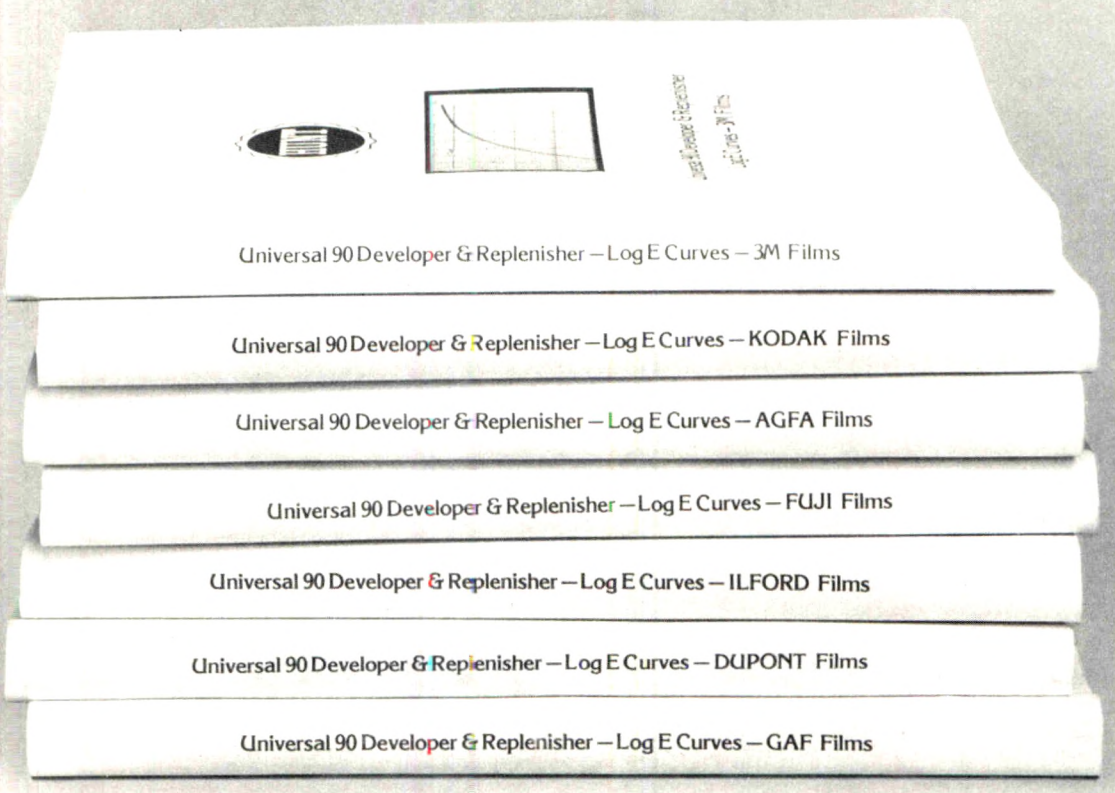
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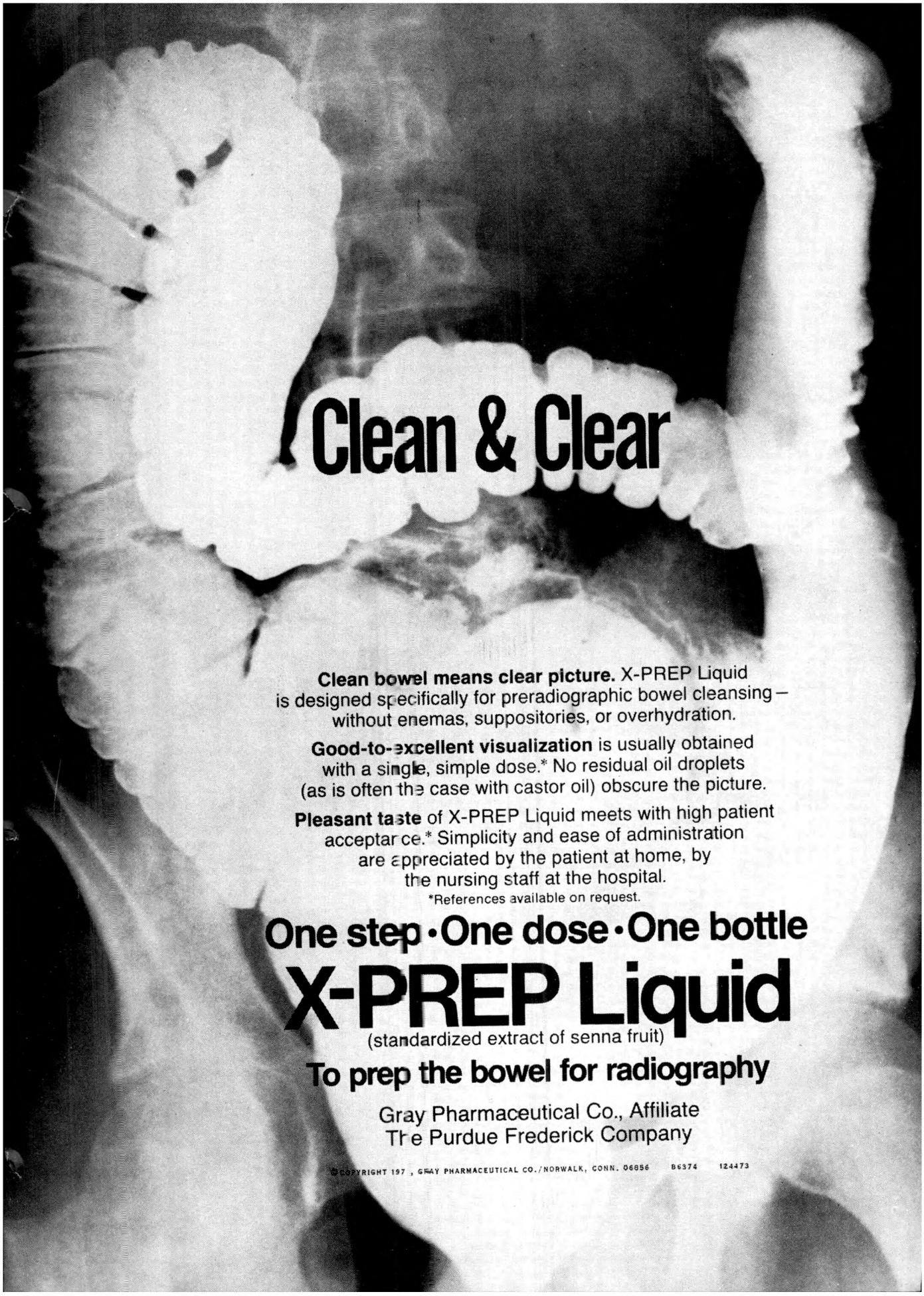
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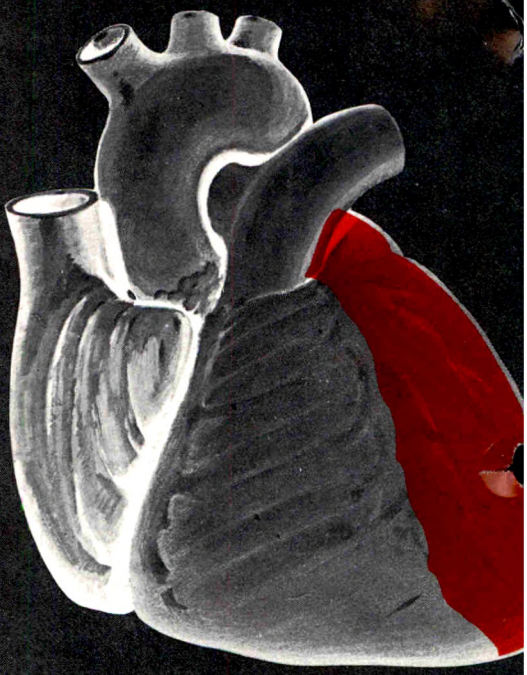
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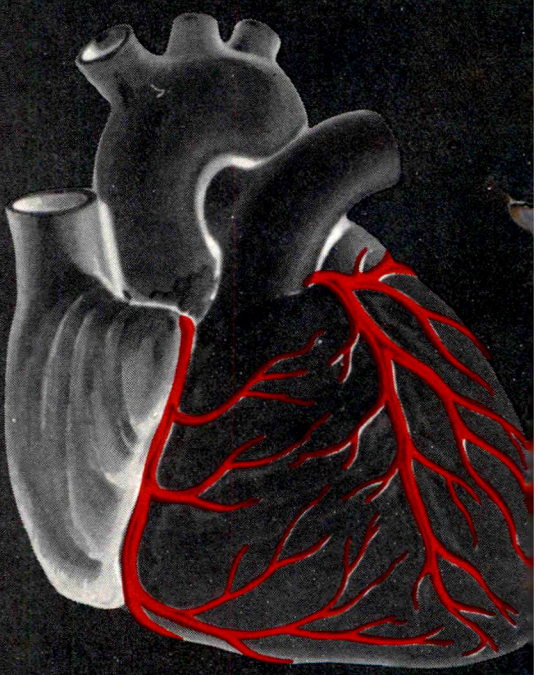
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autions: Diagnostic procedures involving of contrast agents should be performed r the direction of personnel with prereqe training and a thorough knowledge of the cular procedure. Appropriate facilities ld be available for coping with situations h may arise as a result of the procedure for emergency treatment of severe rens to the contrast agent itself; competent onnel and emergency facilities should be able for at least 30 to 60 minutes after I.V. nistration since delayed reactions have known to occur. These severe lifetening reactions suggest hypersensitivity e contrast agent. A personal or family hisof asthma or allergy or a history of a pre- reaction to a contrast agent [illegible]

special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 cc. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated.

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In *excretion urography*, adequate visualization may be difficult or impossible in uremic patients or others with severely impaired renal function (see Contraindications). In *aortography* repeated intra-aortic injections may be hazardous; this also applies to *pediatric angiocardiography* particularly in infants weighing less than 7 kg. (see Warnings). In *peripheral arteriography*, hypotension or moderate decreases in blood pressure seem to occur frequently with intra-arterial (brachial) injections; this is transient and usually requires no treatment. Monitor blood pressure during the immediate 10 minutes after injection. It is recommended that *selective coronary arteriography* not be performed for about 4 weeks after diagnosis of myocardial infarction; mandatory prerequisites to this procedure are experienced personnel, ECG monitoring apparatus, and adequate facilities for immediate resuscitation and cardioversion.

Adverse Reactions: Nausea, vomiting, flushing, or a generalized feeling of warmth are the reactions seen most frequently with intravascular injection. Symptoms which may occur are chills, fever, sweating, headache, dizziness, pallor, weakness, severe retching and choking, wheezing, a rise or fall in blood pressure, facial or conjunctival petechiae, urticaria, pruritus, rash, and other eruptions, edema, cramps, tremors, itching, sneezing, lacrimation, etc. Antihistaminic agents may be of benefit; rarely, such reactions may be severe enough to require discontinuation of dosage. There have been a few reports of a burning or stinging sensation or numbness, of venospasm or venous pain, and of partial collapse of the injected vein. Neutropenia or thrombophlebitis may occur. Severe reactions which may require emergency measures (see Precautions) are a possibility and include cardiovascular reaction characterized by peripheral vasodilatation with hypotension and reflex tachycardia, dyspnea, agitation, confusion, and cyanosis progressing to unconsciousness. An allergic-like reaction ranging from rhinitis or angioneurotic edema to laryngeal or bronchial spasm or anaphylactoid shock may occur. Temporary renal shutdown or other nephropathy may occur.

Adverse reactions as a consequence of *excretion urography* include cardiac arrest, ventricular fibrillation, anaphylaxis with severe asthmatic reaction, and flushing due to generalized vasodilation. Risks of *aortography* procedures include injury to aorta and neighboring organs, pleural puncture, renal damage (including infarction and acute tubular necrosis with oliguria and anuria), accidental selective procedure in presence of preexistent renal disease, retroperitoneal hemorrhage from translumbar approach, spinal cord injury and pathology associated with syndrome of transverse myelitis, generalized petechiae, and death following hypotension, arrhythmia, and anaphylactoid reactions. In *pediatric angiocardiography*, arrhythmia and death have occurred. During *peripheral arteriography*, hemorrhage from puncture site, thrombosis of the vessel, and brachial plexus palsy (following axillary artery injection) have occurred. During *selective coronary arteriography* and *selective coronary arteriography with left ventriculography*, transient ECG changes (most patients); transient arrhythmias (infrequent); ventricular fibrillation (from manipulation of catheter or administration of medium); hypotension; chest pain; myocardial infarction; transient elevation of creatinine phosphokinase (occurred in about 30% of patients tested); fatalities have been reported; hemorrhage, thrombosis, pseudoaneurysms at puncture site, dislodgment of arteriosclerotic plaques, dissection of coronary vessels, and transient sinus arrest have occurred due to the procedure.

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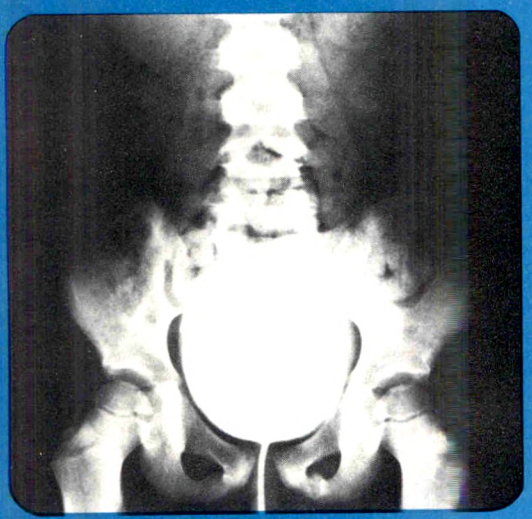
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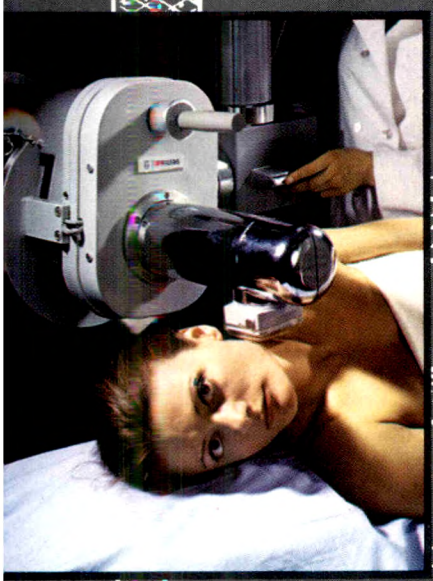
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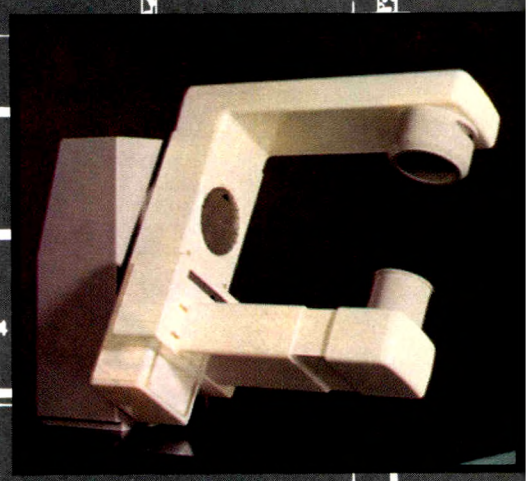
Apart from the megavoltage and ortho-voltage units, the Philips Integrated Therapy Programme includes additional equipment for increased efficiency. The provision of data verification and print-out facilities on mega-voltage units eliminates the possibility of setting errors and reduces the strain on the operator, thereby increasing overall patient safety. In line with the latest interest of the radiotherapist, Philips are now evaluating a Neutron Therapy Unit in clinical use.

If you would like to know more about the Philips Integrated Therapy Programme, please write to: N.V. Philips, Medical Systems, Eindhoven, The Netherlands, or Philips Medical Systems Ltd., The M.E.L. Equipment Company Ltd., Manor Royal, Crawley, Sussex, England.

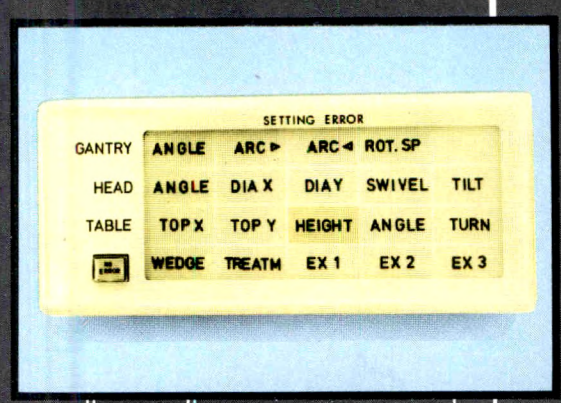








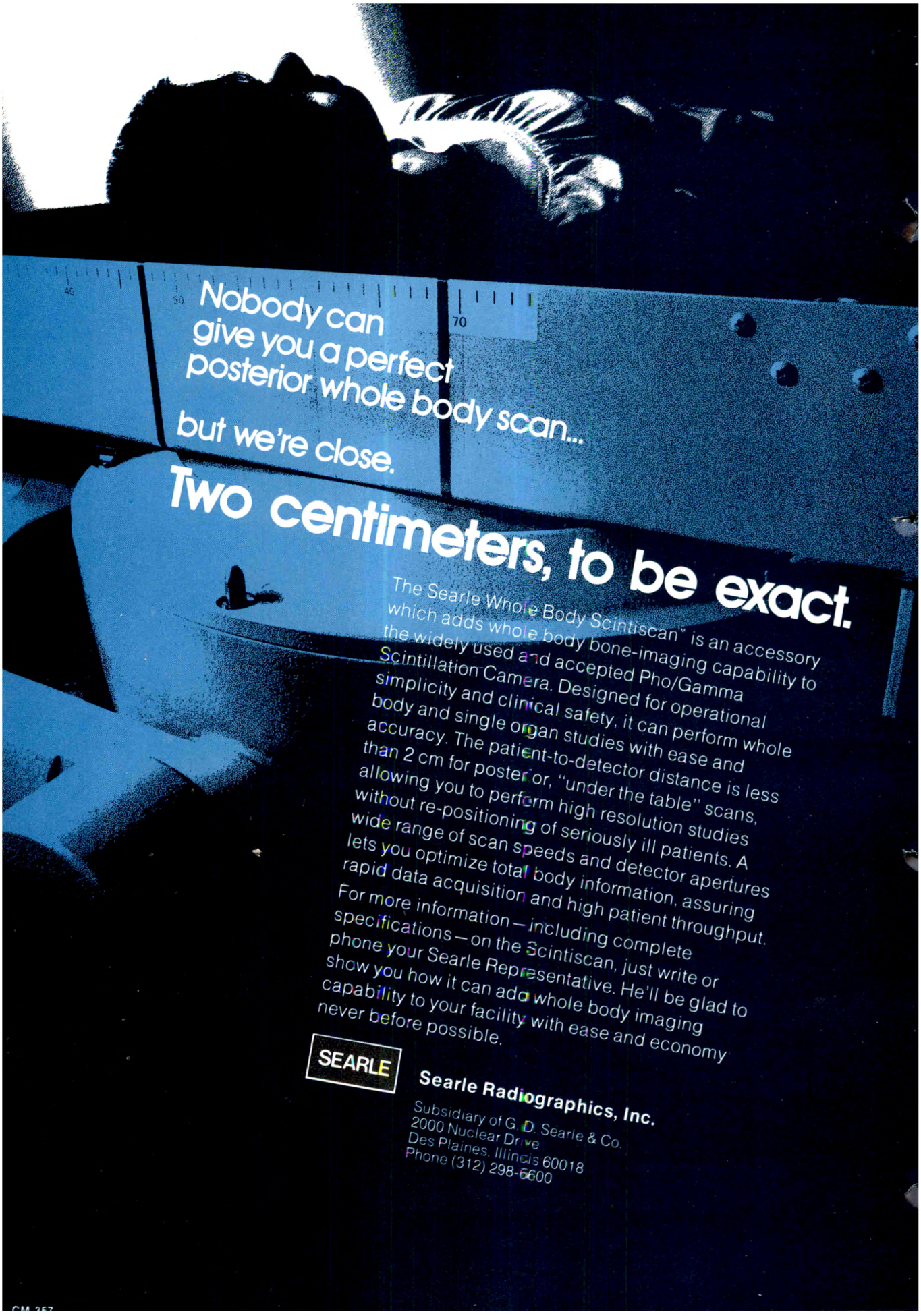




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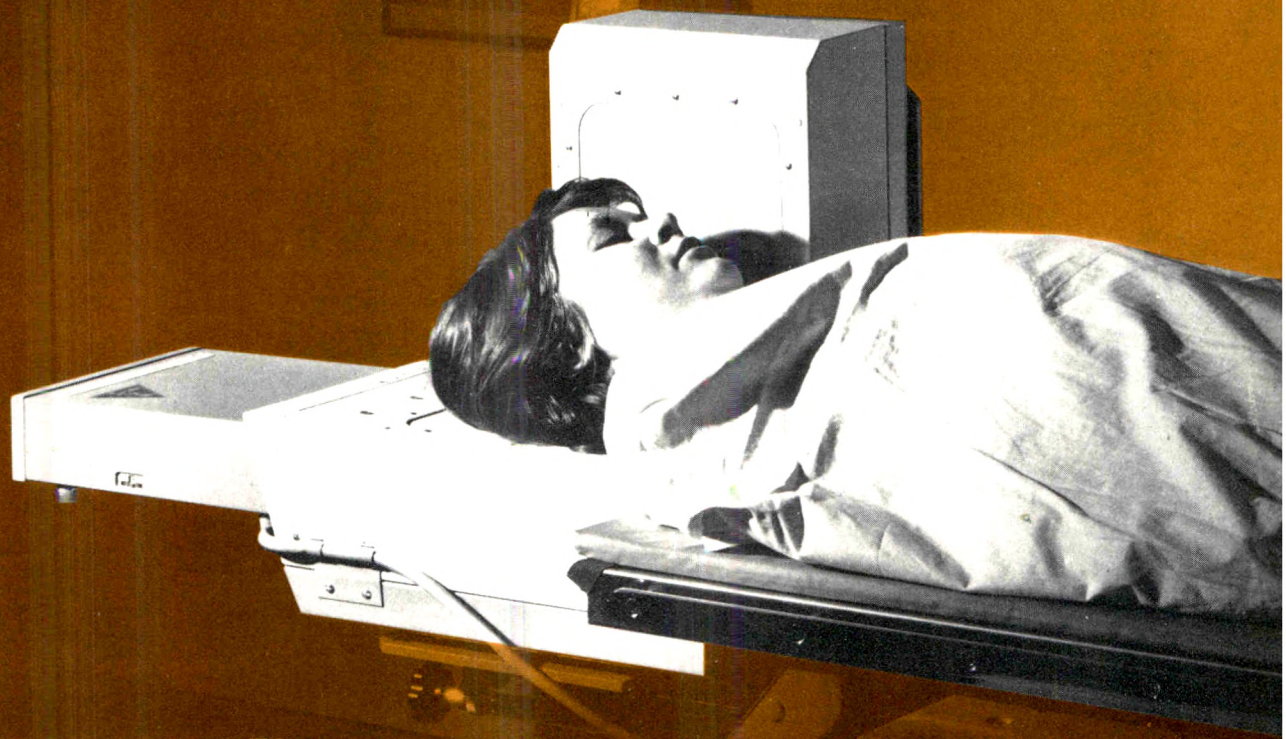
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Alfred S. Ketcham, M.D. . . . . National Cancer Institute
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Robert George Parker, M.D. . . . . University of Washington

NUCLEAR MEDICINE:

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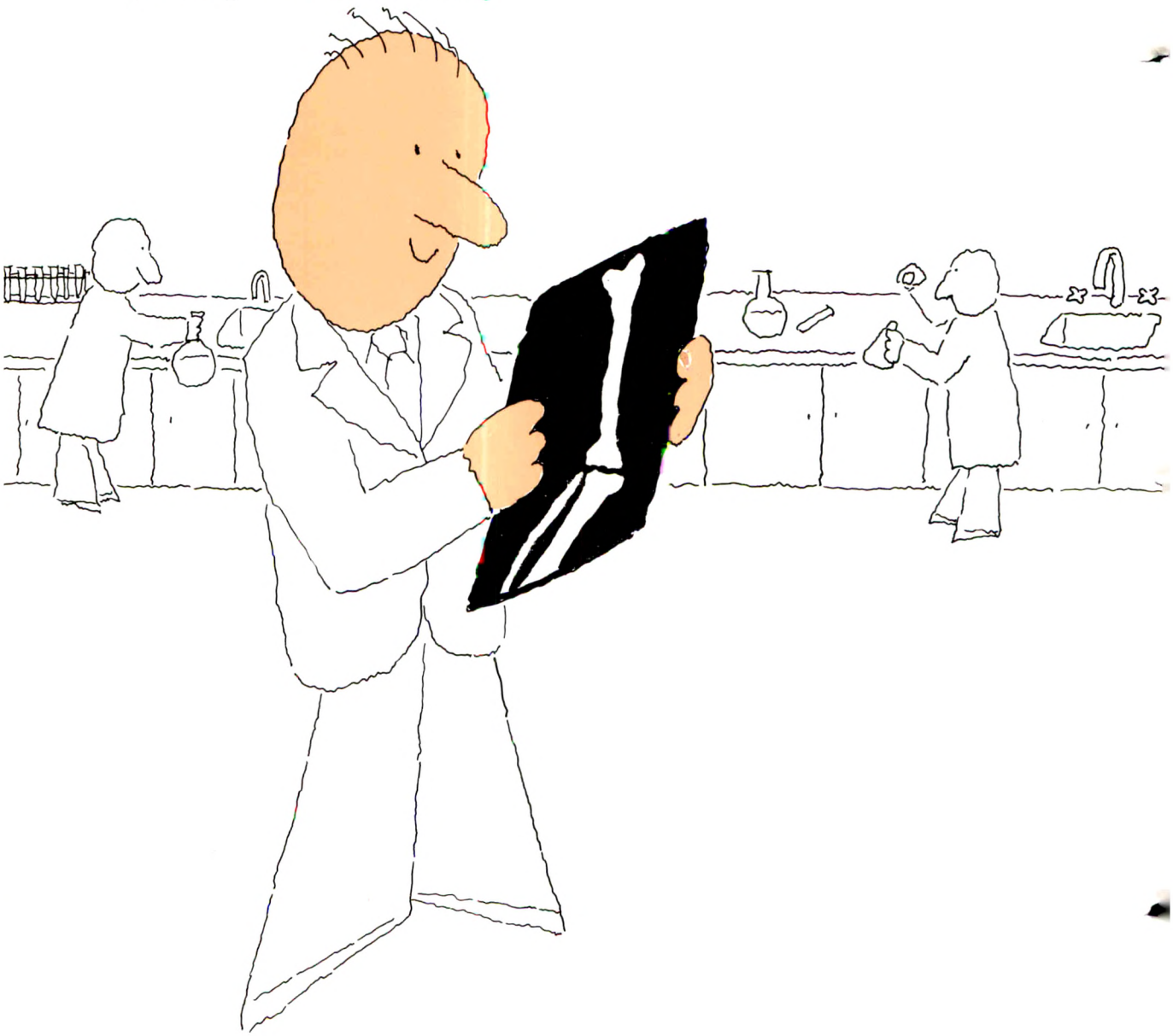
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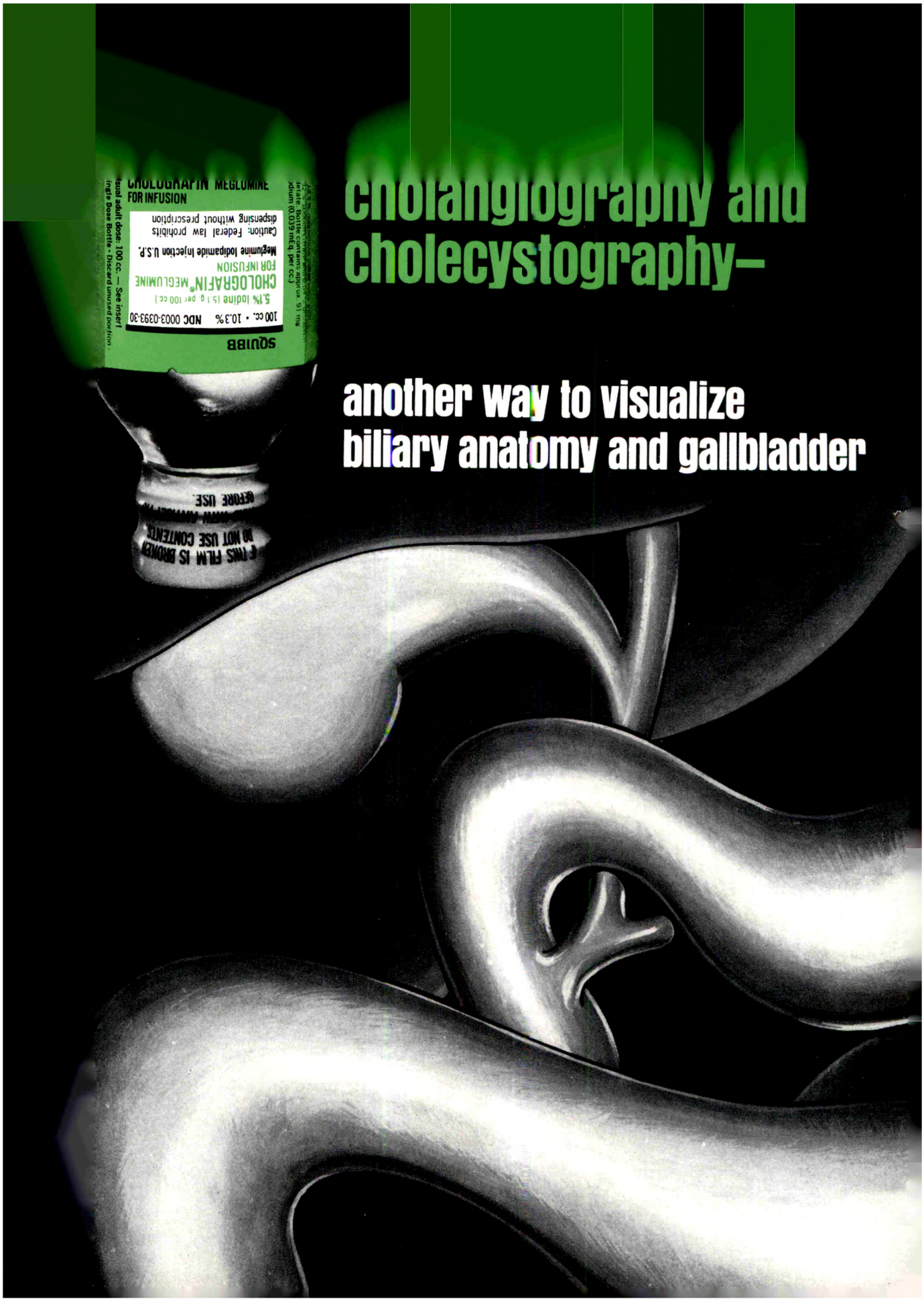
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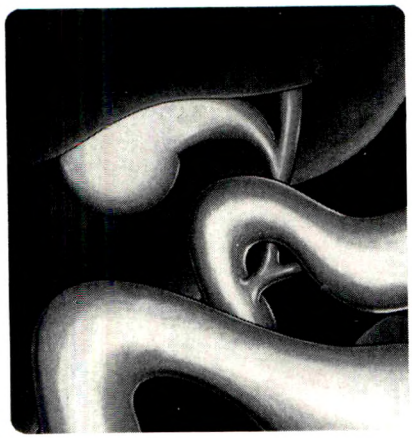
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Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.) is a sterile aqueous solution providing 52% meglumine iodipamide with 0.32% sodium citrate as a buffer and 0.04% disodium edetate as a sequestering agent. The bound iodine content is about 26% (5.2 g./20 ml.). *The solution contains approximately 0.91 mg. (0.039 mEq.) sodium per ml. (18.2 mg./20 ml.).*

CONTRAINDICATIONS: Contraindicated in patients hypersensitive to salts of iodipamide; in patients exhibiting sensitivity reactions to the test dose; in the presence of concomitant severe renal and liver function impairment.

WARNINGS: Intravenous or intra-arterial injections of contrast media have been shown to promote the phenomenon of sickling in persons homozygous for sickle cell disease. Perform thyroid function tests, if indicated, before administration of meglumine iodipamide since iodine-containing contrast media may alter results of such tests. A history of sensitivity to iodine *per se* or to other contrast agents is not an absolute contraindication to the use of meglumine iodipamide but calls for extreme caution in administration.

Usage in Pregnancy: The safety of meglumine iodipamide for use during pregnancy has not been established; therefore, it should be used in pregnant patients only when, in the judgment of the physician, its use is deemed essential to the welfare of the patient.

PRECAUTIONS: Diagnostic procedures involving use of contrast agents should be performed under the direction of personnel with prerequisite training and a thorough knowledge of the particular procedure. Appropriate facilities should be available for coping with situations which may arise as a result of the procedure and for emergency treatment of severe reactions to the contrast agent itself; competent personnel and emergency facilities should be available for at least 30 to 60 minutes after I.V. administration since severe delayed reactions have been known to occur. These severe life-threatening reactions suggest hypersensitivity to the contrast agent. A personal or family history of asthma or allergy or a history of a previous reaction to a contrast agent warrants special attention and may predict more accurately than pretesting the likelihood of a reaction although not the type nor severity of the reaction in the individual. The value of any pretest is questionable. The pretest most performed is the slow I.V. injection of 0.5 to 1.0 ml. of the preparation prior to injection of the full dose; however, the absence of a reaction to the test dose does not preclude the possibility of reaction to the full diagnostic dose. Should the test dose produce an untoward response, the necessity for continuing the examination should be re-evaluated. If deemed essential, examination should proceed with all possible caution. In rare instances, reaction to the test dose may be extremely severe; therefore, close observation and facilities for emergency treatment are indicated. The admixture of Benadryl (Diphenhydramine Hydrochloride) with these meglumine iodipamide preparations may cause a precipitate which may form in the syringe or tubing; if antihistamines are administered concomitantly, they should **not** be mixed with the contrast agent but administered at another site. When the 52% meglumine iodipamide preparation is used, some clinicians feel it may be advisable to have a continuous I.V. infusion running prior to administration of the drug.

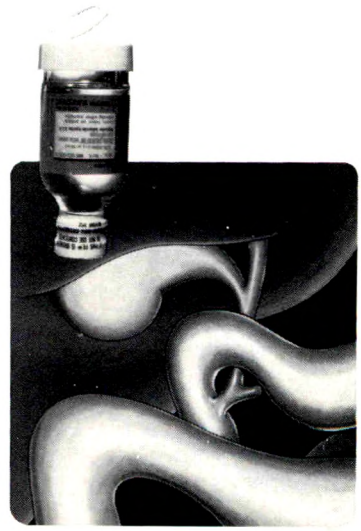
ADVERSE REACTIONS: Local reactions at the site of infusion or injection are not observed unless excessive amounts are extravasated during the infusion or injection. After too rapid administration, mild transient symptoms such as restlessness, sensations of warmth, sneezing, perspiration, salivation, flushing, pressure in the upper abdomen, dizziness, nausea, vomiting, chills, fever, headache, pallor and tremors may occur. These symptoms disappear when the infusion or injection has been completed. Rarely, swollen eyelids, laryngospasm, respiratory difficulties, hypotension, cardiac reactions and cyanosis have been reported. Hypersensitivity reactions may occur. In rare instances, despite the most careful sensitivity testing, anaphylactoid reactions may occur. Renal function tests may be altered and renal failure may occur.

For full information, consult package inserts.

HOW SUPPLIED: Cholografin Meglumine for Infusion (Meglumine Iodipamide Injection U.S.P.)—10.3%—is available in single-dose bottles of 100 ml. Cholografin Meglumine (Meglumine Iodipamide Injection U.S.P.)—52%—is available in single-dose vials of 20 ml.

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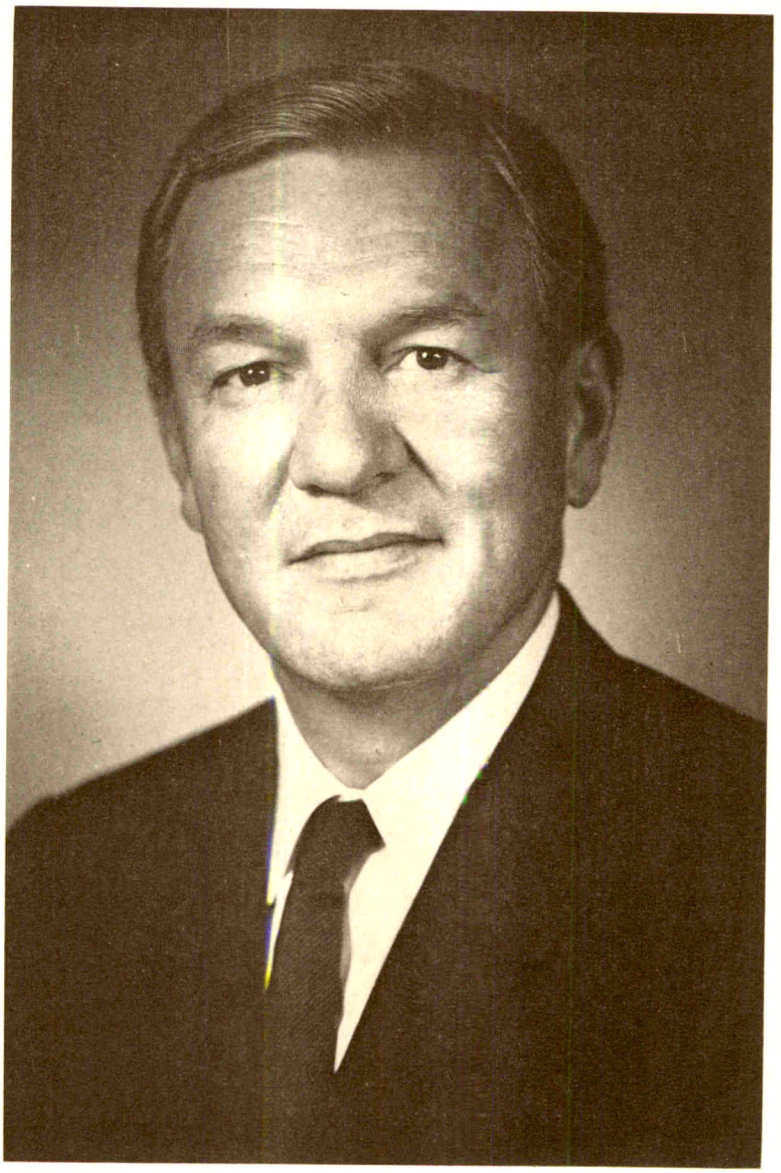
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COLLIN S. MACCARTY, M.D.
CALDWELL LECTURER, 1974

THE AMERICAN JOURNAL OF ROENTGENOLOGY
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VOL. 122 NOVEMBER, 1974 No. 3

INTRODUCTION TO THE CALDWELL LECTURE

By C. ALLEN GOOD M.D.

This lecture to honor Eugene W. Caldwell was first given in 1920, two years after his death. As we continue this tradition, we honor not only Eugene Caldwell, who helped develop the foundations of Radiology, but all physicians and scientists who, at the turn of the century, ripened the science and art of our specialty.

While radiologists predominate among the 53 former Lecturers, there have been more than 20 who brought to the American Roentgen Ray Society information from related branches of science and medicine. Thus, we find among them physicists, anatomists, physiologists, pathologists, clinicians, surgeons and the developer of the hot cathode, high vacuum tube, Dr. W. D. Coolidge.

We are fortunate today to have as the 54th Caldwell Lecturer a man with close ties to Radiology, but who, as a neurosurgeon, can view our specialty from a somewhat different vantage point.

Collin S. MacCarty was born in Rochester, Minnesota on September 20, 1915. He was educated in the local public schools, Dartmouth College, Dartmouth Medical School and Johns Hopkins Medical School. He received the A.B. degree from Dartmouth in 1937 and the M.D. degree from Johns Hopkins in 1940. After a year as surgical intern at Johns Hopkins Hospital, he entered a residency in neurosurgery at Mayo Graduate School of Medicine and was awarded the degree of Master of Science in Neurosurgery by the University of Minnesota in 1944.

Upon completion of his residency, he joined the U. S. Naval Reserve, serving as a neurosurgeon and spending most of his time at the U. S. Naval Hospital in Bethesda, Maryland. During the last six months of this assignment, he was Chief of the Neurosurgical Service.

When his military duties were over, he joined the Neurosurgical Staff of Mayo Clinic in October 1946 and became Chairman of that department in October 1963, a position he still holds. He is also Professor of Neurosurgery at Mayo Medical School.

As would be expected of a dedicated surgeon and scientist who has found the time to practice his specialty, help educate his residents, administer a large department and publish, as author or co-author, two books and more than 130 articles, he has received many honors. Some of the more important are the presidency of the Neurosurgical Society of America, the presidency of the American Association of Neurological Surgeons (formerly the Harvey Cushing

Society) and the presidency of the Medical Staff of Mayo Clinic. He is currently serving on the Board of Overseers of Dartmouth Medical School and on the American Board of Neurological Surgery.

Early in my own residency at Mayo, I had the privilege of spending three months in Surgical Pathology under Dr. William Carpenter MacCarty, our speaker's father. It was a most rewarding experience, and my introduction to the MacCarty family. Later I was to enjoy working with our speaker's elder brother, William C. MacCarty, Jr., who is a radiologist at Mary Hitchcock Hospital in Hanover, New Hampshire and associated with Dartmouth Medical School. Finally, I have had the opportunity to take some small part in the radiologic education of our speaker's second son, Robert, who has recently become a member of the Department of Diagnostic Roentgenology of Mayo Clinic. Thus I have known Collin MacCarty and his family for almost 40 years. This should give me license to tell you something he may not know. When I was studying Surgical Pathology, his father confided that he had been experimenting with a Crookes' tube at the University of Kentucky at the time of the announcement of Roentgen's discovery. He said to me, "If that old boy had been a little slower in his work, it might have been called the MacCarty Ray, not the Roentgen Ray!"

It is now my privilege and honor to present to you Dr. Collin S. MacCarty, who has chosen as the title of his Caldwell Lecture, "A Significant Symbiotic Relationship."

Vol. 122, No. 3

A SIGNIFICANT SYMBIOTIC RELATIONSHIP*

CALDWELL LECTURE, 1974

By COLLIN S. MacCARTY, M.D.
ROCHESTER, MINNESOTA

PRESTON HICKEY[13] delivered the 1928 Caldwell Lecture. In his introduction he stated, "In recalling the names of those notables who have given the preceding lectures, I felt some timidity in accepting the invitation, but when I recollected that probably those who would be in the audience this evening might in their friendship not indulge in too critical comparisons, I felt emboldened to accept."

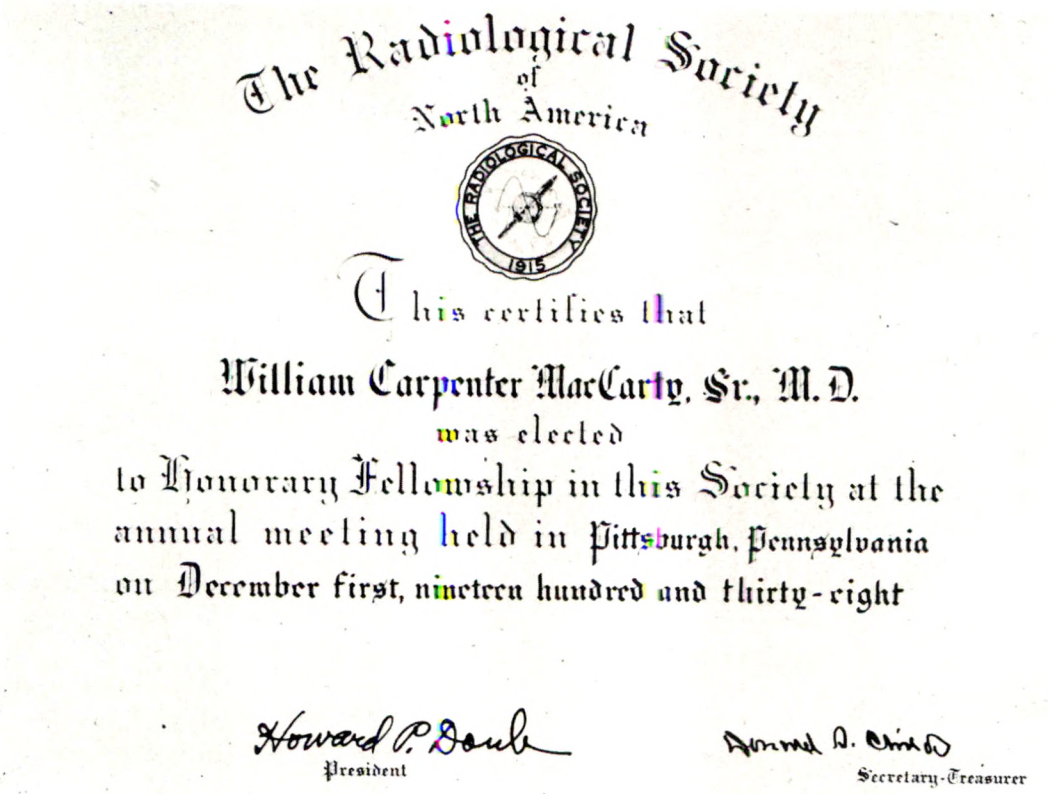
Since then 45 men have appeared before the Annual Meeting of the American Roentgen Ray Society to deliver this Lecture, named in honor of one of the distinguished pioneers in American roentgenology. I accepted this honor with humility and apprehension. It was a revelation to review the names of my predecessors. Some were former friends and colleagues: Walter Alvarez with his distinguished family lived in the block next to us. His son, Luis, recently was awarded the Nobel Prize in physics. The Kirklins, father and son; Allen Good; Jay Bargen, people whom I have known for many years, were others.

At the outset, I should like to indulge in some personal comments to express my particular interest in, and admiration for, your discipline. In 1938 my father (Fig. 1), a surgical pathologist, was made an honorary member of the Radiological Society of North America. My brother is a radiologist, and my son has just been appointed a member of the Department of Radiology at our institution, and he presented his first formal paper before you in Montreal last year. Finally, my colleague, one of my most cherished friends, is your President.

Neurologic surgery has a similar lectureship named for Harvey Cushing (Fig. 2), whom many consider to be the father of American neurosurgery as well as a distinguished physician, scientist and author. He was awarded a Pulitzer Prize for his biography of Sir William Osler. Cushing's interest in roentgenography began in 1896.[25] Walter James Dodd, the assistant pharmacist at the Massachusetts General Hospital, made a tube for the hand-driven static machine which that hospital had purchased after Roentgen's discovery was announced in Germany in 1895. Dr. Cushing wished to use the tube in the outpatient department of that hospital, but he was denied that opportunity. He, therefore, bought one of his own and brought it to Baltimore in September 1896. There he made the first roentgenogram ever seen at Hopkins; it disclosed and localized a bullet lodged in the spinal column of a woman. Until 1897 Cushing did all the x-ray work at Johns Hopkins. And here, incidentally, we have a neat if not unique parallel: only one neurosurgeon has delivered the Cushing Oration: Dr. Wilder Penfield (Fig. 3), and only one neurosurgeon has delivered the Caldwell Lecture: Dr. Wilder Penfield. To have been asked to join such company was as much recognition as any man could expect in his lifetime.

After reviewing Hickey's lecture,[13] I perceived that Eugene W. Caldwell was indeed one of the most illustrious of the early workers in the first two decades of American roentgenology. He was born in Savannah, Missouri, in 1870. (Parenthetically, it is of more than passing interest that Dr. Walter Dandy, the superb Hopkins neurosurgeon, was also a Missourian, having been born in Sedalia in 1886.) Caldwell was graduated from the University of Kansas in electrical engineering. He worked for the

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.
From the Department of Neurologic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.


The Radiological Society
of
North America

THE RADIOLOGICAL SOCIETY 1915

This certifies that

William Carpenter MacCarty, Sr., M.D.

was elected

to Honorary Fellowship in this Society at the annual meeting held in Pittsburgh, Pennsylvania on December first, nineteen hundred and thirty-eight

Howard P. Doub
President

Secretary-Treasurer

FIG. 1. Dr. W. C. MacCarty, the author's father, received honorary fellowship in the Radiological Society of North America in 1938 after delivering the Carman Lecture.

New York Telephone Company and during that time he became interested in roentgen rays, which led him to establish the first x-ray office in New York in 1897. He then became aware of the necessity to study medicine, which he did, and he received his medical degree from the Bellevue Medical School and Hospital. Caldwell's interests covered the entire field of radiology, from therapeutic to diagnostic. In 1902 he published an article in the *New York Medical Journal* entitled "A New Apparatus for the Therapeutic Application of the X-Ray to the Larynx, Tongue, Rectum, Prostate, Cervix of the Uterus, Vagina, etc."[13] Indeed, he was a man of many interests! In 1904 he developed a portable x-ray machine. The same year he wrote: "There is a tendency among medical men either to under estimate the value of the x-ray as an aid to diagnosis or to fail to appreciate its limitations."[13] Caldwell may, in fact, have pioneered neuroradiology, for in 1906, before this society, he described the value of the roentgen rays in the study of the anatomy of the nasal accessory sinuses. In 1910 he and Long concluded that there was no relationship between the degree of carpal development and the quality of the mind. Clark and Caldwell reached no decisive conclusion in a paper entitled "The Sella Turcica in Epilepsy."[13] These at least are contributions to a field if by no other criterion than exclusion.

I liked his obvious sense of humor and insight as displayed in such remarks as "The AP projection required a long exposure and is the one in which the operator is most likely to ruin his best tube and to lose that cheerful but dignified composure that should always characterize the medical man."

In 1917 he "emphasized the difficulty of obtaining reliable statistics of cases examined from a large number of physicians and surgeons owing to their incomplete records," and remarked on looking over the replies to his questionnaire, that the answers to these questions were purely suggestive and seemed to depend somewhat upon the weather or the state of digestion of the person answering."[13] In discussing the roentgen diagnosis of gallstones he again pointed out "that this difference in statistics is due to the temperament of the diagnostician as well as his skilled judgement" and in speaking of the right upper quadrant he remarked, "It is in this danger zone that the number of killed and wounded resulting from faulty interpretation is greatest."

Caldwell was a fine teacher of the art. He

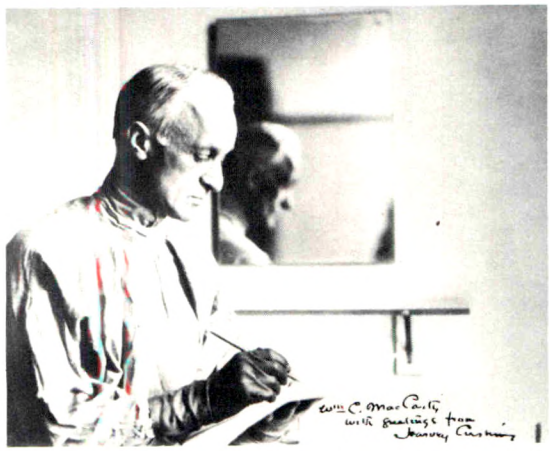


Fig. 2. Dr. Harvey Cushing, pioneer neurosurgeon, who made the first roentgenograms at Johns Hopkins Hospital. (From MacCarty, C. S. Past personalities and present problems. [The 1971 AANS presidential address.] *J. Neurosurg.*, 1971, *35*, 633–645. By permission of American Association of Neurological Surgeons.)



Fig. 3. Dr. Wilder Penfield delivered the Annual Cushing Oration in 1971 and the Caldwell Lecture in 1951.



FIG. 4. Dr. W. M. Craig and Dr. Walter Dandy during World War II. Dr. Dandy introduced ventriculography in 1918 and pneumography in 1919. (From MacCarty, C. S. Past personalities and present problems. [The 1971 AANS presidential address.] *J. Neurosurg.*, 1971, *35*, 633–645. By permission of American Association of Neurological Surgeons.)

emphasized "the first principle of roentgen diagnosis, namely that the roentgenogram is not a picture but a special kind of projection and must be interpreted by one competent to so do." Of the roentgenologist he said: "His opinion should be valued rather than his so called x-ray pictures." Finally not long before his premature demise on June 23, 1918, Caldwell's "stereoscopic fluoroscope was rapidly approaching a successful termination."[13]

NEURORADIOLOGY

One gorgeous Saturday in September 1940, in Baltimore, Dr. Dandy (Fig. 4) invited his "brain team" to the baseball game.[26] I remember how it seemed inconceivable that we could finish the "list" by game time, nor could we imagine who would "cover" the patients when the whole "outfit" was to be at the Orioles' ball park. But there was no real concern because Dandy was one of the most skilled surgeons who operated with incredible speed, and his senior and assistant residents were approaching his ability. I was the intern. Those were the days of the "Halsted training program," which meant that all residents in surgery spent 2 years with Dandy. This was the basis on which Dandy ordered Ken Pickrell, who was 1 year beyond his neurosurgical experience, to cover all our duties. Dandy was a physician and surgeon of "the old school," believing in the hierarchic order of medical teaching and authority. As a consequence, when we reached the ball park, each resident received two hot dogs and "the boy"—the intern—received only one.

Neuroradiology probably dates from the time when Dandy described ventriculography.[7,22–24,27] According to Campbell,[27] "On January 3, 1917, a patient of Dr. Dandy's 'whose abdomen he was about to explore for intestinal perforation chanced to have a chest x-ray taken on the way to the operating room. Air was clearly visible under the diaphragm.' . . . this was the very roentgenogram that originally suggested to Dandy the use of air to outline the cerebral ventricles." The following year (1918) his classic paper appeared in the *Annals of Surgery*, and in 1919[7,22–24,27] he described the injection of air into the lumbar subarachnoid space to visualize the intracranial structures as well as the upper cervical part of the spinal canal. He suggested in that paper the injection of air into the subarachnoid space as an adjunct to the diagnosis of tumors of the spinal cord, in addition to its use in studying the intracranial spaces.

I am clearly suggesting to you that a neurosurgeon initiated a subspecialty of diagnostic roentgenology, and you have been unable to rid yourselves of us through these many years. Recent developments may, however, alter our relationship and, let us hope, in continued harmony.

Although Dandy did describe visualization of the spinal subarachnoid space with the aid of air, he was aware of its limitations in respect to the accuracy of outlining spinal cord tumors.

Radiopaque materials were sought to define the spinal canal visually, but none of those proposed were safe and practical until Lipiodol was introduced by Sicard and Forestier[8] in 1921. Initially, this medium was used to outline the epidural

space, but when the substance was inadvertently introduced into the subarachnoid space by these men it was seen at once that new vistas in the diagnosis of disease of the spinal cord were at hand. They presented these initial experiences with subarachnoid myelography in 1922.[8] Sicard, basically a neurologist, was born in 1872, and he died in Paris on January 28, 1929. His student, Jacques Forestier, was born in 1890 and following their collaborative work with Lipiodol myelography, he transferred his interests to rheumatology. In 1929 he introduced the use of gold salts in the treatment of rheumatoid arthritis. Both men have been properly recognized by your profession. Forestier was made an honorary fellow of the American College of Radiology.

Dr. W. Jason Mixter, a neurosurgeon at the Massachusetts General Hospital, through his friendship with Sicard, received some Lipiodol before the days of mechanical tables, and he administered it *via* the cisterna magna.[8] Dr. John D. Camp's design of the tilting table further facilitated the technique of myelography.[8] The irritating effects of Lipiodol were recognized by the early investigators. Removal of the oil by the lumbar route was adopted following the suggestion of Kubik and Hampton[8] in 1941, although this possibility initially had been suggested by Sicard and Forestier, long previously! In 1944 ethyl iodophenylundecylate (Pantopaque) came into general clinical use, supplanting Lipiodol as a contrast medium for study of the spinal subarachnoid space and, in recent years, the posterior fossa.

Egas Moniz (Fig. 5) is a name familiar to most physicians. He was born Antonio Caetano deAbreu Freire, in Avanca, a small village in northern Portugal, in 1874. He adopted the pen name of Egas Moniz, a hero of the Portuguese resistance against the Moors in the Middle Ages, during his student days at Coimbra, where he wrote political pamphlets. Moniz studied under Babinski, Pierre Marie and Dejerine. He became professor of neurology in 1911 at the University of Lisbon when that school was reorganized. In addition to his medical work, he continued his interest in politics, becoming minister of foreign affairs and ambassador to Spain. After World War I he led the Portuguese delegation to the Versailles peace conference.[4,9]



Fig. 5. Dr. Egas Moniz, neurologist and diplomat, developed cerebral angiography in 1927.

Moniz' epic paper, "Arterial Encephalography: Importance in the Localization of Cerebral Tumors," was published in July 1927.[9] It is of considerable interest that in his initial attempts at cerebral angiography he used the percutaneous route and a 70 per cent solution of strontium bromide. Because he was not successful in direct puncture of the carotid arteries, Moniz called on his surgical colleagues to expose the vessels in the cervical area. One of these men, Professor Almeida Lima (Fig. 6), continued to collaborate with Moniz during Moniz' life-

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Fig. 6. Dr. Almeida Lima, Dr. Moniz' surgical colleague.

time until his death in 1955. Lima participated in the development of prefrontal lobotomy for which Moniz was awarded the Nobel Prize in Physiology and Medicine in 1949.[9] I attended the 40th anniversary celebration of the development of cerebral angiography at the Egas Moniz Institute of the University of Lisbon, and I recall vividly an exhibit of important contributions to the technique of angiography from its inception in 1927. Permit me to embarrass your President by mentioning that one milestone depicted was the electronic subtraction technique developed by Colin Holman.[14]

Dandy, Sicard, Forestier, Mixter, Moniz, Lima and others from nonradiologic specialties have cooperated with radiologists with the result that an exciting and important new specialty has developed, with horizons which would seem almost limitless.

I should like briefly to review the current state of the art as observed from the vantage point of this neurologic surgeon.

Ventriculography is rarely a primary diagnostic radiographic procedure. Usually it is preceded by radioisotope scanning, the new computerized axial tomography and arteriography. If these procedures do not disclose what is suspected, but do reveal evidence of obstructive hydrocephalus or marked increased intracranial pressure, the use of ventriculography is indicated to establish or rule out the presence of a surgical lesion. In the newborn, anterior fontanelle ventriculography and ventricular estimation remain useful tools in the diagnosis of communicating or obstructive hydrocephalus and in identifying subdural hematomas.

Pneumoencephalography likewise is rarely a primary test, but it is frequently preceded by radioisotope scanning, subarachnoid radioiodinated serum albumen studies, angiography and computerized axial tomography. Pneumography should not be performed in the presence of obviously increased intracranial pressure.

ANGIOGRAPHY

Since World War II angiography has become a most valuable adjunct to discovering the causes of subarachnoid hemorrhage, localizing brain tumors, and identifying the nature of brain tumors and the causes of cerebral arterial and venous diseases. With the advent of magnification and subtraction techniques, it may be that angiography has approached the epitome of its capabilities. These refinements have allowed the experts to identify colloid cysts of the third ventricle (Fig. 7), heretofore recognizable[15,16] only by ventriculography or pneumography, and then at some increased risk. Possibly even more dramatic is the ability of the angiographer to identify microadenomas[19] of the adenohypophysis (Fig. 8) smaller than 10 mm. in diameter. As a consequence, the diagnosis of functioning adenomas can be made at an early stage, the lesions can be approached by the transsphenoidal route and removed, the defect

is thus corrected and normal pituitary function can be preserved on many occasions.

Angiography of the spinal arteries and veins, developed by neuroradiologists particularly in France[12] and in a few centers elsewhere,[10,11] is in its infancy. The study of vascular malformations of the spinal cord has encouraged neurosurgeons to operate on these challenging lesions with better results (Fig. 9). Identification of tumors of the spinal cord by this technique in our institution has not yet attained the accuracy obtained for cerebral angiography.

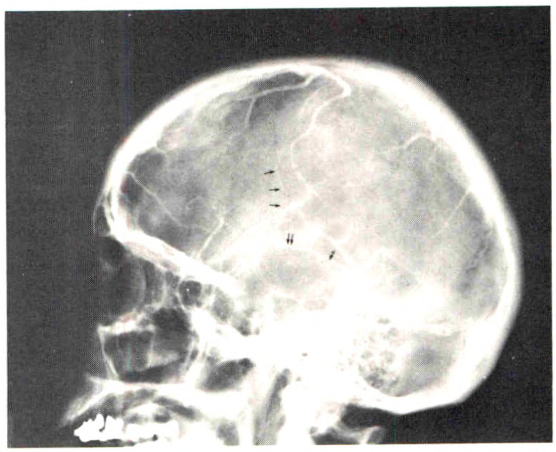


Fig. 7. The venous phase of the angiogram reveals elevation of the origin of the internal cerebral vein, the deformity being caused by a colloid cyst of the third ventricle.

Myelography and Rhombencephalography

The usefulness of instilling positive contrast material into the spinal subarachnoid space, the posterior fossa[5,20] and its cavities is acknowledged. One must not, however, perform such tests unless facilities are at hand for the immediate performance of surgical treatment. We still need to be reminded of this periodically, for in recent years we have had a patient become paraparetic in the postanesthesia room awaiting operation only a short time after myelography revealed a high degree of obstruction in the cervicothoracic area from an intramedullary cyst.

Tumors of the cerebellopontine angle can be accurately identified and operated upon before they have enlarged[5,20] to produce the classic syndrome, meaning that the surgeon is enabled to remove the tumors with less consequent morbidity, particularly with respect to the facial nerve. More and more often, tumors of the foramen magnum are being distinguished from diseases such as multiple sclerosis, particularly those tumors lying posteriorly, by the simple expedient of performance of the test with the patient lying on his back.

In the experience of some, direct puncture of cystic lesions of the spinal cord and instillation of contrast medium have been informative.[21] I am not enthusiastic about the value of this technique. Cysts of the spinal cord constitute a topic provocative of extended discussion, so much so that it would require more time than this entire lecture is allotted.

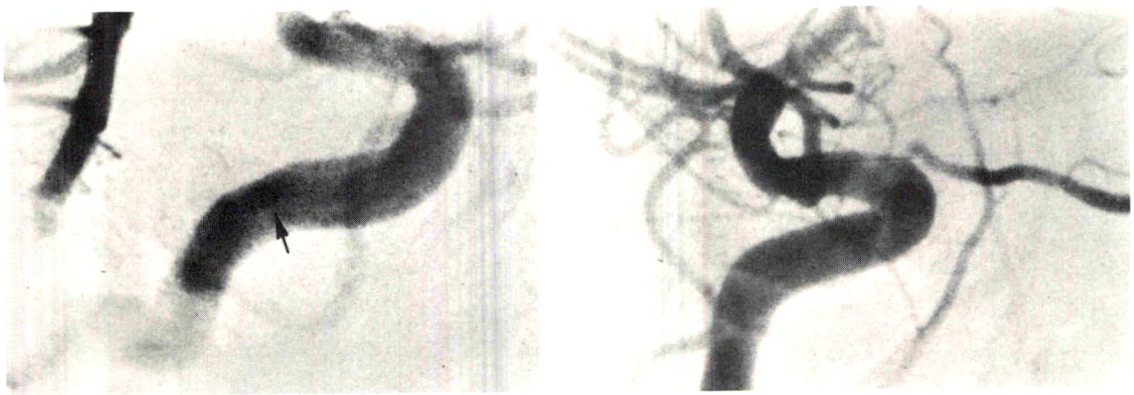


Fig. 8. Microadenoma of the pituitary visualized by angiography.

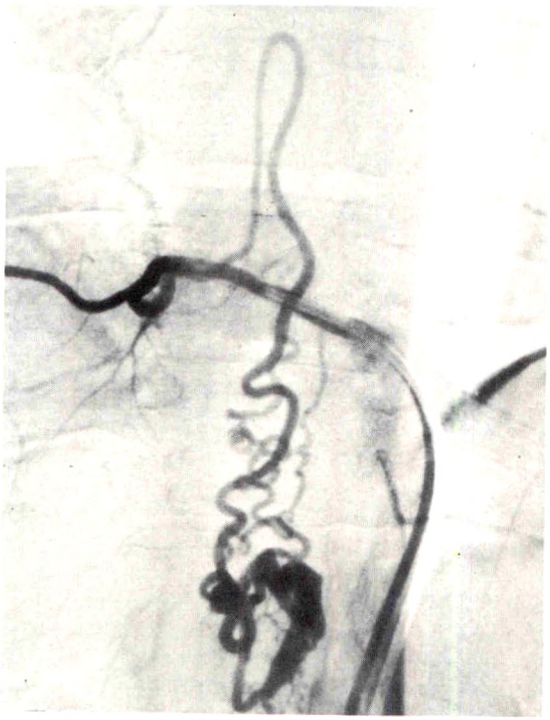


FIG. 9. The angiogram displays an arteriovenous malformation fed by the artery of Adamkiewicz.

COMPUTERIZED AXIAL TOMOGRAPHY

Had the ingenious Caldwell been of our generation, he might well have developed computerized axial tomography, because of his background in both engineering and radiology. Hounsfield, an engineer with E.M.I., Ltd., described the machine in the *New Scientist* in 1972. He collaborated with Ambrose, from the Radiology Department of Atkinson Morley's Hospital, Wimbledon, London, in the preparation of a paper on use of the apparatus in the study of intracerebral diseases which was presented in 1972 before the November meeting of the Radiologic Society of North America.[18] Their initial publication appeared in 1973.[2,3] The second machine was installed by the Mayo Clinic and was in operation on June 20, 1973, in the radiology section of St. Marys Hospital. It has been enthusiastically endorsed by members of the Department of Neurologic Surgery during the first year of its operation. A second has been installed. I find myself in no position to talk to you about the physics and computer calculations of this magnificent device. I will say simply that I am astounded when I consider that two detector crystals, each monitoring half of an x-ray beam and remaining in alignment with the x-ray source taking 160 readings of photon transmission through the head with each traverse, and 180 such traverses made varying 1° with each scan, will result in 28,800 readings'[6] having been taken by each detector on the completion of a test. The computer then processes these readings. It calculates 6,400 absorption values within each two adjacent slices of cranial tissue 1.3 cm. in thickness. The results are displayed as a digital print-out and as a cathode-ray tube picture. The brighter the area of print-out (being proportional to its absorption coefficient), the more dense is the tissue. Bone is most dense and gray matter is next most dense; white matter and the fluid in the ventricles and subarachnoid spaces are the least dense components in the normal cranial compartment. Interpretation of the results of such study is dependent on analysis of the cathode-ray display as well as on the digital print-out of the density and atomic numbers of the tissue in question.

Baker and associates[6] reviewed our experience in such studies with our first 500 patients. We examine 15 to 16 patients daily. By orienting the plane of the slices 20 to 25° toward the feet in relation to the orbitomeatal line, we have been able to include 85 to 90 per cent of the intracranial neural structures in the study, including particularly the contents of the posterior fossa. If lesions of the base or vertex are suspected, additional alterations can be made. The initial experience of Baker and his colleagues[6] with this new method produced a percentage of error of 3.6, which made the procedure second only to pneumography in accuracy of diagnosis, and "the error rate was slightly more than one-half of that observed in cerebral angiographic diagnosis." The percentage of error inherent in isotope brain-scanning in our institution is not comparable to that of other methods.

Baker and his colleagues found that a high diagnostic correlation was obtained in the presence of many clinically suspected conditions, "such as hydrocephalus (92 per cent), metastasis (69 per cent), dementia or degenerative states (67 per cent), suspected mass lesions (62 per cent) and infarctions (48 per cent)." They also pointed out that the "more striking indication of the value of C.A.T., however, was evident in those instances when an unanticipated condition was revealed. A tumor or parenchymatous lesion was discovered in 25 per cent of patients suspected of having multiple sclerosis, 21 per cent of those thought to have infarcts, 20 per cent of those with seizure disorders, 17 per cent of those with indeterminate focal cerebral dysfunction, 8 per cent of those with headache and 7 per cent of those with dementia."

Presenile dementia may be caused by occult hydrocephalus,[1] infarction or mass lesions. Surgical attack has something to offer because it makes possible shunting of the occult hydrocephalus or removal of the responsible neoplasm. Computerized axial tomography can accurately detect tumors and identify hydrocephalus and cerebral atrophy. It would seem that in the near future pneumography, angiography, isotope brain-scanning and cisternal radioiodinated serum albumin studies may be entirely eliminated in the case of persons with dementia!

Because of the variability of densities of brain tumors, we anticipate continually improved accuracy in interpretation of the EMI scan. Our experience would suggest that the neuroradiologist is able to tell the neurosurgeon whether the lesion at hand is a meningioma, cystic astrocytoma, solid astrocytoma, glioblastoma multiforme, Rathke pouch cyst, colloid cyst, pinealoma, hemangioendothelioma, metastatic tumor, or acoustic neurilemmoma (Fig. 10).

Appraisal of the role of computerized axial tomography in the management of trauma to the head is rapidly being resolved. Cerebral edema can be detected, as can epidural or subdural and intracerebral hematomas. "The costly night-time emergency neuroradiologic special study may be disappearing from the scene."[6]

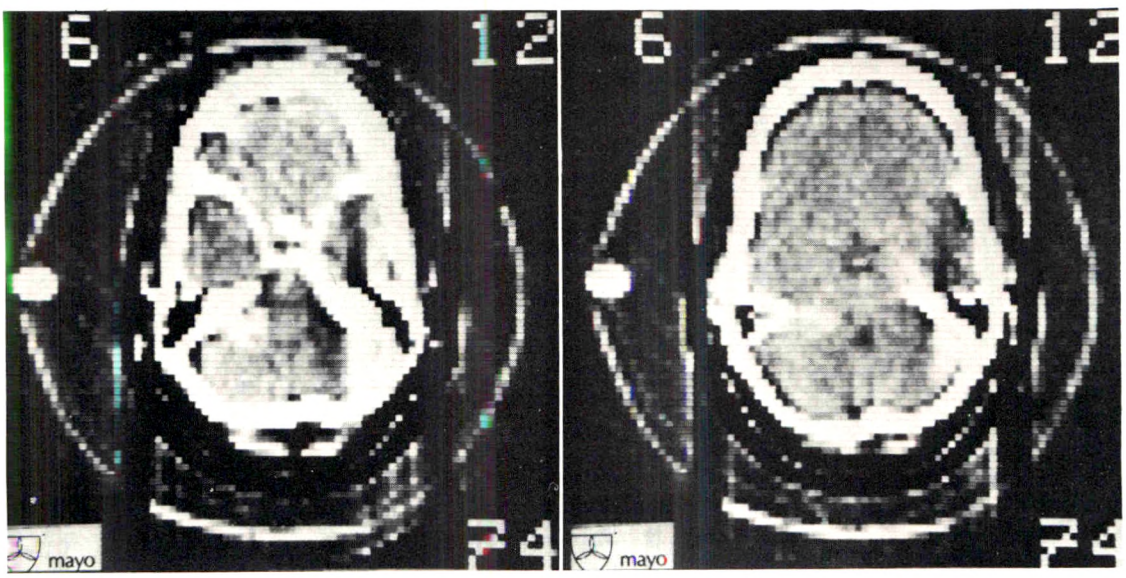


FIG. 10. Positive contrast enhanced computerized axial tomography displaying a left acoustic neuroma.

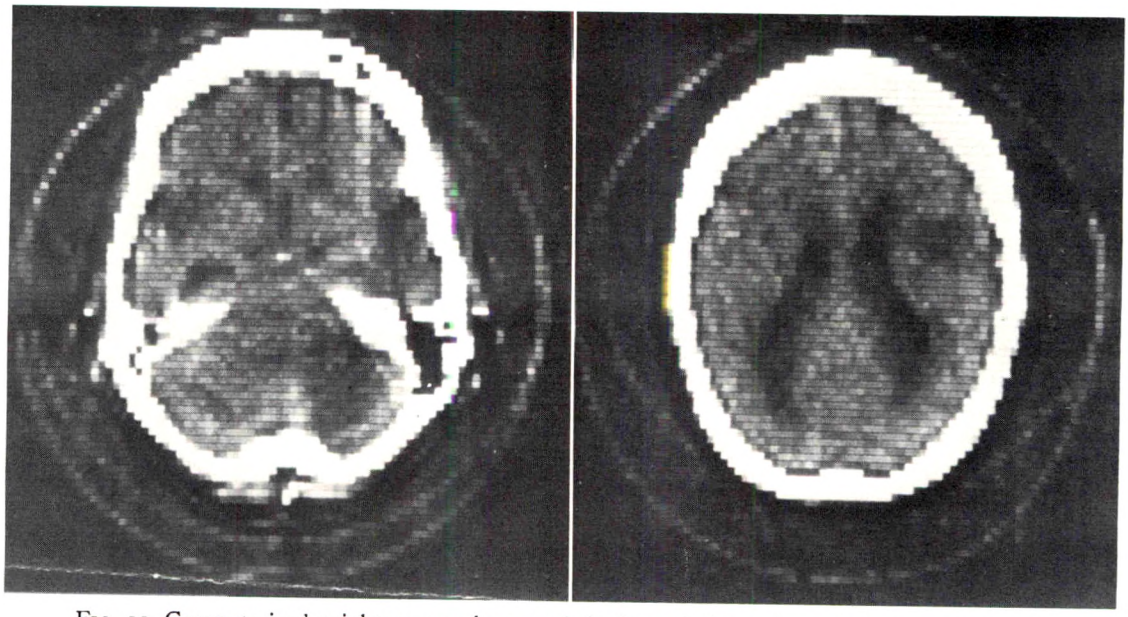


Fig. 11. Computerized axial tomography reveals hydrocephalus and a radiolucent area in the right cerebellar hemisphere, the right temporal region and possibly the left hemisphere.

One of the most promising aspects of this equipment is its application in following the course of brain disease. It can be used to distinguish a postoperative blood clot from postoperative cerebral or cerebellar edema. The effectiveness of dexamethasone is graphically displayed by serial scans as in the case of this elderly gentleman who was suffering from cerebral metastasis of a large-cell carcinoma of the lung. The initial scan (Fig. 11) revealed apparently multiple metastatic processes as represented by areas of decreased density in the cerebellum and cerebrum. Therapy with dexamethasone resulted in rapid clinical improvement and reduction of ventricular size and resolution of the lesion as exhibited in the EMI scan (Fig. 12). As the use of steroids was reduced or discontinued, symptoms again occurred and again the pattern described by the scan deteriorated (Fig. 13).

I venture to suggest that not many of you know how long it takes for a subacute hydrocephalus secondary to a cerebellar tumor to resolve after removal of the tumor. A 42 year old woman's preoperative scan (Fig. 14) revealed a rather marked obstructive hydrocephalus secondary to a cystic hemangioendothelioma of the right cerebellar hemisphere. Within the week—6 days to be exact—after operation her ventricles had returned to normal size (Fig. 14).

Computerized axial tomography has a high degree of accuracy in the diagnosis of spontaneous intracerebral hematomas. Still, the surgeon needs more information as to the cause of the hematoma than the procedure can elicit, as illustrated by the following:

In a 16 year old boy awaiting removal of a cast prior to operation for thoracolumbar scoliosis, left hemiplegia rapidly developed and the patient became markedly obtunded within a few minutes. Emergency EMI scan revealed a large right cerebral hematoma encroaching on the right lateral ventricle (Fig. 15). Despite this disclosure, it was necessary to carry out carotid angiography to ascertain, if possible, the source of the bleeding, such as an arteriovenous anomaly or a congenital aneurysm (Fig. 16). Indications of the angiogram were almost within normal limits and the EMI

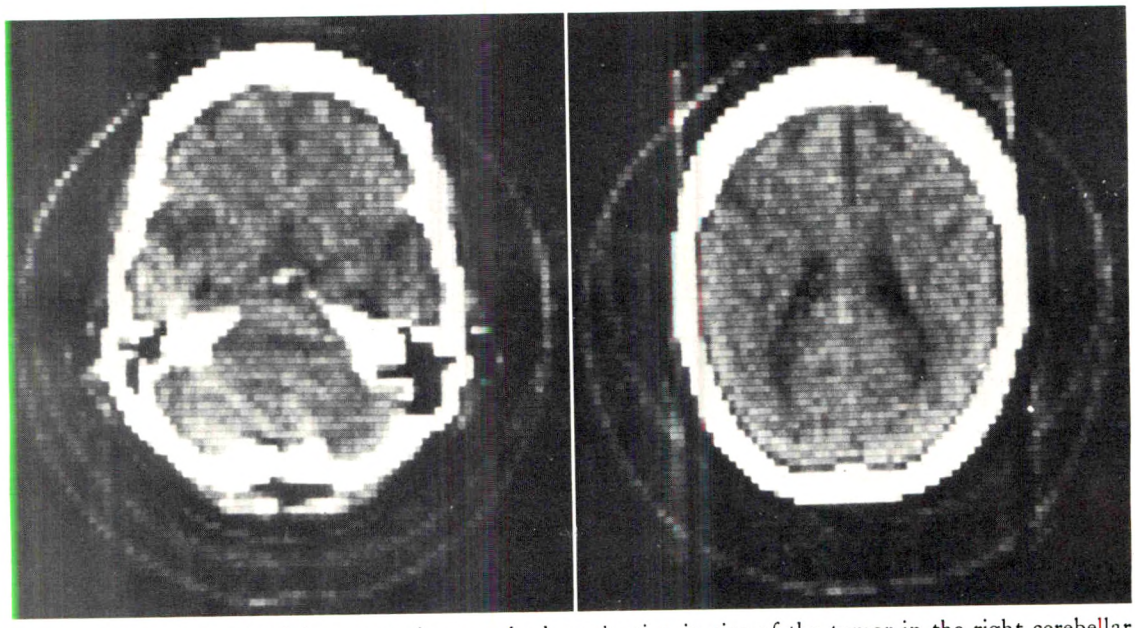


Fig. 12. Computerized axial tomography reveals the reduction in size of the tumor in the right cerebellar hemisphere and cerebral hemispheres and reduction in size of the ventricular system as the result of dexamethasone therapy.

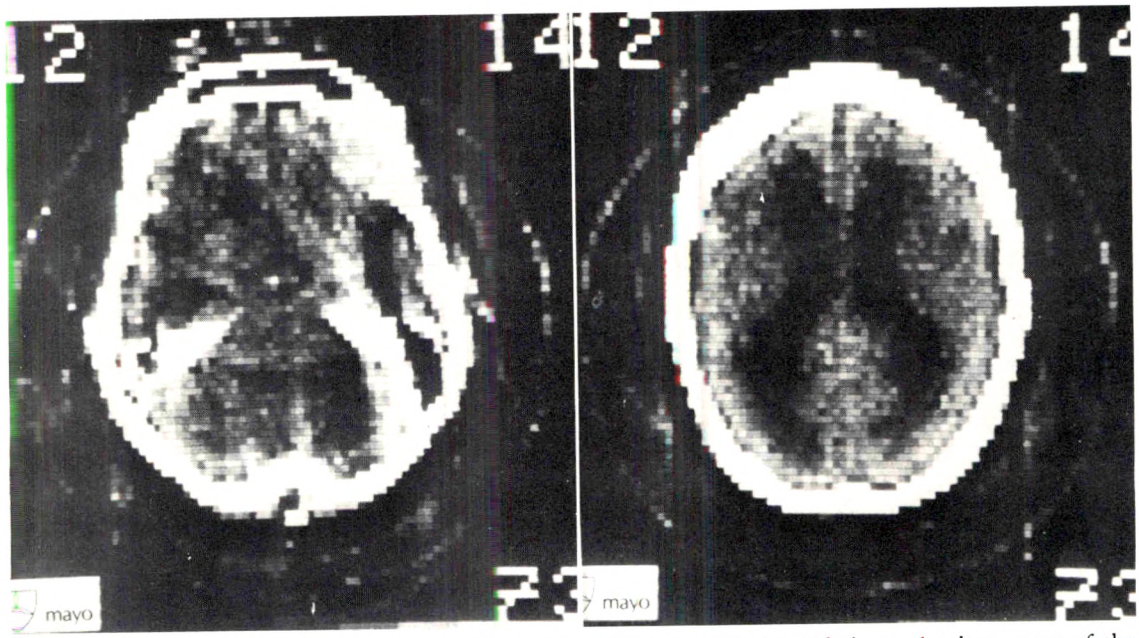


Fig. 13. Same case as that in Figures 11 and 12 with recurrence of hydrocephalus and enlargement of the cerebellar tumor after the discontinuance of dexamethasone therapy. The lesion proved to be metastatic adenocarcinoma of the lung.

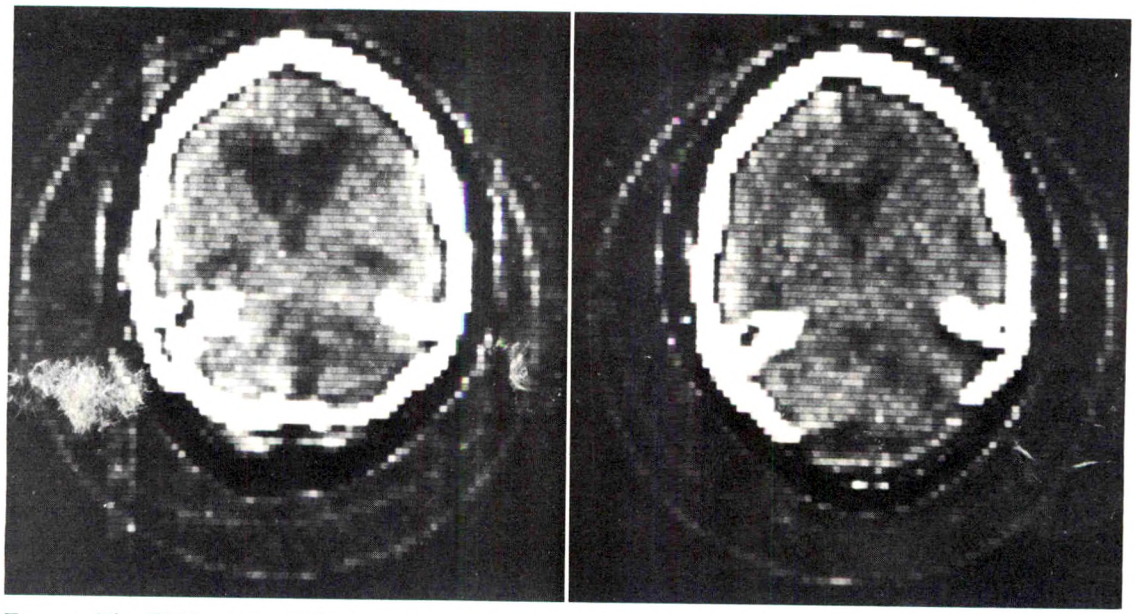


FIG. 14. The EMI scan reveals the solid portion of a hemangioendothelioma of the right cerebellar hemisphere, the cystic portion of the tumor and the hydrocephalus; 6 days following surgery, computerized axial tomography revealed the ventricular system to have returned to essentially normal size. (From Baker, H. L., Jr., Campbell, J. K., Houser, O. W., Reese, D. F., Sheedy, P. F., and Holman, C. B. Computer assisted tomography of the head: an early evaluation. *Mayo Clin. Proc.*, January, 1974, *49*, 17–24.)

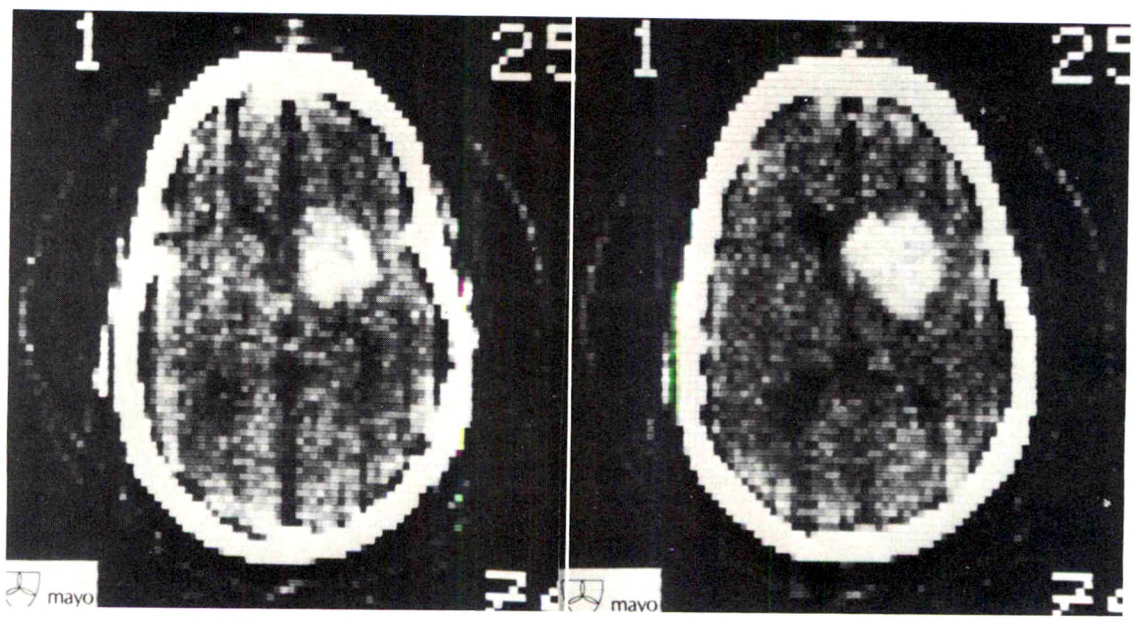


FIG. 15. The EMI scan shows the spontaneous intracerebral hematoma with rupture of the hematoma into the ventricular system.

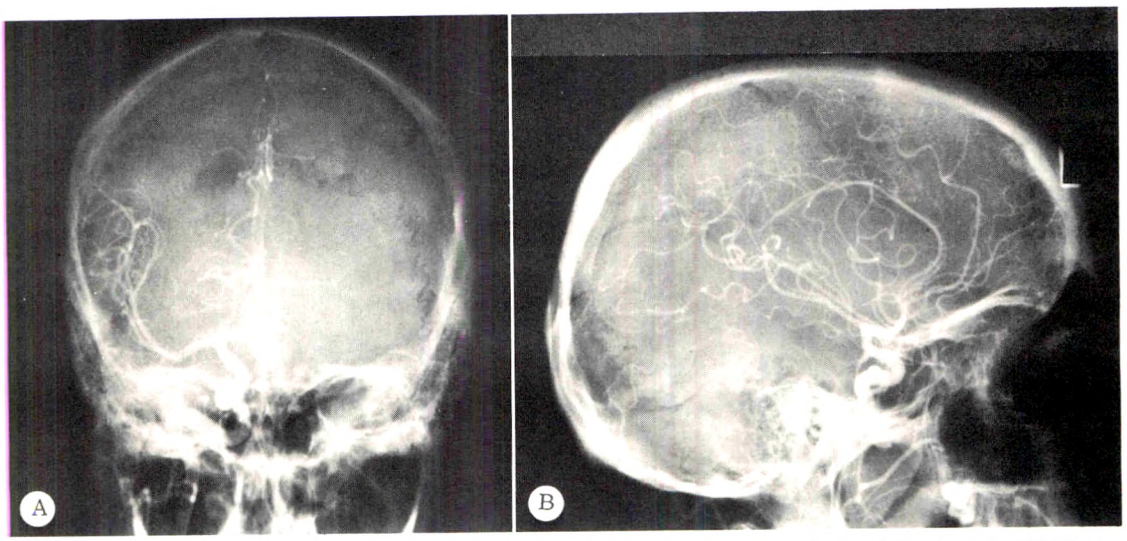


Fig. 16. (*A* and *B*) Angiograms of the same patient as in Figure 15 immediately following the EMI scan revealed very little distortion of the intracerebral vessels. Operation was successfully accomplished on the basis of the clinical findings and the EMI scan.

scan appeared to be infinitely more conclusive. Operation was accomplished successfully.

CONCLUSIONS

The effectiveness of EMI scans has been enhanced by the adjunctive use of contrast materials and by improvement of diagnostic acumen and equipment. The procedure surely will become the most nearly accurate single neuroradiologic examination. The salient advantages of the method are apparent in the lack of morbidity and mortality which follow its application. I shall not be enticed into a prediction as to the future of other diagnostic neuroradiologic procedures. You may speculate on these as well as I. However, the influence exerted by the machine on our practice is evident and it is extending. It is this aspect that I should like to discuss in my concluding remarks.

We train neurosurgeons to operate anywhere in the entire nervous system. The facts of the matter are that many spend their time practicing neurology, performing diagnostic tests, caring for head injuries and performing operations for protruded intervertebral disks. Computerized axial tomography is clearly a radiologic procedure. There will be a decline in cerebral angiography, as there has been in ventriculography and pneumography. Actually, there are too many neurosurgeons in the United States[17] performing procedures which probably would be better done by others. As we train new neurologists and neuroradiologists, neurosurgeons necessarily will be eliminated, or released, if you wish, from these tasks. Neurosurgical training programs already are reducing the number of residents by virtue of pressures from within and without teaching institutions. The number of residency programs surely will be reduced. With the advent of these social, educational and economic pressures, the scope of neurosurgery will be constricted and the numbers of neurosurgeons reduced. Finally, the baton will be passed to the neurologists and radiologists in the matter of how best they can contribute to ideal care for the patient. It is, however, absolutely imperative that the neurologist, radiologist and neurosurgeon work in complete harmony as well as with knowledge of what each is doing. What could be more disastrous than for an uninformed neurologist to order a pneumogram for a 12 year old child with papilledema and cerebellar ataxia and a busy

radiologist to perform the procedure, the patient becoming comatose and opisthotonic, and no neurosurgeon or operating room available? We may hope that disasters such as this will be eliminated by the judicious interpretation of computerized axial tomography and by the efforts of better-informed neurologists, neurosurgeons and neuroradiologists. My relationship with my colleagues in these disciplines has been enviable.

In closing, I should like to express my indebtedness to John Camp, Allen Good and David Pugh for their invaluable counsel and guidance in the early days and, in more recent years, the matchless help and notable acumen of Colin Holman, Bud Baker, Bob Scanlon, Dave Reese, Wayne Houser, Pat Sheedy and Dick Fulton.

I am grateful indeed to your Society for conferring upon me the distinction inseparable from the privilege of delivering the Caldwell Lecture.

REFERENCES

1. Adams, R. D., Fisher, C. M., Hakim, S., Ojemann, R. G., and Sweet, W. H. Symptomatic occult hydrocephalus with "normal" cerebrospinal fluid pressure: treatable syndrome. *New England J. Med.*, 1965, *273*, 117–126.
2. Ambrose, J. Computerized x-ray scanning of brain. *J. Neurosurg.*, 1974, *40*, 679–695.
3. Ambrose, J., and Hounsfield, G. Computerized transverse axial tomography. *Brit. J. Radiol.*, 1973, *46*, 148–149.
4. Antunes, J. L. Egas Moniz and cerebral angiography. *J. Neurosurg.*, 1974, *40*, 427–432.
5. Baker, H. L., Jr. Cerebellopontine angle myelography. *J. Neurosurg.*, 1972, *36*, 614–624.
6. Baker, H. L., Jr., Campbell, J. K., Houser, O. W., Reese, D. F., Sheedy, P. F., and Holman, C. B. Computer assisted tomography of head: early evaluation. *Mayo Clin. Proc.*, 1974, *49*, 17–27.
7. Bruwer, A. J. Classic Descriptions in Diagnostic Roentgenology. Volume I. Charles C Thomas, Publisher, Springfield, Ill., 1964, pp. 923–940.
8. Bruwer, A. J. Classic Descriptions in Diagnostic Roentgenology. Volume I. Charles C Thomas, Publisher, Springfield, Ill., 1964, pp. 941–965.
9. Bruwer, A. J. Classic Descriptions in Diagnostic Roentgenology. Volume I. Charles C Thomas, Publisher, Springfield, Ill., 1964, pp. 966–982.
10. Di Chiro, G., Doppman, J., and Ommaya, A. K. Selective arteriography of arteriovenous aneurysms of spinal cord. *Radiology*, 1967, *88*, 1065–1077.
11. Di Chiro, G., and Wener, L. Angiography of spinal cord: review of contemporary techniques and applications. *J. Neurosurg.*, 1973, *39*, 1–29.
12. Djindjian, R. Angiography of spinal cord. *Surg. Neurol.*, 1974, *2*, 179–185.
13. Hickey, P. M. Caldwell lecture, 1928. Am. J. Roentgenol. & Rad. Therapy, 1931, *25*, 177–195.
14. Holman, C. B. Evaluation of closed-circuit television technics in neuroroentgenology. *Acta radiol.* (Diag.), 1966, *5*, 241–249.
15. Houser, O. W. Personal communication.
16. Little, J. R., and MacCarty, C. S. Colloid cysts of third ventricle. *J. Neurosurg.*, 1974, *40*, 230–235.
17. Odom, G. L. Neurological surgery in our changing times: 1972 AANS presidential address. *J. Neurosurg.*, 1972, *37*, 255–268.
18. Ommaya, A. K. Computerized axial tomography of head: EMI-scanner; new device for direct examination of brain "*in vivo*." *Surg. Neurol.*, 1973, *1*, 217–222.
19. Powell, D. F., Baker, H. L., Jr., and Laws, E. R., Jr. Primary angiographic findings in pituitary adenomas. *Radiology*, 1974, *110*, 589–595.
20. Scanlan, R. L. Roentgen diagnosis of acoustic neuroma with particular reference to use of pantopaque. *Laryngoscope*, 1964, *74*, 999–1003.
21. Schlesinger, E. Personal communication.
22. Troland, C. E., and Otenasek, F. J. Selected Writings of Walter E. Dandy. Charles C Thomas, Publisher, Springfield, Ill., 1957, pp. 39–44.
23. Troland, C. E., and Otenasek, F. J. Selected Writings of Walter E. Dandy. Charles C Thomas, Publisher, Springfield, Ill., 1957, pp. 55–60.
24. Troland, C. E., and Otenasek, F. J. Selected Writings of Walter E. Dandy. Charles C Thomas, Publisher, Springfield, Ill., 1957, pp. 61–72.
25. Tucker, A. It Happened at Hopkins: A Teaching Hospital. (Prepared on behalf of Johns Hopkins Medical Journal.) Johns Hopkins Hospital, Baltimore, Md., 1973, p. 102.
26. Walker, A. E. A History of Neurological Surgery. Williams & Wilkins Company, Baltimore, 1951, pp. 583.
27. Wilkins, R. H. Neurosurgical Classics. Johnson Reprint Corporation, New York, 1965, pp. 242–256.

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EFFECT OF ANGIOGRAPHIC CONTRAST MEDIA AT THE CELLULAR LEVEL IN THE BRAIN: HYPERTONIC *VS.* CHEMICAL ACTION*

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ELECTRON microscopic studies have resulted in observations that are related to the concept of the blood-brain barrier: (1) due to the sparsity of an extracellular space in the brain, transfer of matter must take place primarily from the blood into the intracellular spaces;[8,11] (2) closely apposed surfaces (tight junctions) characterize the interface between parenchymal capillary endothelial cells of the brain;[2] and (3) pinocytotic vesicles in the endothelial cells of blood vessels are involved in transport between blood and adjacent parenchymal cells. These vesicles are present in other organs of the body, but appear to be comparatively small in number in the brain.[12]

It has been well established that it is the parenchymal capillary endothelium which impedes the passage of matter from the vascular compartment into the brain.[2,7,11–13,15] Under normal conditions the brain capillary endothelial cells serve as a protective barrier to matter circulating in the vascular compartment. The tight junctions between these endothelial cells are thought to constitute one of the structural bases of the blood-brain barrier.

In an earlier report from this laboratory, tight junctions were shown to open following repeated injections of angiographic contrast media.[16] Using saccharated iron as electron opaque tracer material, iron particles were seen in the open junctions following repeated injections of contrast media. In addition, numerous pinocytotic vesicles, seen infrequently in controls, were seen in the capillary endothelial cells. These vesicles contained iron particles, suggesting significant transport from blood to brain as a result of the repeated injections of angiographic contrast media.

Our object in this paper is to review these changes and to compare them to the effect produced by hypertonic saline (5% NaCl) injections. Sodium chloride 5% is known to have approximately the same osmolality as the contrast media in common usage for cerebral angiography.[1,3–5]

METHOD

Right craniotomies were followed by right common carotid artery catheterizations performed under direct vision in 6–12 pound cats of both sexes using intravenous pentobarbital anesthesia. A 20 gauge polyethylene catheter was positioned in the artery over a guide wire which had been placed through a tiny arteriotomy made with iridectomy scissors. Deep cerebral biopsies were obtained at the conclusion of each experiment, entering the right parietal convexity and extending through the floor of the lateral ventricle and into the region of the third ventricle. Specimens from cerebral cortex, white matter, ependyma, and choroid plexus were obtained and placed immediately in 1% buffered

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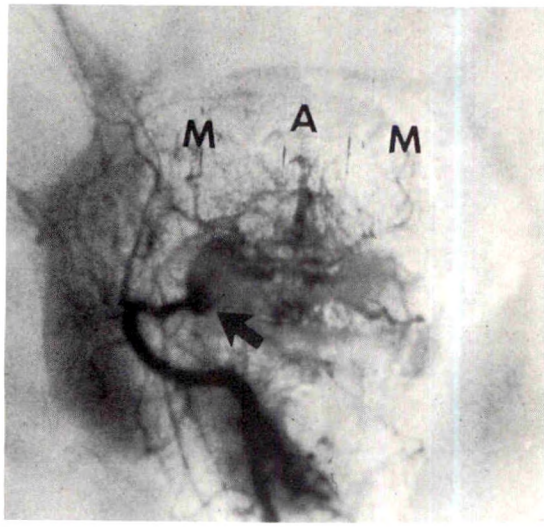


FIG. 1. Anteroposterior common carotid arteriogram (subtraction film) obtained with 3 ml. of Hypaque 50, containing 100 mg. Armidexan. Note that the middle (M) and anterior (A) cerebral arteries are opacified bilaterally with this volume of contrast media injected into the common carotid artery. The internal carotid artery is rudimentary in the adult cat and the intracranial arteries are supplied principally through the rete externum (carotid plexus) (arrow).

osmium tetroxide. The tissue was rapidly dehydrated in ethanol and embedded in Swiss Araldite. Thin sections were cut on a diamond knife with a Porter-Blum MT-1 microtome and examined without staining in an RCA EMU-3F electron microscope.

Control animals received: (1) injection of 3 ml. normal saline every 2 minutes for a total of 15 injections with biopsy obtained following the last injection; (2) injection of a combination of 2 ml. normal saline plus 1 ml. (100 mg.) saccharated iron compound* at the same time interval as (1) with biopsy obtained following the last injection; and (3) craniotomy only with no arterial injections but elapsed time from craniotomy to biopsy the same as (1) and (2).

The experimental models (Fig. 1) employed repeated injections of (1) sodium diatrizoate 50%† (14 animals), (2) meglumine iothalamate 60%‡ (2 animals), or (3) sodium chloride 5% (6 animals) (one group utilizing 3 ml. of the contrast media or hypertonic saline, containing 100 mg. saccharated iron, every 2 minutes for a total of 15 injections in 30 minutes, and a second group using 3 ml. of contrast media or hypertonic saline, containing 100 mg. saccharated iron, every $7\frac{1}{2}$ minutes for a total of 5 injections in 30 minutes).

* Armidexan.
† Hypaque 50.
‡ Conray 60.

RESULTS

In the control group receiving no injections of saline, tight junctions are closely apposed and pinocytotic vesicles are rare (Fig. 2, *A* and *B*). In controls receiving 15 injections of normal saline the tight junctions remain closed, but a few large pinocytotic vesicles can be seen and, when the saccharated iron electron microscopic tracer material is used, tracer particles can be identified in these vesicles (Fig. 3, *A* and *B*). Rarely, tracer particles can be identified in the adjacent perivascular space in the normal saline controls.

Repeated injections of angiographic contrast media cause increased pinocytosis and opening of tight junctions. This phenomenon has been reported previously with Hypaque 50,[16] but also occurs with Conray 60 (Fig. 4, *A* and *B*). Even with Decadron pretreatment, which prevents the development of much of the associated glial edema that occurs,[15] the pinocytotic activity is increased and the tight junctions can be found to open (Fig. 6, *A* and *B*).

If biopsy is delayed for one hour following the 15 injections of contrast media sequence, the tight junctions are normal in appearance and little pinocytotic activity is seen, but tracer particles can be found in the adjacent glial cells as evidence that the endothelial barrier had been disrupted but rapidly returned to its normal appearance (Fig. 7, *A* and *B*).[14]

In animals receiving 5 injections of contrast media over 30 minutes rather than 15 injections, pinocytotic activity was not as marked as in the animals with 15 injections but was increased compared to normal saline controls and tracer particles were

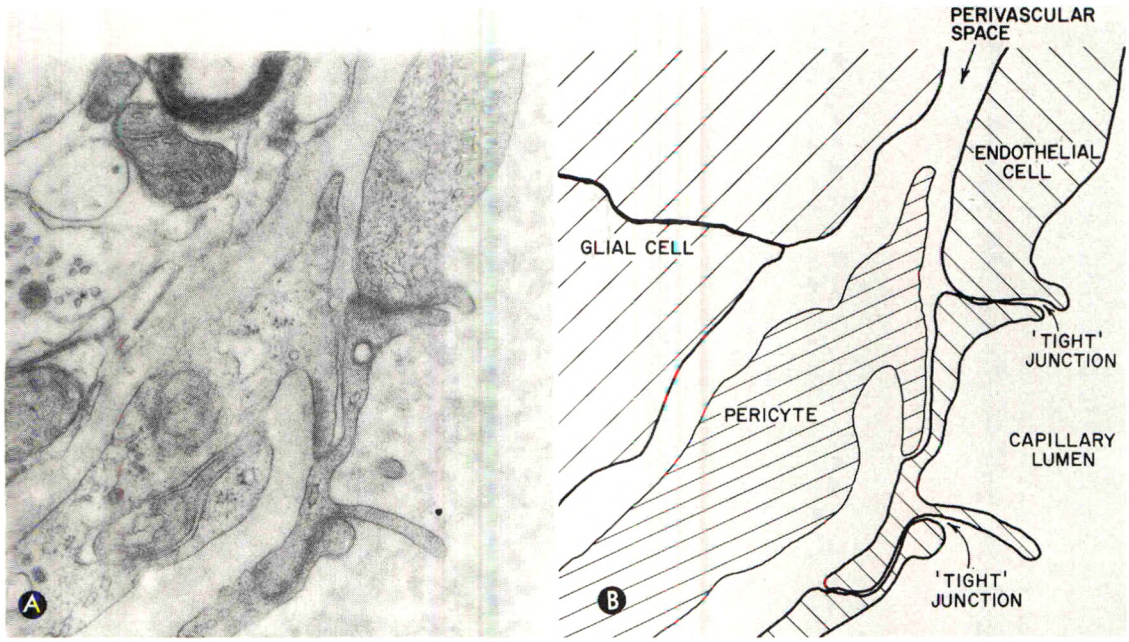


FIG. 2. (*A*) Electron micrograph and (*B*) schematic diagram of noninjected control. Note visualization of 3 endothelial cells lining the capillary lumen. Two "tight junctions" are illustrated. Immediately to the left of the endothelial cells is a small perivascular space containing a pericyte (fibroblast). The boundaries of the perivascular space are endothelial cells on the right and glial cells on the left. Cerebral cortex 76,000 ×

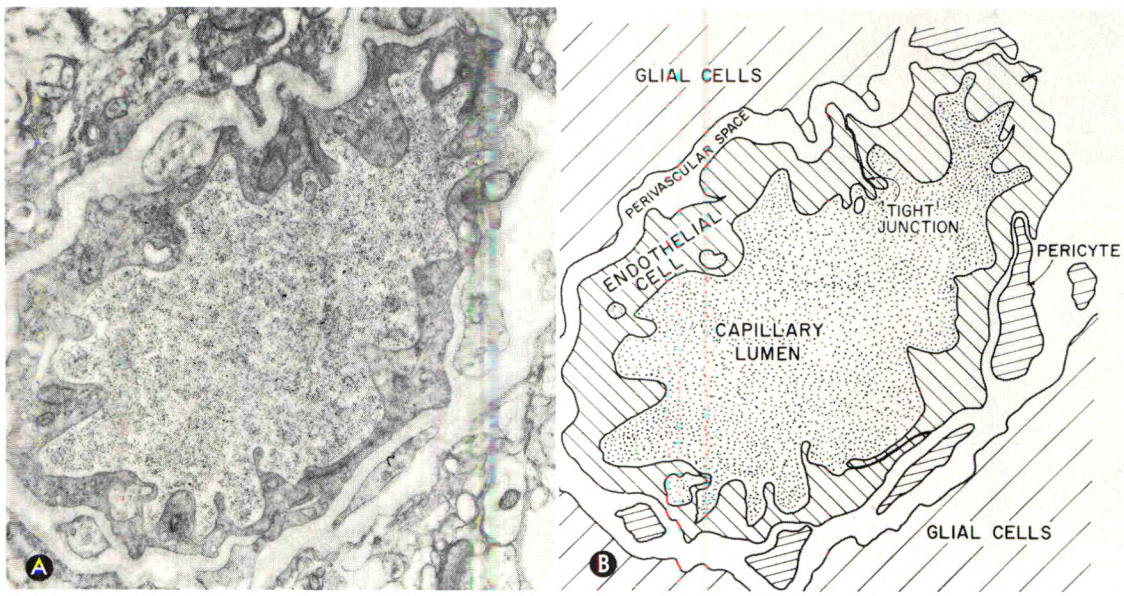


FIG. 3. (*A*) Electron micrograph and (*B*) schematic diagram of control receiving 15—3 ml. injections of normal saline containing Armidexan. Note the normal appearance of tight junctions between adjacent endothelial cells. A few large pinocytotic vesicles are seen to contain tracer particles. White matter 37,400 ×.

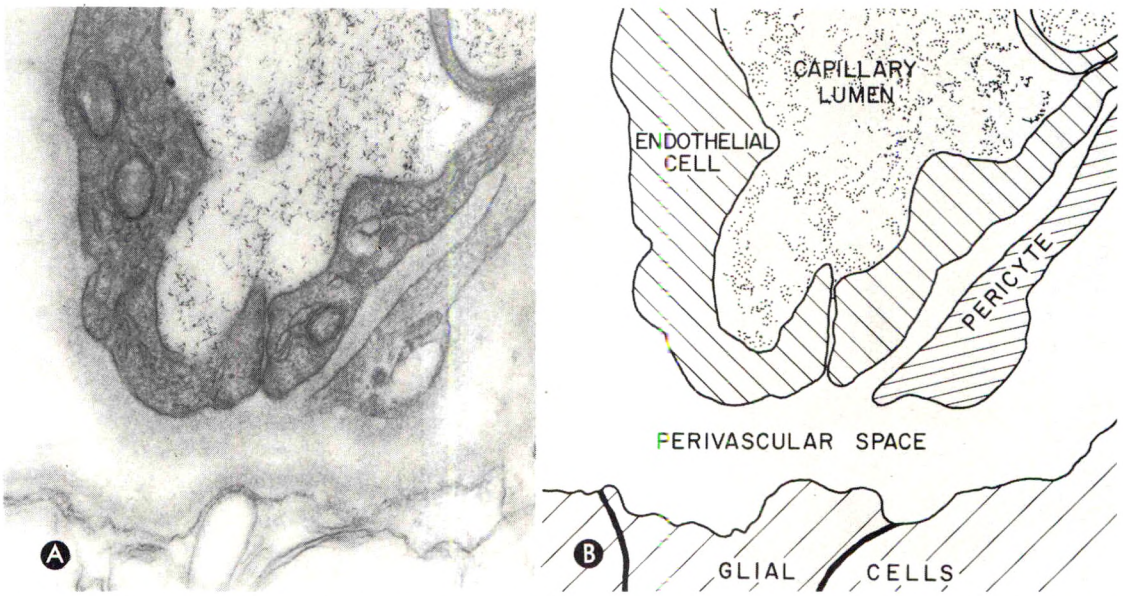


FIG. 4. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of Conray 60. The tight junction illustrated is opened throughout its course. Large endothelial pinocytotic vesicles are not noticeable in this field, but are common in animals receiving 15 injections of Conray 60 as well as Hypaque 50. Cortex 103,000 ×.

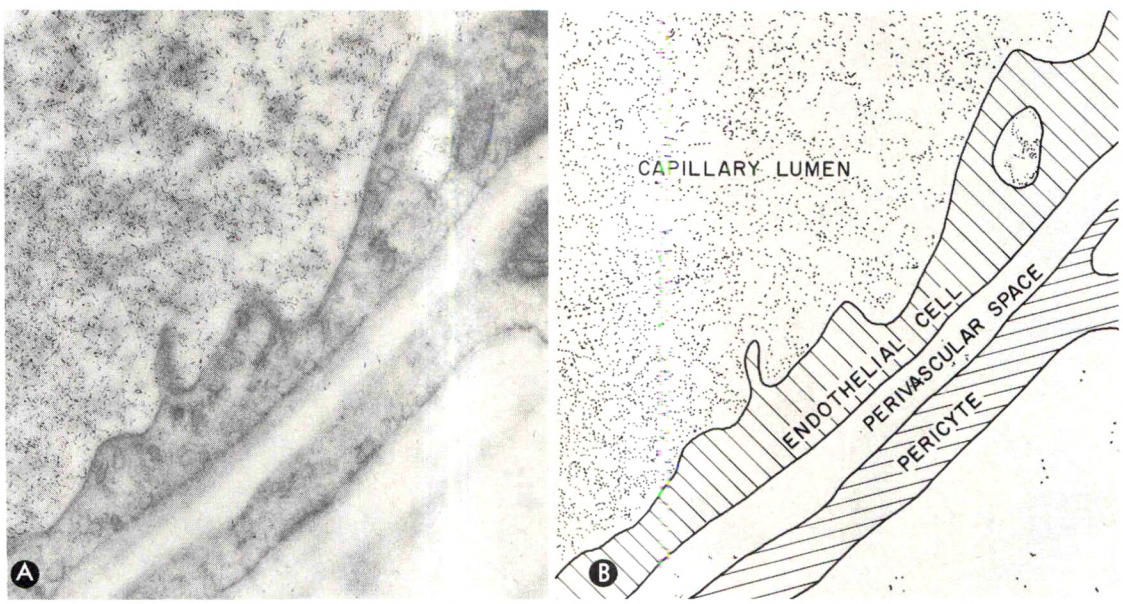


FIG. 5. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of Hypaque 50 to illustrate a common appearance of the perivascular space. No tight junctions are seen in this section. One large pinocytotic vesicle contains tracer particles. The capillary lumen contains numerous tracer particles. Scattered tracer particles are located in the perivascular space. The perivascular space (basal lamina) which surrounds the endothelium of capillaries is in continuity with the cerebral subarachnoid space. Around small capillaries it is a potential space only; but theoretically, at least, substances which are located in this space can migrate into the cerebral subarachnoid space.

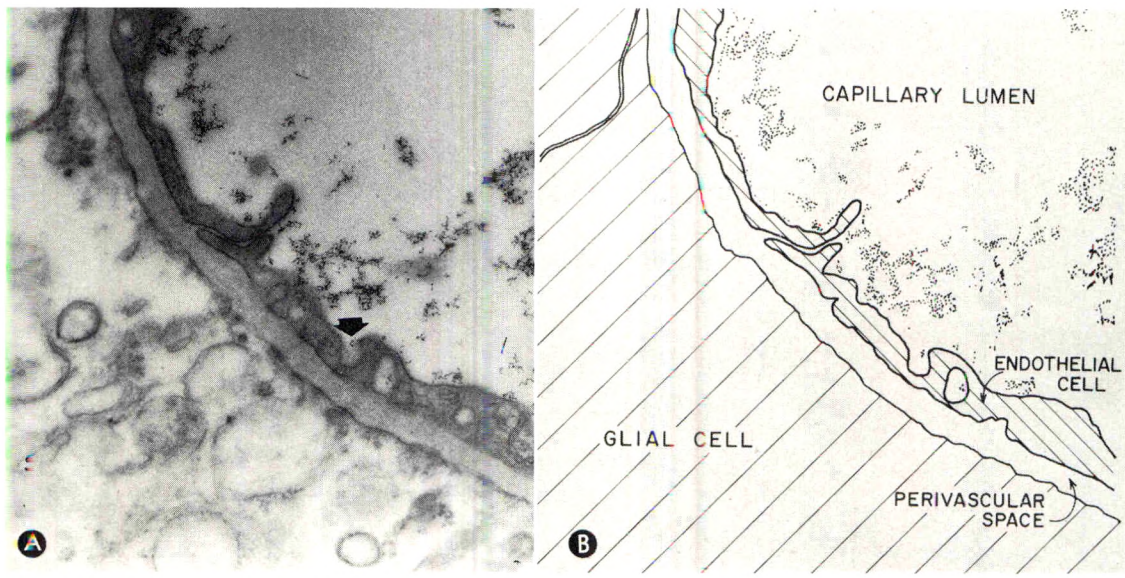


Fig. 6. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of Hypaque 50; 4 mg. of Decadron had been administered both intravenously and intramuscularly prior to the experimental protocol. The tight junction in the center of the field is opened throughout its course. Numerous large pinocytotic vesicles containing tracer particles are seen including one (arrow) opening into the capillary lumen. White matter 76,000 ×.

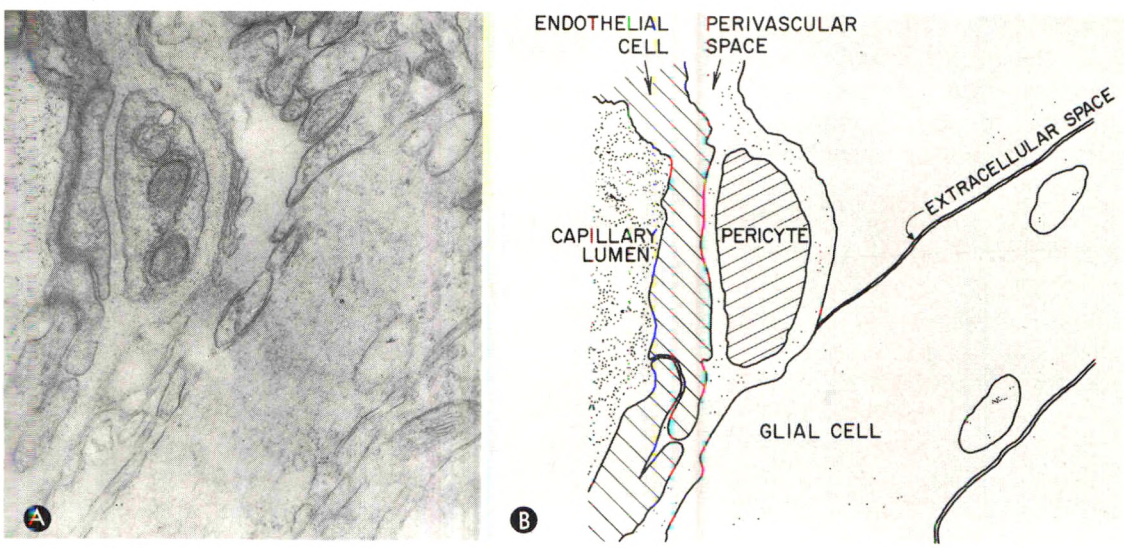


Fig. 7. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of Hypaque with biopsy 1 hour after the last injection. The capillary is on the left. The single tight junction is closed and the appearance of the endothelial cells is similar to that seen in the noninjected control animals. The perivascular glial cells are not edematous. However, tracer iron particles are present in the glial cells indicating prior blood-brain transport. Cortex 76,000 ×.

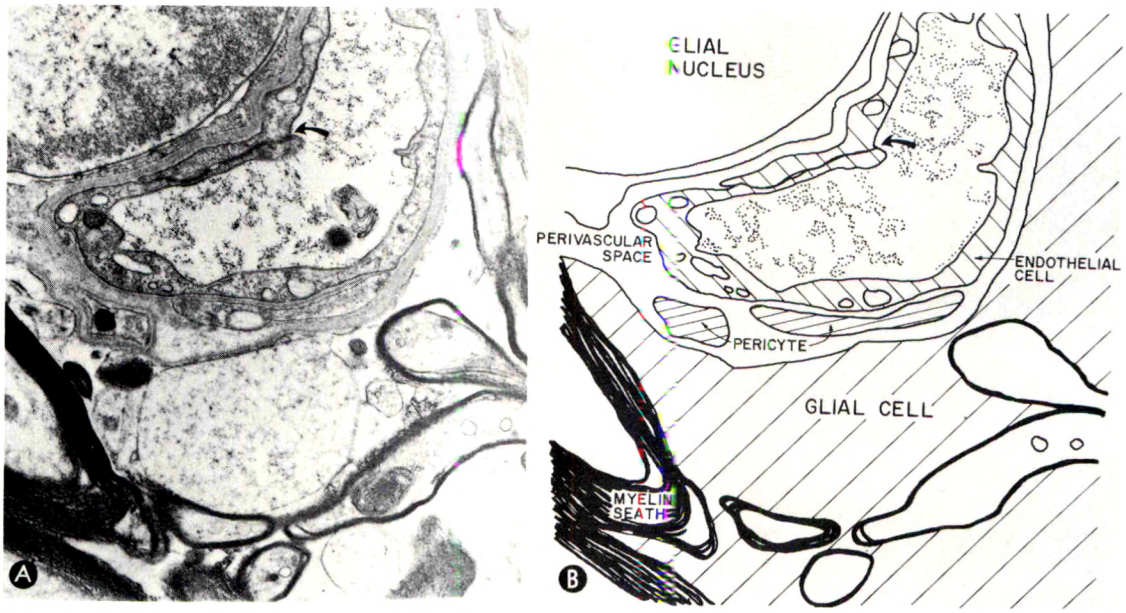


FIG. 8. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of NaCl 5% (hypertonic saline). Medium power. Note multiple, large pinocytotic vesicles in the endothelial cells. The single tight junction (arrow) has multiple areas of apposition and is normal in appearance. White matter 27,000 ×.

identified in the perivascular space indicating passage across the capillary endothelium. No opened tight junctions were identified.

In the group receiving 15 injections of hypertonic saline, pinocytosis occurred to the same degree as that seen in animals with 15 injections of contrast media, but it was not possible to identify any opened tight junctions (Fig. 8, *A* and *B*; and 9, *A* and *B*). In animals receiving 5 injections of hypertonic saline over 30 minutes, the amount of pinocytotic activity quantitatively simulated that seen in animals with 5 injections of contrast media.

DISCUSSION

The blood-brain barrier is not, we feel, an all or none phenomenon based on the observation that a limited amount of pinocytotic transport can occur when normal saline is injected into the common carotid artery, frequently, at short intervals (Fig. 3, *A* and *B*). However, because of the tight junctions between adjacent endothelial cells, which remain unaltered, and are peculiar to the parenchymal capillaries of the brain, this endothelium functions as a moderately effective barrier to matter circulating in the vascular compartment.

Repeated injections of angiographic contrast media break through the endothelial barrier, *i.e.*, blood-brain barrier by: (1) increasing pinocytotic activity; and (2) opening tight junctions between the endothelial cells. It is a well known fact that cerebral capillaries show no macromolecular transport in physiologic circumstances.[6] Likewise, the tight junctions do not allow passage of matter through them under physiological conditions.[2] Thus, the repeated injections of contrast media alter both the pinocytotic transport and the tight junctions in disrupting the blood-brain barrier.

Once through the endothelial barrier, tracer material can be seen to migrate into: (1) the perivascular space, which communicates (potentially) with the subarachnoid space (Fig. 5, *A* and *B*); and (2) into the intracellular space, *i.e.*, glial cells (Fig. 6, *A* and *B*); but (3) we have not identified tracer material in the extracellular space.

When 15 injections of contrast media are given every 2 minutes, but biopsy delayed

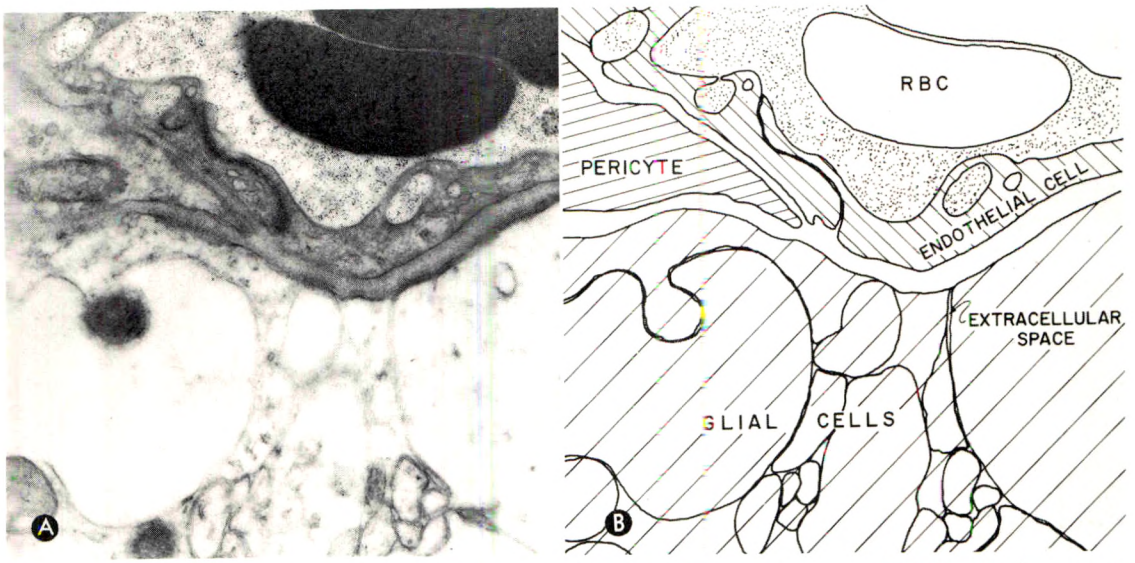


Fig. 9. (*A*) Electron micrograph and (*B*) schematic diagram after 15 injections of NaCl 5% (hypertonic saline). Higher power. The multiple, large pinocytotic vesicles in the endothelium can be seen to contain tracer particles. The single tight junction is normal in appearance. Cortex 52,000 ×.

for 1 hour following the last injection, the tight junctions are closed and the number of pinocytotic vesicles simulates those in control animals with saline injections. However, iron particles, which are used as the electron microscopic tracer material, are seen in the glial cells indicating prior blood-brain transport (Fig. 7, *A* and *B*).

The alteration in the blood-brain barrier produced by repeated injections of angiographic contrast media, therefore, appears to represent a reversible process. Evidence that it has occurred remains, however, in the form of iron particles seen in the perivascular glial cells. Thus, recovery from the insult to the blood-brain barrier brought about by the frequent repeated injections of angiographic contrast media occurs rapidly.

Rapoport and co-workers[9,10] have theorized that hypertonic solutions *per se* osmotically shrink endothelial cells and open tight junctions, and it is a popular theory that it is the hypertonic quality of contrast media which is responsible for its ill effects on the brain. Our findings in these studies have led us to conclude that repeated injections of angiographic contrast media result in transgression of the blood-brain barrier by 2 mechanisms.

Firstly, the hyperosmolar quality of the contrast media leads to increased pinocytosis, and it is this quality which can be reproduced by substituting hypertonic saline injections for contrast media injections. However, one must invoke a second quality for the contrast media based on chemical structure to account for opening of tight junctions between the endothelial cells which occurs with the contrast media, but not with the hypertonic saline injections of similar osmolality.

SUMMARY

Repeated injections of angiographic contrast media led to disruption of the endothelial barrier of the cerebral parenchymal capillaries (blood-brain barrier) experimentally by (a) promoting pinocytosis and (b) opening the tight junctions which exist between the endothelial cells.

Two mechanisms appear to be involved in this process. The hyperosmolar quality of the contrast media is responsible for the increased pinocytosis, while the chemical nature of the contrast agents used (Hy-

paque 50, and Conray 60), results in opening of the tight junctions.

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REFERENCES

1. BØRDALEN, H., WANG, H., and HOLTERMANN, H. Osmotic properties of some contrast media. *Invest. Radiol.*, 1970, *5*, 559–565.
2. BRIGHTMAN, M. V., and REESE, T. S. Junctions between intimately apposed cell membranes in vertebrate brain. *J. Cell. Biol.*, 1969, *40*, 648–677.
3. HARRINGTON, G., MICHIE, G., LYNCH, P. R., *et al.* Blood-brain barrier changes associated with unilateral cerebral angiography. *Invest. Radiol.*, 1966, *1*, 431–440.
4. HILAL, S. K. Hemodynamic changes associated with intra-arterial injection of contrast media. *Radiology*, 1966, *86*, 615–633.
5. HILAL, S. K. Trends in preparation of new angiographic contrast media with special emphasis on polymetric derivatives. *Invest. Radiol.*, 1970, *5*, 458–468.
6. JOÓ, F. Increased production of coated vesicles in brain capillaries during enhanced permeability of blood-brain barrier. *Brit. J. Exper. Path.*, 1971, *52*, 646–649.
7. KARNOVSKY, M. J. Ultrastructural basis of transcapillary exchanges. *J. Gen. Physiol.*, 1968, *52*, 64–95.
8. LUSE, S. A., and HARRIS, B. Electron microscopy of brain in experimental edema. *J. Neurosurg.*, 1960, *17*, 439–446.
9. RAPOPORT, S. I., BACHMAN, D. S., and THOMPSON, H. K. Chronic effects of osmotic opening of blood-brain barrier in monkey. *Science*, 1972, *176*, 1243–1245.
10. RAPOPORT, S. I., HORI, M., and KLATZO, I. Reversible osmotic opening of blood-brain barrier. *Science*, 1971, *173*, 1026–1028.
11. REESE, T. S., FEDER, N., and BRIGHTMAN, M. W. Electron microscopic study of blood-brain and blood cerebral fluid barriers with microperoxidase. *J. Neuropath. & Exper. Neurol.*, 1971, *30*, 137–138.
12. REESE, T. S., and KARNOVSKY, M. J. Fine structure localization of blood-brain barrier to exogenous peroxidase. *J. Cell. Biol.*, 1967, *34*, 207–217.
13. VICK, N. A., and BIGNER, D. D. Microvascular abnormalities in virally-induced canine brain tumors. *J. Neurol. Sc.*, 1972, *17*, 29–39.
14. WALDRON, R. L., II, BRIDENBAUGH, R. B., and DEMPSEY, E. W. Alterations at cellular level in brain following repeated injections of angiographic contrast media: insult and recovery. Presented at the American Society of Neuroradiology, Boston, Mass., April, 1973.
15. WALDRON, R. L., II, BRIDENBAUGH, R. B., and DEMPSEY, E. W. Alterations at cellular level in brain following repeated angiography: protective effect of corticosteroids. Presented at the Association of University Radiologists, Vancouver, British Columbia, May, 1973.
16. WALDRON, R. L., II, BRIDENBAUGH, R. B., PURKERSON, M., and DEMPSEY, E. W. Effect of angiographic contrast media at cellular level in brain. *Radiology*, 1973, *108*, 187–189.

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CEREBRAL ANGIOGRAPHIC FINDINGS IN DISSEMINATED INTRAVASCULAR COAGULATION*

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DISSEMINATED intravascular coagulation (DIC) is an acquired hemorrhagic thrombotic syndrome which occurs as a result of the presence of thrombin in the systemic circulation. It is not a distinct pathologic entity, but rather a pathophysiologic state which is an intermediary mechanism of disease. [3,6,7,9] DIC occurs in the microcirculation and should be distinguished from thromboembolic disease involving major vessels.[1]

There are 3 processes which can cause disseminated intravascular coagulation: (1) endothelial cell injury with secondary exposure of collagen which activates Hageman factor XII and the intrinsic clotting system; (2) tissue injury with a resultant release of tissue thromboplastin which in the presence of factor VII can activate the extrinsic clotting system; or (3) injury to red cells and/or platelets with the release of phospholipid which accelerates blood coagulation. These injury mechanisms, with their subsequent effect on the clotting systems eventually result in the formation of a proteolytic enzyme, thrombin.[3]

When thrombin is released in the systemic circulation, it has a number of effects; not the least of which are the cleavage of fibrinogen to form fibrin, aggregation of platelets, and the consumption of specific plasma proteins. Secondary fibrinolysis is also activated by thrombin, probably through its activation of plasminogen. Plasmin also results in fibrinogen degradation products which participate in the hemorrhagic diathesis. The combination of activation of clotting mechanisms and the secondary fibrinolysis causes the clinical manifestation of hemorrhage and fibrin thrombus formation. DIC presents clinically with the picture of bleeding at multiple sites and occasionally with acrocyanosis or thrombotic episodes.[3,5,6,15]

Some of the disease states in which DIC is seen include: tissue injury as seen in obstetrical, surgical, or neoplastic disorders; endothelial injury such as gram negative or positive septicemia, viremia or prolonged hypotension; platelet or red cell injury such as hemolysis; and finally reticuloendothelial system injury exemplified by liver injury or post splenectomy.[3,4,6,7,9,10] The clinical features of this disorder include a hemorrhagic thrombotic diathesis, a specific coagulation test profile, the presence of fibrin thrombi and the response to heparin therapy. Three abnormal screening tests are important for the diagnosis. These are the prothrombin time, fibrinogen level, and platelet count. Confirmatory tests for fibrinolysis are the thrombin time, euglobulin lysis time, or detection of fibrinogen degradation products. Heparin is the treatment of choice and when used in correctly diagnosed cases, leads to a prompt response in the clotting factors.[3]

REPORT OF CASES

CASE 1. E. S.—032739, a 72 year old white female was admitted on July 6, 1972, for radiation therapy for recently uncovered endometrial carcinoma, complicated by an episode of *Bacteroides* septicemia. In the second week of her radiation therapy, she suddenly became confused and disoriented. Neurologic evaluation revealed impaired cognitive function, complete left homonymous hemianopsia, abnormal posturing and impaired rapid alterating movements in the left extremities without drift or weakness, and extinction of touch and pain

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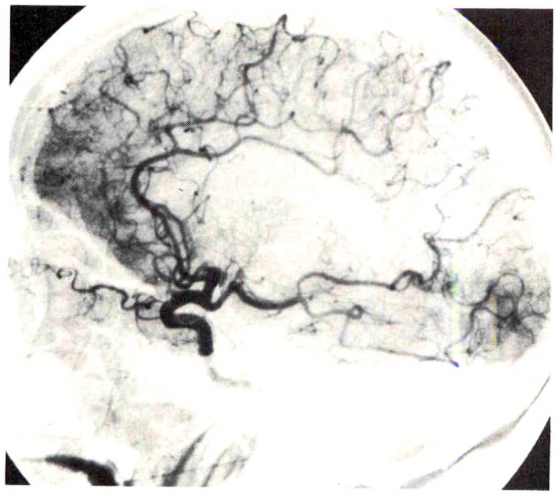


FIG. 1. Case 1. *July 31, 1972.* Left carotid arteriogram. There is complete occlusion of the left middle cerebral artery distal to the origin of the lenticulostriate arteries.

stimuli on the left side with double simultaneous stimulation. The remainder of the sensory examination was normal, except for mislocalization of touch stimuli on the left side. Her neurologic findings remained stable, but on the following day she became aphasic, and developed right central facial weakness, mild weakness in the left arm and leg, and bilateral hyper-reflexia. Laboratory studies revealed a hematocrit of 23 per cent, a platelet count of 60,000, prothrombin time of 18.7 sec. over a control of 11.4 sec., thrombin time 24.9 sec. with a control of 19.9 sec., fibrinogen 270 mg. per cent, ethanol gel test for fibrin split products positive. A left carotid arteriogram on July 31, 1972, demonstrated complete occlusion of the left middle cerebral artery distal to the origin of the lenticulostriate arteries (Fig. 1). There was delayed retrograde filling of the major branches of the middle cerebral artery and filling defects were demonstrated in these occluded vessels (Fig. 2; and 3). Intravenous Heparin was begun. Over the next 48 hours, she showed further neurologic deterioration, with flaccid paralysis of the right arm and leg, loss of visual responses to threat, and to optokinetic stimuli, and progressive decline in level of consciousness. Pupillary responses remained normal. Thereafter, until her death 4 weeks later, she remained neurologically stable, but developed multiple purpuric lesions, and loss of pulses and gangrenous changes in both feet. Despite continuous Heparin therapy, and improvement in her abnormal coagulation studies to or toward normal, she showed no significant functional recovery and died following hypotension and septicemia.

CASE II. M.A.—032600, a 55 year old white female, whose admission was prompted by 2 episodes of transient left arm and left leg weakness within the previous month. Her neurologic examination on admission was unremarkable, but within 24 hours she had developed a dense left hemiparesis and left homonymous hemianopsia. Chest roentgenogram demonstrated a

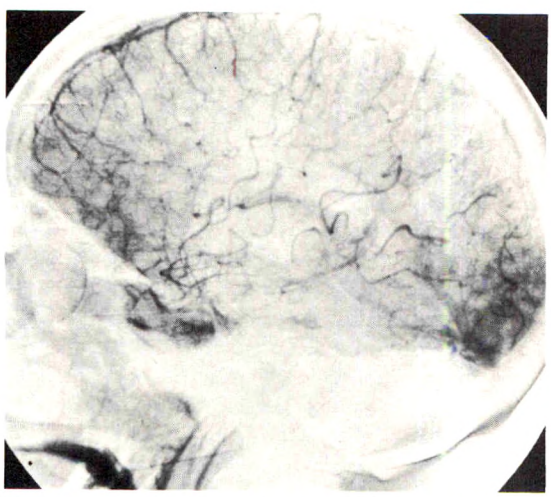


FIG. 2. Case 1. Left carotid arteriogram, early venous phase. There is delayed retrograde filling of the major branches of the middle cerebral artery.

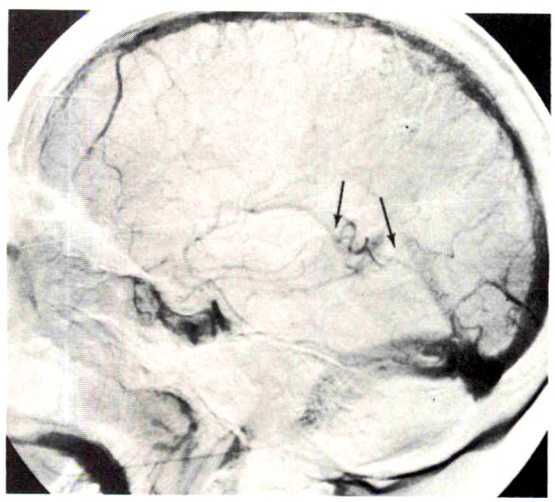


FIG. 3. Case 1. Left carotid arteriogram, late venous phase. Filling defects can be seen in an occluded posterior parietal branch of the middle cerebral artery (arrows).

left hilar mass. On transfer to the Hershey Medical Center, June 30, 1972, she was disoriented, lethargic, but responsive to simple commands. She showed moderate left-sided weakness, arm greater than leg, left-sided hyper-reflexia, clasp-knife resistance to passive movement, and an extensor plantar response on the left. Multiple cutaneous ecchymoses, and prolonged bleeding from venapuncture sites were noted. A right carotid arteriogram the same day demonstrated complete occlusion of the major trunk of the middle cerebral artery, with delayed retrograde filling of the ascending frontoparietal artery via leptomeningeal collaterals from the pericallosal artery (Fig. 4; and 5). On the following day she became more agitated and developed weakness of the right face and arm, dysphasia and loss of all evidence of vision, including response to optokinetic stimuli and to visual threats, while pupillary responses to light were preserved. Laboratory studies on admission revealed a platelet count of 86,000, fibrinogen 110 mg. per cent, and thrombin time 24.9 sec. with a control of 17.6 sec. Other hematologic studies were within normal limits. After 3 days of Heparin therapy, the platelet count had risen to 244,000 and fibrinogen to 618 mg. per cent; other hematologic abnormalities were also reversed. Her neurologic deficits failed to improve, and she developed Cheyne-Stokes respiration, progressive unresponsiveness to painful stimuli, and hypotension leading to death on her 6th hospital day.

Postmortem examination showed bronchogenic carcinoma, metastatic to the mediastinum, and multiple areas of intravascular thrombosis in varying stages of organization, involving large and medium size arteries in heart, liver, spleen, brain and other organs. Friable fibrinoid material was present on the left heart valves, and was identified within several arteries also. The brain showed multiple areas of cortical and subcortical ischemic infarction, involving right central and parietal areas, left medial occipital lobe, and right cerebellar hemisphere.

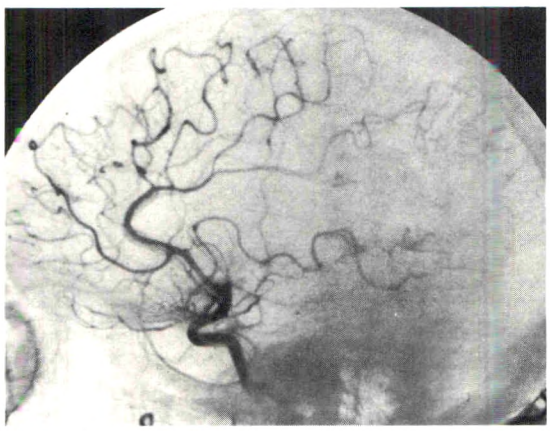


Fig. 4. Case II. *June 30, 1972.* Right carotid arteriogram. There is complete occlusion of the middle cerebral artery just distal to the origin of the posterior temporal artery.

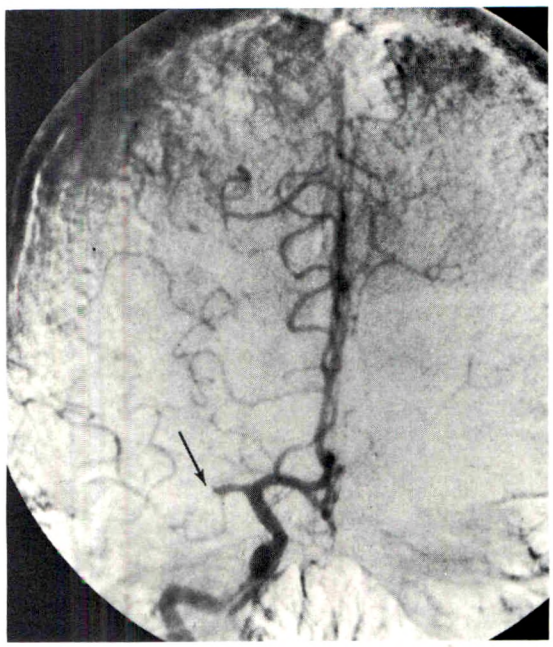


Fig. 5. Case II. Right carotid arteriogram. Anteroposterior projection. This view confirms the occlusion of the middle cerebral artery (arrow).

Case III. R.H.—040358, a 60 year old white male, developed bilateral calf thrombophlebitis, and was treated briefly with anticoagulants. Three weeks later, on December 23, 1972, he awoke from sleep with aphasia and right hemiparesis. He was admitted to a local hospital, and thereafter showed stepwise deterioration in neurologic function over the next 4 days. On transfer to the Hershey Medical Center on January 3, 1973, he was severely obtunded, with normal vital signs. Responses to painful stimuli were incomplete and purposeless, including decerebrate posturing on the left, and decorticate movements on the right. Oculocephalic movements were conjugate and brisk in all directions. He intermittently showed bursts of low amplitude nystagmus on reflex

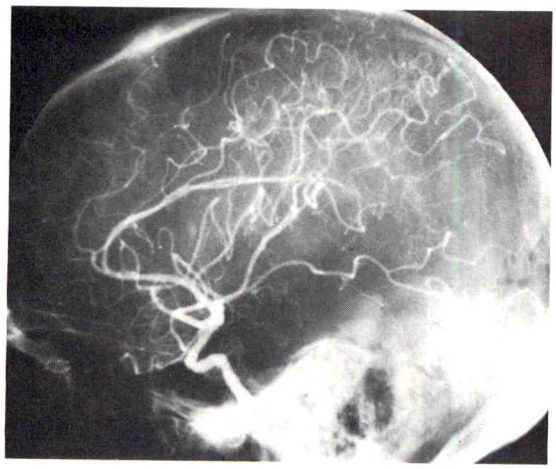


FIG. 6. Case III. *January 4, 1973.* Right carotid arteriogram, early arterial phase. There are multiple occlusions of intermediate sized vessels in the distribution of the ascending frontoparietal and posterior parietal branches of the middle cerebral artery.

conjugate lateral eye movements. There were no responses to corneal stimuli, loud noises or visual threat. The response to optokinetic testing was equivocal. Reflexes were hyperactive throughout, with bilateral extensor plantar responses. There were multiple petechial and purpuric skin and nail bed lesions in varying stages of evolution. Carotid angiography on January 4, 1973, revealed multiple intracranial arterial occlusions with delayed collateral filling, primarily in the distribution of the ascending frontoparietal branch of the right middle cerebral artery (Fig. 6–8) and complete occlusion of the proximal left middle cerebral artery. Laboratory studies were consistent with acute disseminated intravascular coagulation, with platelet count of 78,000, prothrombin time of 16 sec. over 12 sec., PTT of 59 sec. over 34 sec., fibrinogen 155 mg. per cent. Fibrin split products were present. Heparin therapy was begun, with prompt remission in his hematologic abnormalities in all areas. Over 5 days he became more alert, regained strong purposive movements in all 4 extremities, but remained mute, incessantly restless, and unresponsive to all verbal and visual stimuli. He showed no responses to visual threat, or to optokinetic tape movements. Funduscopic and pupillary findings remained normal. He slowly improved to the point of ambulation without support, but mutism, cortical blindness and probable cortical deafness persisted. Despite initial good response to continuous Heparin therapy over the first 6 weeks, he showed an abrupt relapse in his coagulation abnormalities in the setting of a gram negative septicemia, became obtunded and developed left lower extremity gangrenous changes. Increased Heparin dosage again controlled his coagulation abnormalities, but his general condition deteriorated and he

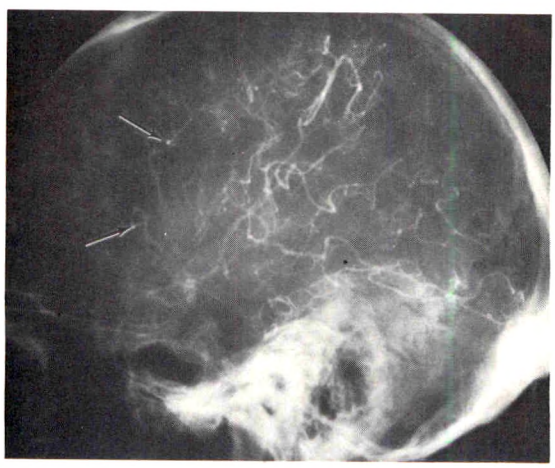


FIG. 7. Case III. Right carotid arteriogram, late arterial phase. Persistent opacification of end-on vessels indicating slowing of circulation and occlusion of peripheral vessels (arrows).

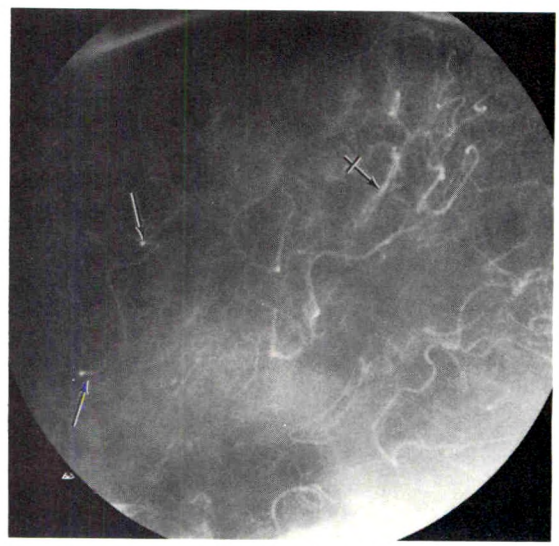


FIG. 8. Case III. Magnification view of the right frontal area demonstrating the occluded vessels (arrows) in the capillary phase of the arteriogram. There is "smudging" along the course of some vessels suggesting extravasation of contrast material (crossed arrow).

died 2 weeks later following prolonged hypotension.

Case iv. R.S.—039877, a 49 year old white male with a 2 year history of thrombocytopenic purpura, had undergone splenectomy 2 months before, with return of platelet counts to normal values. In this setting, he developed a pulmonary embolism, a rapid fall in platelets to 50,000, and positive tests for fibrin split products, and was admitted elsewhere for anticoagulation therapy. The day following discharge, he complained of headache, vomiting, and difficulty using his left hand. On the following day, he developed reiterative focal seizures in the left upper extremity. On neurologic examination, he was alert and oriented, despite repetitive stereotyped focal seizures, occurring about every 15 minutes and characterized by tonic head and ocular deviation to the left, and clonic twitching of the left face, which spread to the left arm, and then gradually abated over 10–15 seconds. Funduscopic examination showed mild blurring of both disk margins, with a single small linear hemorrhage. There was paralysis of the left arm, and moderate weakness of the left leg on voluntary movement. There was a left extensor plantar response. Perception of all sensory modalities was impaired in the left arm and leg. Lumbar puncture showed an opening pressure of 360 mm.

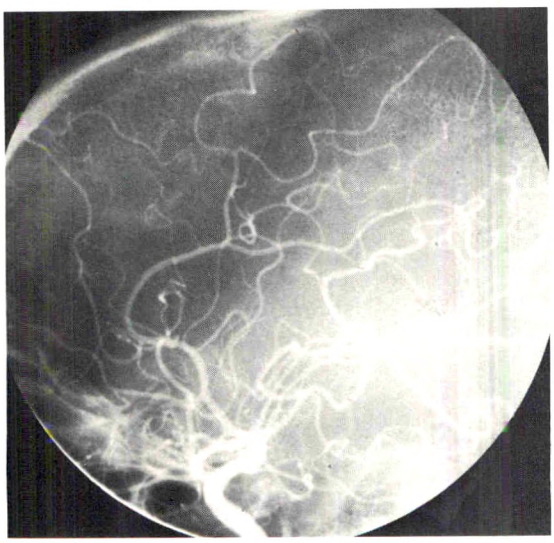


Fig. 9. Case iv. *March 22, 1973.* Right carotid arteriogram. Multiple occlusions of small and intermediate size vessels in the frontal and parietal lobes.

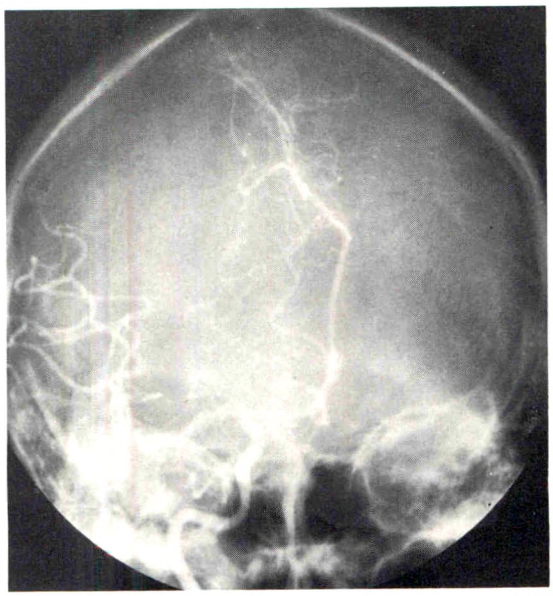


Fig. 10. Case iv. *March 24, 1973.* Right carotid arteriogram. Anteroposterior projection. There is a massive shift of the pericallosal artery from right to left.

water, with 11,750 red blood cells and 37 white blood cells, predominantly polymorphonuclear. The supernatant was xanthochromic. A right carotid arteriogram on March 22, 1973, showed multiple occlusions of smaller cerebral arteries in the frontal and parietal areas, without evidence of mass effect (Fig. 9). Laboratory studies revealed a platelet count of 25,000, but all other screening studies for consumption coagulopathy were normal. Despite anticonvulsant therapy, focal seizures persisted through the next 48 hours, but he remained neurologically stable until the fourth hospital day, when he developed acute obtundation, bradycardia and a complete right third nerve palsy involving both pupillary and oculomotor functions. A repeat right carotid arteriogram on March 24, 1973, demonstrated a large avascular mass in the right frontal lobe consistent with a deep parenchymal hematoma. Multiple occlusions of branches of the middle cerebral artery were also seen (Fig. 10–12). An emergency right frontal craniotomy was performed with evacuation of approximately 200 cc. of clotted blood. He remained unresponsive, with dilated, fixed pupils, decerebrate motor response to painful stimuli, and bilateral extensor plantar responses. He showed relentless neurologic deterioration consistent with trans-

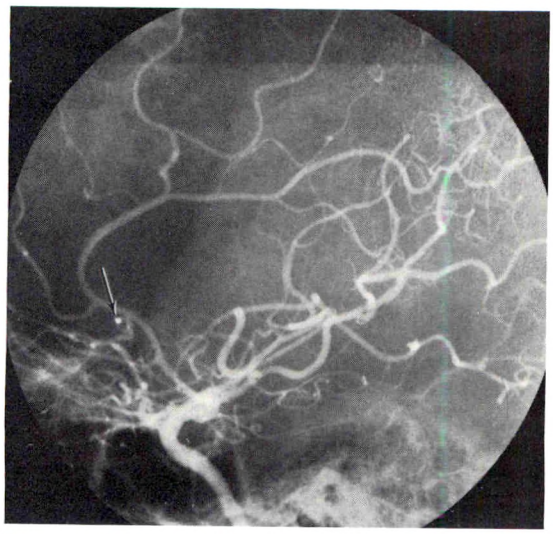


FIG. 11. Case IV. *March 24, 1973.* Right carotid arteriogram. Magnification roentgenogram. There are major intermediate arterial occlusions present in the distribution of the ascending frontoparietal artery. Persistent end-on opacification of vessels indicating occlusions (arrow).

tentorial herniation, and expired on the 5th hospital day.

At postmortem examination, the brain showed multiple and bilateral cerebral hemispheric lesions consistent with hemorrhagic infarction, and multiple intraarterial thrombi and fibrin platelet aggregates.

DISCUSSION

Disseminated intravascular coagulation is a pathophysiologic state which is seen in a variety of disease processes. In DIC, fibrin thrombi occur initially in vessels of microscopic size and then subsequently involve larger vessels.[1,3,6,9] At autopsy, many of these patients have marantic endocarditis, and embolization of fibrin clots to the major intracranial arteries undoubtedly accounts for some of the findings. The spectrum of angiographic changes is a manifestation of the severity of the intravascular thrombotic process and the severity of endothelial damage to the involved vessels. There are no pathognomonic angiographic signs of disseminated intravascular coagulation in the microcirculation of the brain. The cerebral angiographic changes include major vessel occlusions such as the middle cerebral artery. More commonly, intermediate size vascular occlusions are seen. When these intermediate vessels are not completely occluded, stasis is demonstrated with slowing of the circulation in the distribution of these vessels. Intraluminal filling defects can be seen in the intermediate sized vessels, particularly on magnification films. Another manifestation of stasis is the persistence of opacification of the end-on lumen of involved vessels in the capillary and even in the venous phases of the arteriogram. Collateral circulation *via* the leptomeningeal vessels is sluggish and may be absent. Frequently a blush is present around involved vessels which may represent extravasation of contrast material through the damaged endothelium.

Luxury perfusion[8] can be present at the periphery of involved areas of brain. Occasionally, streaming is seen in cortical veins and cortical venous thromboses occur. There is a remarkable absence of spasm or narrowing in small vessels and indeed dilatation of patent arteries is frequently seen. This is in contradistinction to the pattern in angiitis or drug abuse pa-

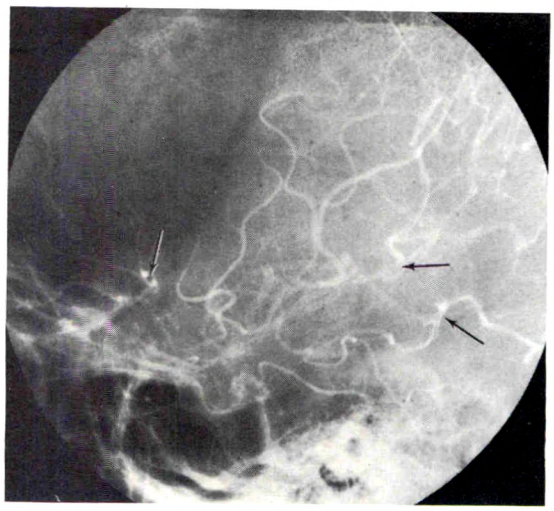


FIG. 12. Case IV. Right carotid arteriogram, capillary phase. Intraluminal filling defects are present in the occluded vessels (arrows). There is a large avascular mass in the frontal lobe.

tients,[2,11,13,14] or in experimental cerebral emboli.[12]

Finally, mass lesions are encountered which may be secondary to infarction or intracerebral hematoma.

Undoubtedly, angiographers have noted many of these changes in the past, particularly in cases of low grade intravascular coagulation. Perhaps the most distinctive change which is noted in DIC is the relatively large area of brain that is involved in comparison to the so-called stroke or usual embolic episode. Furthermore, the dilatation of proximal vessels and uninvolved vessels is unusual in most stroke syndromes and probably represents a secondary vasodilatation due to involvement of large areas of the microcirculation of the brain and perhaps its effects on the autoregulatory mechanisms of cerebral perfusion.[8]

SUMMARY AND CONCLUSIONS

We have reviewed the clinical and cerebral angiographic findings in 4 patients presenting to our Neurology Service with a fulminating clinical syndrome of acute, multifocal cerebral injury, consistent with multiple cerebral ischemic infarctions with or without a hemorrhagic component. Three had definite, and 1 had presumptive laboratory evidence of disseminated intravascular coagulation.

All showed angiographic changes of acute thromboembolic occlusion of large and medium sized intracranial arteries, involving multiple sites bilaterally, but with predilection for the middle cerebral arteries.

Postmortem examination in 2 cases has confirmed the presence of widespread cerebral and systemic thromboembolism, with both ischemic and hemorrhagic changes in brain and other organs. In both, marantic or nonbacterial endocarditis was found as well, raising the question of whether embolization of the fibrin-platelet aggregates from this lesion could alone account for the findings we described.

We suspect that endocardial involvement may determine the form that the basic syndrome may take, and is always present when cerebral infarction dominates the clinical picture in patients with the DIC syndrome. However, clinical and postmortem evidence of venous involvement in 2 of our patients would suggest *in situ* vascular deposition of fibrin products as an additional factor in pathogenesis, in some patients at least.

While the angiographic changes described are not pathognomonic for the syndrome of disseminated intravascular coagulation, they are sufficiently distinct from other forms of cerebral vascular disease in their location and distribution to strongly support the clinical diagnosis of this entity. No patient had evidence of preexisting arteriosclerotic cerebral vascular disease, nor could any other potential site for thromboemboli be found, such as a patent foramen ovale or postinfarction mural thrombus. No element of vasculitis or, for that matter, any other form of primary angiopathy was seen in the tissues studied. Angiographic findings have correlated well with the patient's neurologic findings, and with neuropathologic findings at postmortem examination.

It has been suggested that the basic pathogenetic mechanism in the syndrome can be arrested by Heparin therapy, but only if diagnosis and treatment are initiated early in the course. When, as in our patients, the syndrome presents with rapidly progressing cerebral involvement, the prognosis will remain poor, unless improved methods of diagnosis permit its recognition within its early stages. We believe that neuroradiographic studies can contribute much toward this goal.

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REFERENCES

1. Bull, B. S., and Kuhn, I. N. Production of schistocytes by fibrin strands: scanning electron microscope study. *Blood*, 1970, *35*, 104–111.
2. Citron, B. P., Halpern, M., McCarron, M.,

Lundberg, G. D., McCormick, R., Pincus, I. J., Tatter, D., and Haverback, B. J. Necrotizing angiitis associated with drug abuse. *New England J. Med.*, 1970, *283*, 1003–1011.

3. Colman, R. W., Robboy, S. J., and Minna, J. D. Disseminated intravascular coagulation (DIC): approach. *Am. J. Med.*, 1972, *52*, 679–689.
4. Colman, R. W., Braun, W. E., Busch, G. J., Dammin, G. J., and Merrill, J. P. Coagulation studies in hyperacute and other forms of renalallograft rejection. *New England J. Med.*, 1969, *281*, 685–697.
5. Engel, A., Alexander, B., and Pechet, L. Activation of trypsinogen and plasminogen by thrombin preparations. *Biochemistry*, 1966, *5*, 1543–1551.
6. Kwaan, H. C. Disseminated intravascular coagulation. *M. Clin. North America*, 1972, *56*, 177–191.
7. Kwaan, H. C. Disorders of fibrinolysis. *M. Clin. North America*, 1972, *56*, 163–176.
8. Lassen, N. A. Luxury-perfusion syndrome and its possible relation to acute metabolic acidosis localised within brain. *Lancet*, 1966, *2*, 1113–1115.
9. McKay, D. G. Disseminated Intravascular Coagulation: An Intermediary Mechanism of Disease. Hoeber Medical Division, Harper & Row, New York, 1965.
10. McKay, D. G., and Margaretten, W. Disseminated intravascular coagulation in virus diseases. *A.M.A. Arch. Int. Med.*, 1967, *120*, 129–152.
11. Rumbaugh, C. L., Bergeron, R. T., Scanlan, R. L., Teal, J. S., Segall, H. D., Fang, H. C. H., and McCormick, R. Cerebral vascular changes secondary to amphetamine abuse in experimental animal. *Radiology*, 1971, *101*, 345–351.
12. Rumbaugh, C. L., Davis, D. O., and Gilson, J. M. Experimental cerebral emboli angiographic evaluation of autologous emboli in dog. *Invest. Radiol.*, 1968, *3*, 330–336.
13. Rumbaugh, C. L., Bergeron, R. T., Fang, H. C. H., and McCormick, R. Cerebral angiographic changes in drug abuse patient. *Radiology*, 1971, *101*, 335–351.
14. Weiss, S. R., Raskind, R., Morganstern, N. L., Pytlyk, P. J., and Baiz, T. C. Intracerebral and subarachnoid hemorrhage following use of methamphetamine ("speed"). *Internat. Surg.*, 1970, *53*, 123–127.
15. Wilner, G. D., Nossel, H. L., and Leroy, E. C. Activation of Hageman factor by collagen. *J. Clin. Invest.*, 1968, *47*, 2608–2615.

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CEREBRAL ANGIOGRAPHY IN MOMENTARY CARDIAC ARREST AND SYSTEMIC HYPOTENSION IN THE DOG*

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PROLONGATION of the arterial phase during serial cerebral angiography occurs not only in marked intracranial hypertension, systemic hypertension, and diffuse arteriolar-capillary blockage,[20] but also in syncope, such as vaso-vagal syncope, carotid sinus syncope, and cardiac standstill, as a result of generalized cerebral ischemia. The latter conditions are apparently due to a marked reduction of blood pressure which produces the stasis of contrast medium in the arterial tree.

In the present study, serial cerebral arteriography was performed in dogs during momentary cardiac arrest induced by acetylcholine chloride, and during systemic hypotension induced by infusion of Arfonad (trimethaphan). This was done to confirm our clinical observations and to correlate the degree of hypotension with the duration of prolongation of the arterial phase.

MATERIAL AND METHOD

Sixteen dogs weighing 14–25 kg. were used; 12 for momentary cardiac arrest and 4 for hypotensive study. The animals were anesthetized with intravenous sodium pentothal, 25 mg./kg. body weight and additional doses were given when necessary.

During the entire procedure, each animal was intubated and ventilated with 40 per cent oxygen and 60 per cent air. Blood gas analysis was performed immediately prior to each cerebral angiography. The P_aCO_2 was held between 30 and 40 mm./kg. by adjusting the respirator.

Bilateral paratracheal incisions were made to expose both common carotid arteries and 1 of the internal jugular veins. Two catheters were inserted into each common carotid artery near the carotid bifurcation for injection of contrast medium and recording of blood pressure. A third catheter was inserted into the superior vena cava for injection of acetylcholine chloride and trimethaphan.

Blood pressures and electrocardiograms (ECG) were monitored on a Sanborn two-channel recorder. Electroencephalography (EEG) was recorded in those animals in which momentary cardiac arrest was done. Steel needles were inserted into holes drilled through the skull and were connected to an EEG amplifier in the polygraph. Standard recordings were taken from frontoparietal and parieto-occipital areas using the bipolar technique.

Momentary cardiac arrest was induced by rapid injection of acetylcholine chloride (0.1–1.0 mg./kg.) into the superior vena cava. Each dog had 2 to 3 cerebral angiograms performed: at the beginning, during, and at the end of the cardiac arrest. Angiograms were done in the lateral view by means of Sanchez-Perez film changer, using 2:1 magnification technique.

In each of 8 dogs, 12 exposures were made in 6 seconds at an 0.5 second interval. In each of 4 dogs, 12 exposures were made in 12 seconds at 1.0 second intervals. Twelve control angiograms were also obtained. Ten cubic centimeters of methylglucamine diatrizoate and sodium diatrizoate (Renografin 60) was injected in 1

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second by a Cordis injector.

Systemic hypotension was produced in 4 dogs by rapid infusion of Arfonad (0.05 mg./ml. in 5 per cent glucose in water) into the superior vena cava, thus controlling the rate of blood pressure fall. Each dog had 4 angiograms taken at mean blood pressures of 10–50 mm. Hg. Blood pressure was lowered in steps of approximately 10 mm. Hg. Cerebral angiograms were obtained at each step reduction. After each study, blood pressure was elevated and maintained at control level by the intravenous infusion of Aramine solution. Twelve exposures in 6 seconds were obtained in those animals in which the mean arterial pressure was above 30 mm. Hg, and 12 exposures in 12 seconds were obtained in those animals in which the mean arterial pressure was below 30 mm. Hg. The EEG activity was not recorded.

RESULTS

Cardiac arrest occurred within 3 to 8 seconds after the beginning of the injection of acetylcholine solution and continued for 3 to 25 seconds.[19] A few heart beats were usually present before the end of the cardiac arrest. The duration of the cardiac arrest was proportional to the dose of the injection.[19] In most cases, bradycardia and tachycardia were present in the recovery period. Bradycardia persisting for a few seconds followed by 1–2 minutes of tachycardia or 1–2 minutes of bradycardia without associated tachycardia was observed. Atrioventricular block, idioventricular rhythms, and atrial fibrillation were also often present.

The mean carotid pressure fell rapidly from 85–125 mm. Hg to 40–70 mm. Hg within a second, and it further dropped to baseline in 3–4 seconds following the onset of asystole. In some animals the carotid pressure remained in the 10–30 mm. Hg range for about 1 minute before returning to normal. However, in others, the pressure gradually returned to normal levels as soon as cardiac contraction began. A complete recovery of the carotid pressure required 2–6 minutes after resumption of cardiac contractions.

When the cardiac arrest persisted for less than 7 seconds, there were usually no EEG abnormalities.[1,12] However, in those animals in which the cardiac arrest was longer than 8 seconds, or whose pressure remained at a low level even after the arrest, EEG changes were noted. These changes consisted of slow waves beginning 10–20 seconds after the cardiac arrest and continuing for 5–20 seconds. Following this, an isoelectric EEG was usually present and continued for 10–25 seconds (Fig. 1). Cerebral electrical activity reappeared within a few seconds after return of the heart beat. However, in those animals in which the pressure continued to remain at a low level (10–30 mm. Hg), the EEG activity did not recover until the pressure was elevated above 40 mm. Hg.

With systemic hypotension induced by infusion of Arfonad, the heart rate (control range 130–180 beats/min.) fell by 10–20 per cent within 2 minutes of the injection. In 2 animals, cardiac arrest occurred when the mean arterial pressure dropped below 25 mm. Hg for more than a few minutes. Myocardial insufficiency (ST elevation and T wave inversion) was occasionally seen, and usually returned to normal within a few minutes of the end of profound hypotension.

Angiographic findings in control dogs. The entire angiogram, from the beginning of the arterial phase to the end of the venous phase, seldom lasted more than 6 seconds. The arterial phase usually required 2–3 seconds followed by an equal period of time for the venous phase. The capillary phase was extremely short and rarely well visualized (Fig. 2).

Angiographic findings during momentary cardiac arrest and during systemic hypotension. The most characteristic finding during momentary cardiac arrest was a prolonged arterial phase in both the intracranial and extracranial arteries during the 6 or 12 second course of the study. Depending upon when the contrast medium was injected and how long the severe hypotension persisted (10 to 30 mm. Hg), prolongation of

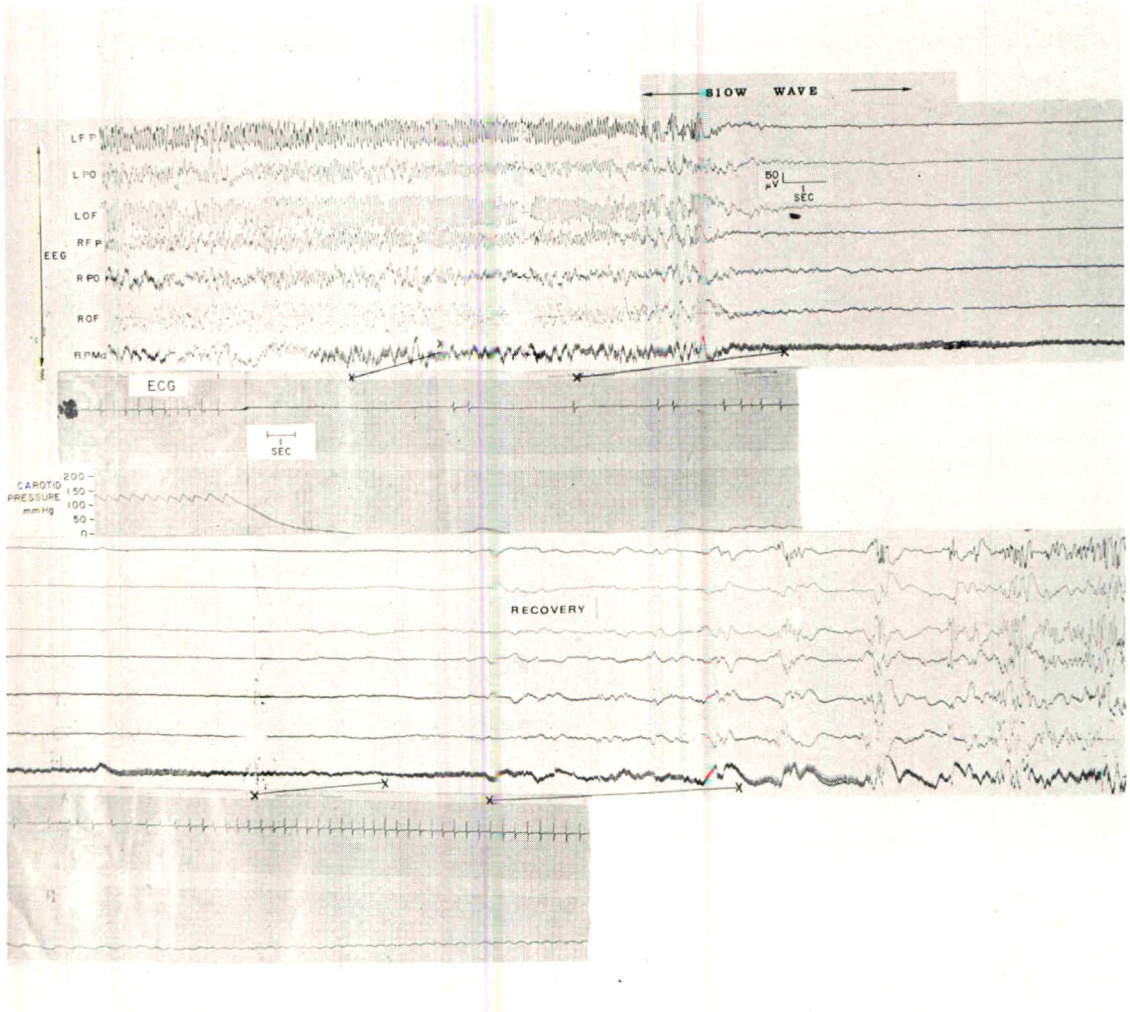


Fig. 1. Simultaneous recording of EEG (electroencephalogram), ECG (electrocardiogram), and carotid pressure during the rapid injection of acetylcholine (0.7 mg./kg.) into the superior vena cava. Cardiac arrest occurs 4 seconds after the injection and continues for 18 seconds. Carotid pressure falls rapidly from 125 mm. Hg to 60 mm. Hg in 1 second and it further drops to 10 mm. Hg in 3–4 seconds. The pressure continues to remain at a low level for 2½ minutes before returning to normal. Slow waves in the EEG appear in 10 seconds and remain for 6 seconds. Following this, an isoelectric EEG is present and remains for 16 seconds. Reference recording is taken from right parietal to right mandible. Suppression and burst effect is seen in the recovery period. (Horizontal bar 1.0 sec.; vertical bar 50 μV; R = right; L = Left; FP = frontal parietal recording; PO = parietal occipital; OF = occipital frontal.)

the arterial phase lasted the entire course of the study, even after the cardiac contractions recovered (Fig. 3; 4; and 5). Although the arterial trees appeared to be denser than those in the control studies, no or only slight dilatation of the vessels was demonstrated. However, when large doses of acetylcholine (>0.9 mg./kg.) were used, significant dilatation was invariably noticed. With a mean arterial pressure of 40 mm. Hg after restoration of cardiac contraction, angiograms demonstrated slight prolongation of the arterial phase (3–4 sec.). With a mean arterial pressure of 50 mm. Hg, prolongation was not seen.

Trimethaphan (Arfonad) is a ganglionic blocking agent[23] which also has a direct vasodilator effect.[21] When the mean blood pressure was lowered to about 30 mm. Hg by this drug, the arterial phase continued

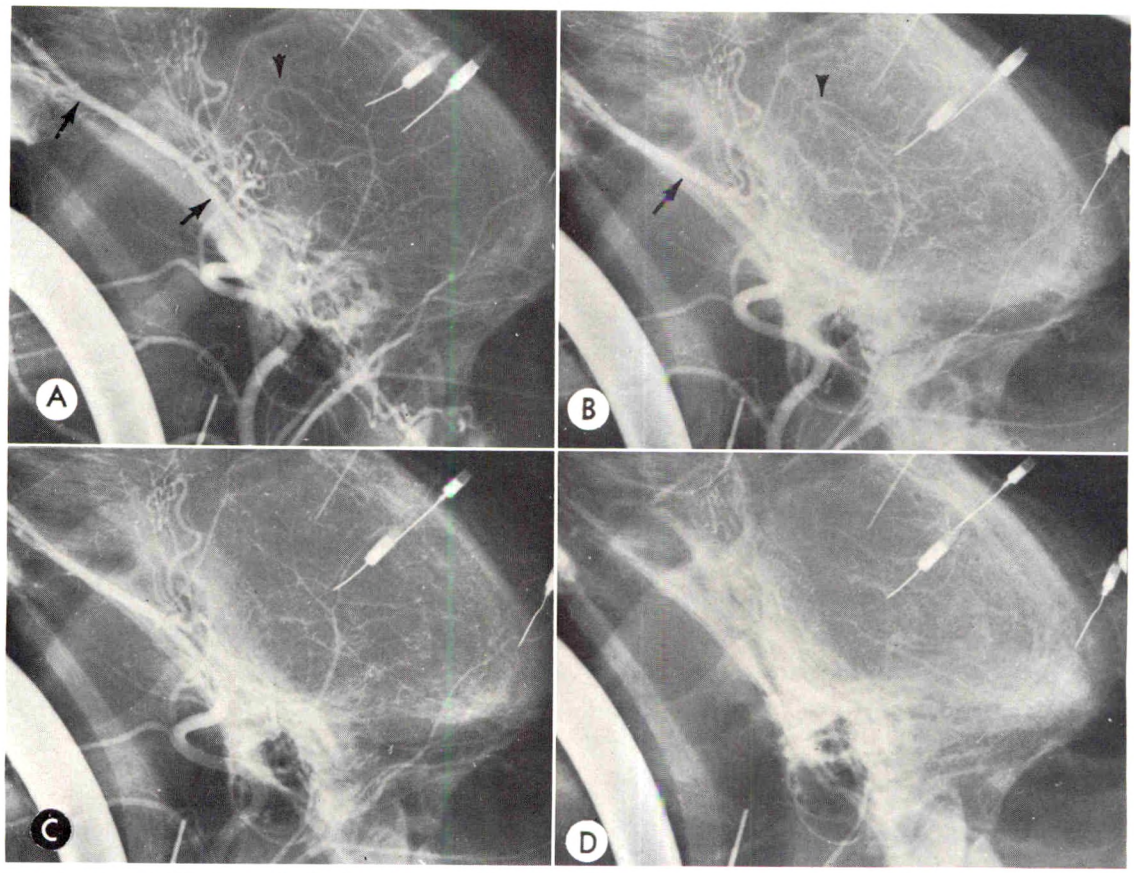


FIG. 2. Normal angiographic findings in control dog. (*A*) 1 sec., (*B*) 2.5 sec., (*C*) 3 sec., (*D*) 5 sec. after injection. The branches of the external carotid artery are markedly enlarged, especially the branches of the internal maxillary artery (arrows) while the intracranial arteries are surprisingly small (arrowhead represents the anterior cerebral artery). The arterial phase usually requires 2–3 seconds followed by an equal period of time for the venous phase. The capillary phase is extremely short.

for 4–5 seconds. It remained longer at the pressure of below 30 mm. Hg. Prolongation was usually not present at a pressure of 40 mm. Hg (Fig. 6; and 7). In 1 dog, prolongation of the arterial phase was not seen even at a pressure below 30 mm. Hg until after bradycardia. No visible or only slight dilatation of the intracranial vessels was noticed.

DISCUSSION

Changes in cortical electrical activity are a consequence of deficient oxygenation of the central nervous system following circulatory changes evoked by exogenous drugs. The changes secondary to administered acetylcholine are similar to those induced by asphyxiation,[1] which produces a combination of cerebral anoxic anoxia and acidosis caused by CO_2 retention in the blood. Meyer and Marx[22] explained that EEG changes secondary to asphyxic anoxia appeared to depend primarily upon anoxic brain damage. The critical level for mean cortical pO_2 was 10 mm. Hg. When the cortical pO_2 fell below 6–8 mm. Hg, there was a rapid efflux of sodium ions from the extracellular space and an increase in the extracellular potassium ion activity, as well as EEG changes of diffuse slow waves of increased amplitude. When the cortical pO_2 fell further, between 2–4 mm. Hg, the sodium and potassium ion movements increased, and the EEG became isoelectric. Prompt restoration of respiration or cerebral blood flow resulted in recovery of

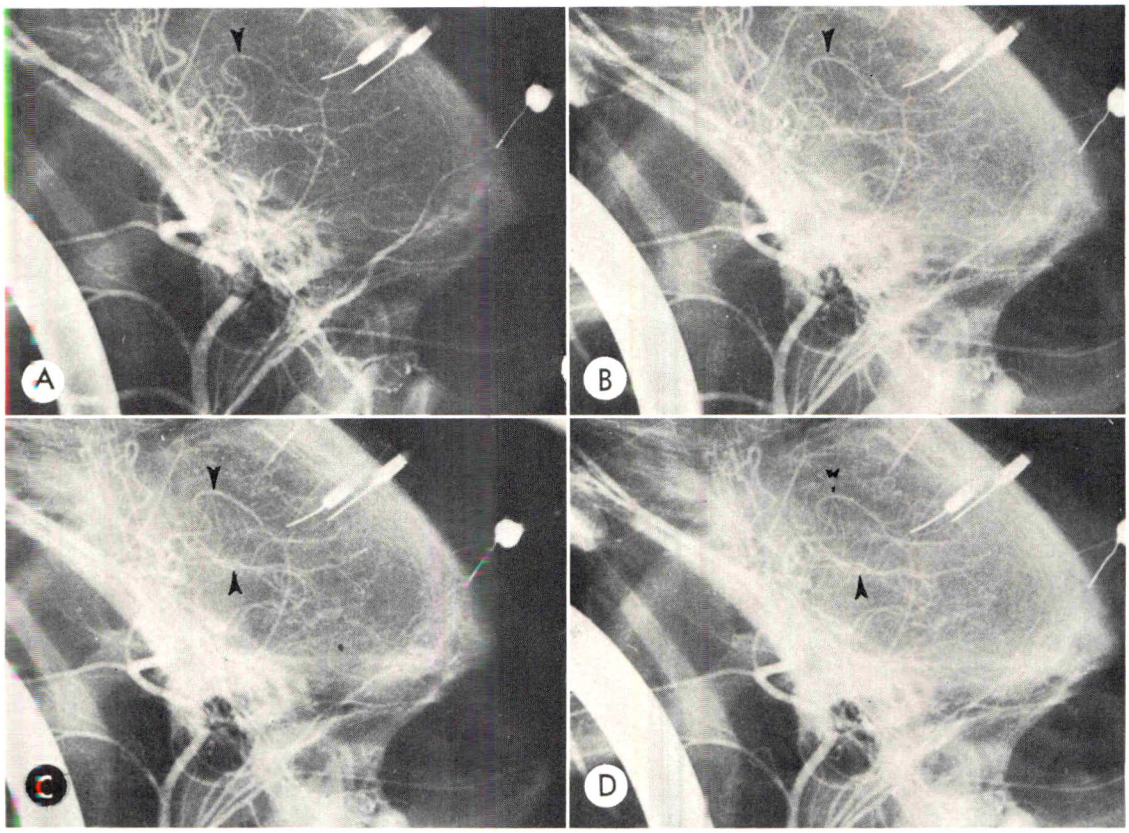


FIG. 3. Angiographic findings during momentary cardiac arrest. (*A*) 1 sec., (*B*) 3 sec., (*C*) 4.5 sec., (*D*) 5.5 sec. after injection. When angiography is performed immediately after the onset of the 8 second cardiac arrest, prolongation of the arterial phase lasts the entire 6 second course of study. No visible dilatation of the vessels is noted (arrowheads represent the anterior and middle cerebral arteries). Multiple radiopaque needles were used for EEG recording.

cortical oxygen tension, ionic homeostasis, and EEG activity. The EEG findings in this study appear to be compatible with others.[1,12,28] Recovery time of the EEG appeared to be directly related to duration of hypotension and total amount of drugs given. Wiederholt *et al.*,[28] in their study of the effect of systemic hypotension upon the cerebral electrical activity, observed that an isoelectric EEG presented in 13 seconds after the mean arterial pressure dropped from 130 mm. Hg to 30–40 mm. Hg by rapid intravenous infusion of Arfonad (trimethaphan). However, the EEG activity was only transiently depressed when hypotension was induced by hemorrhage. They concluded that profound systemic hypotension alone should not be responsible for loss of EEG activity.

Autoregulation of the cerebral blood flow circulation was first described by Fog in 1934.[9] Using the pial-window technique of Forbes,[11] he observed arteriolar constriction when systemic blood pressure was elevated and *vice versa*. Since then, several different observations pertaining to the pressure-flow relationships of the brain have been published. However, the mechanism of autoregulation of the cerebral circulation has still remained a subject of debate. Basically, 3 theories have been emphasized: (1) the myogenic theory.[5,6,10,13,15] It is based on the contraction observed in other muscles in response to stretch. Bayliss[2] postulated that the smooth muscle cells of the arteries or arterioles reacted in a similar way to an increase in internal pressure. Raised intramural pressure potenti-

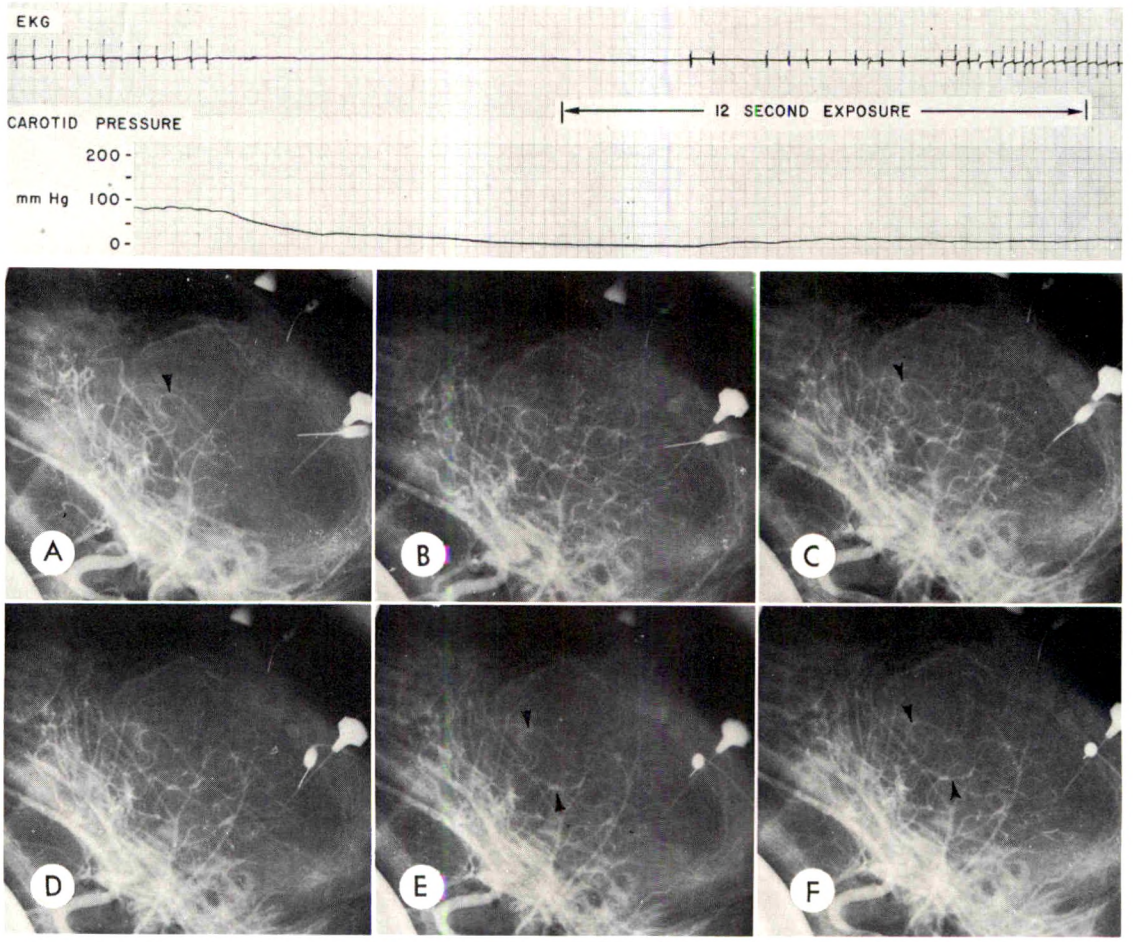


FIG. 4. (*A*) 2 sec., (*B*) 4 sec., (*C*) 6 sec., (*D*) 8 sec., (*E*) 1; sec., (*F*) 11 sec. after injection. When angiography is performed at the end of cardiac arrest, while the pressure remains at a low level (below 25 mm. Hg), prolongation of the arterial phase lasts the entire 12 second course of the study, even after cardiac contractions begin again (arrowheads represent the anterior and middle cerebral arteries).

ates vasomotion which leads to a rise in vascular resistance and *vice versa*. The precapillary resistance vessels are considered to be the site of the changes in resistance. (2) The metabolic theory.[14,17,24] It implies that autoregulation is a flow-dependent phenomenon. The flow passively follows changes in the perfusion pressure, leading to an altered chemical state in the tissue. Secondarily, the change in concentration of the metabolites induces changes in the caliber of the resistant vessels, which act to keep the flow constant. Hypoxia and hypercapnia induce vasodilatation and hypocapnia vasoconstriction. (3) The tissue-pressure theory.[5,15,25] According to this hypothesis, autoregulation is purely a mechanical event. It occurs in the low pressure vessels by the pressure of tissue fluid on them. When the arterial blood pressure is raised, there is an increase in outflow of fluid from the capillaries into the extravascular space. This outpouring of fluid will cause the perivascular pressure to increase. Since the venous vessels have the lowest internal pressure and little rigidity, they would be most vulnerable to changes in tissue pressure, tending to collapse and thereby raise the postcapillary resistance. In recent years, the 2 former theories have re-

ceived wider support than the latter. Another theory is that autoregulation of the cerebral blood flow may be influenced by the autonomic nervous system.

A normal cerebral blood flow can be maintained over a fairly wide range of arterial blood pressures. Kleinerman *et al.*[16] observed that during high spinal anesthesia the mean arterial blood pressure in normotensive patients fell 30 per cent (from 93–63 mm. Hg) from the control value, which did not change cerebral blood flow significantly. A similar observation was made by Dewar *et al.*[4] under the action of hexamethonium. Finnerty *et al.*,[8] in their study of cerebral ischemia induced by intravenous infusion of hexamethonium, found that when cerebral ischemia occurred it disclosed the fol-

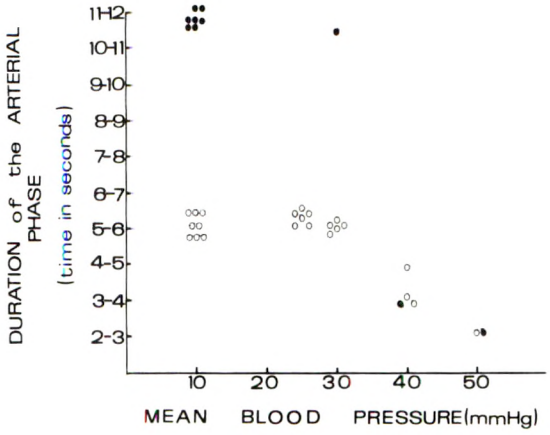


FIG. 5. Relationship between the duration of prolongation of the arterial phase and the severe systemic hypotension induced by infusion of acetylcholine chloride (○ = 6 second course of the study; ● = 12 second course of the study).

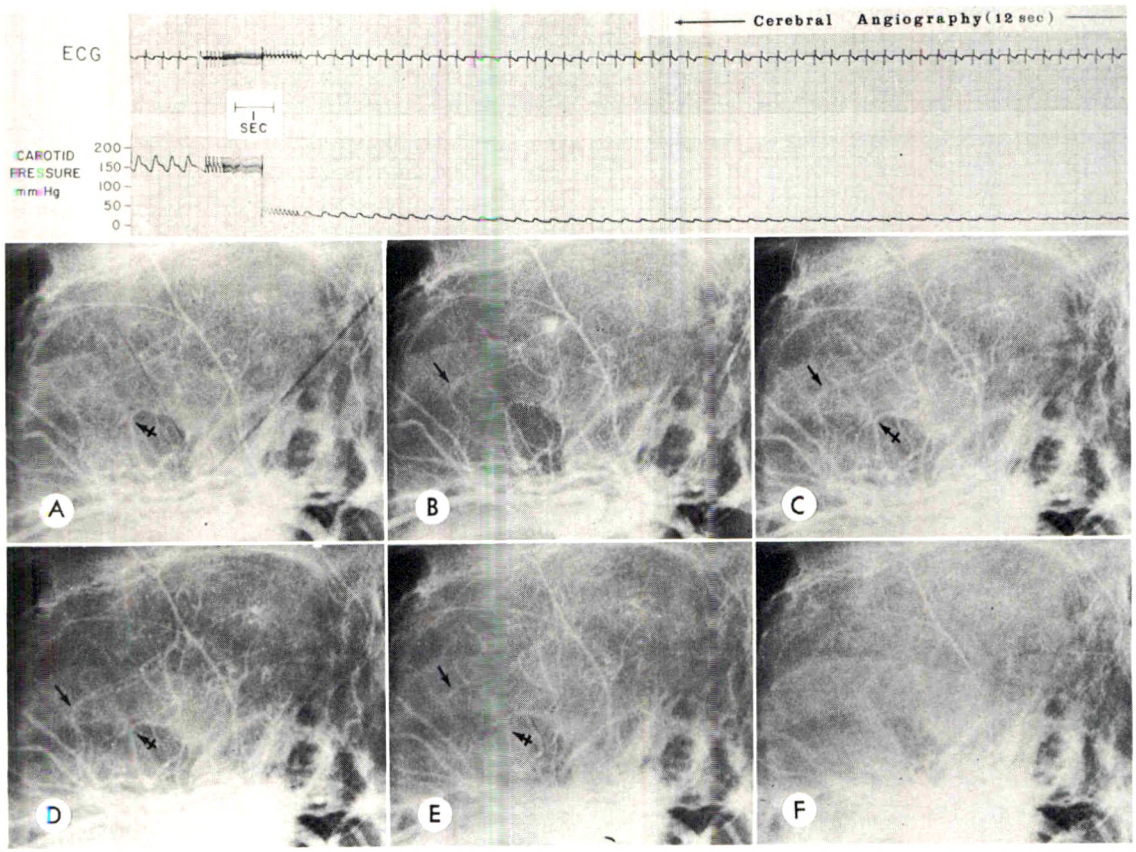


FIG. 6. (*A*) 2 sec., (*B*) 3 sec., (*C*) 4 sec., (*D*) 5 sec., (*E*) 6 sec., (*F*) 7 sec. after injection. Angiographic findings during severe systemic hypotension induced by infusion of Arfonad in the presence of normal cardiac function. The 12 second course of the angiography was performed at a mean pressure of 30 mm. Hg, the duration of the arterial phase is 5 to 6 seconds.

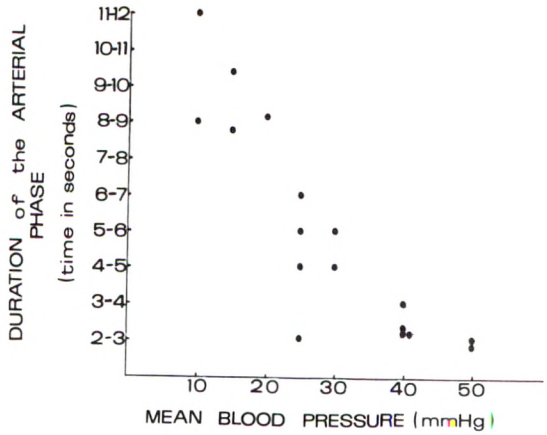


FIG. 7. Relationship between the duration of prolongation of the arterial phase and the severe systemic hypotension induced by the infusion of Arfonad.

lowing: (1) the mean arterial pressure fell from a mean value of 109 mm. Hg to 48 mm. Hg; (2) the rate of cerebral blood flow decreased significantly from a mean of 51 to 31.5 ml./100 gm./min.; (3) the mean cerebral vascular resistance also fell significantly from 2.2 to 1.5; and (4) the cerebral oxygen consumption was not changed appreciably from control values. Although considerable variation was seen in the level of arterial pressure at which signs of cerebral ischemia occurred, it ranged from a mean value of 89 mm. Hg in the malignant hypertensive group to 29 mm. Hg in the normotensive group over 60 years of age. The value of cerebral blood flow was not appreciably different in the individual groups studied. Harper[14] observed that in mongrel dogs, the mean blood pressure was reduced from the average initial value of 155 mm. Hg to 80–90 mm. Hg without any significant alteration in blood flow. Thereafter, the blood flow declined with the blood pressure. Schallek and Walz,[27] in their study of the effect of Arfonad induced hypotension on the electroencephalogram, concluded that the rate of blood pressure fall with trimethaphan determined whether or not EEG activity was abolished; the slower the drop, the less effect on the EEG. In contrast, Wiederholt *et al.*[28] reported that EEG activity always disappeared below 40 mm. Hg regardless of the rate of drop of blood pressure.

Brierley *et al.*[3] found that if the cerebral perfusion pressure in animals (rhesus monkey) had fallen to 25 mm. Hg, maximal vasodilatation was observed through the cranial window because the cerebrovascular resistance had probably reached a minimal value. Thereafter, any change in arterial PCO_2 or pH did not affect the calibers of cerebral arteries or arterioles. A similar observation was made by Harper[14] in the dog, *i.e.*, if mean arterial pressure was lowered to 50 mm. Hg, cortical blood flow was not influenced by changes in arterial PCO_2.

Ekström-Jodal *et al.*[6,7] considered a mean arterial pressure of 50 mm. Hg as the lower limit of autoregulation in the dog. In their study of pressure flow relationship in hypotension in the dog induced by either papaverine infusion or by carbon dioxide inhalation, they observed that when the arterial blood pressure was lowered there was a successive decrease in the cerebrovascular resistance. However, in hypotension below the autoregulatory range, there was a marked increase in cerebrovascular resistance. The relation between the pressure and the flow was found to be curvilinear with the convexity toward the pressure axis, which was in contrast to the linear relationship observed by others.[14,26]

Prolongation of the arterial phase during momentary cardiac arrest is apparently due to a marked reduction of the arterial pressure, which causes a simultaneous marked decrease in blood flow. Secondarily, this severely impaired cerebral autoregulation produces a stasis of injected contrast medium in the arterial tree. A similar finding is also seen in profound systemic hypotension induced by Arfonad, but to a lesser degree. The duration of the prolonged arterial phase correlates with the degree of hypotension below the mean blood pressure of 40 mm. Hg (Fig. 5; and 7). Of further interest was that 1 animal, in whom profound hypotension (25 mm. Hg) was induced rapidly (within 1 minute) by Arfonad, had no significant prolongation of the arterial phase until bradycardia developed.

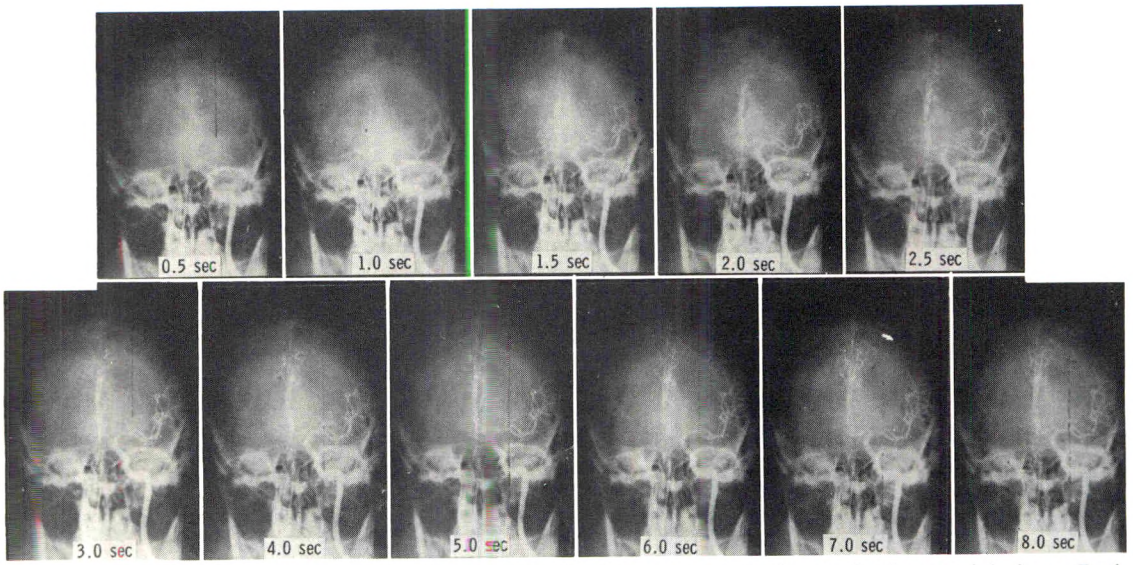


Fig. 8. Serial anteroposterior carotid angiograms obtained over 8 seconds show only the arterial phase. Both anterior cerebral arteries are filled from the injected left side. The right middle cerebral artery is opacified at 1.0 seconds through 2.0 seconds. The posterior cerebral arteries are partially filled at 1.5 seconds. Contrast opacification of the internal carotid, both anterior cerebral arteries, and left middle cerebral artery is seen throughout the study. (Courtesy of Lee *et al.*, *Neuroradiology*, 1973, *5*, 233–236.[18])

No visible or only slight dilatation of the vessels is noticed in both conditions. Vasodilatation is most likely secondary to the effects of the injected drugs. Progressive vasodilatation was observed when the doses of acetylcholine chloride were increased. Because of the small size of the intracranial arteries in the dog, it seems not feasible for an accurate evaluation of the status of the vessels in hypotension, even in 2× magnification.

The present study helps clarify our clinical observation of a patient who experienced a slight headache and irregular heart beat during direct carotid arteriography. Only the arterial phase was present in the entire 8 second study (Fig. 8). This phenomenon was believed to be due to momentary cardiac arrest or heart block during the angiographic procedure.[18]

SUMMARY

Serial cerebral angiography was performed in dogs during momentary cardiac arrest induced by acetylcholine and in systemic hypotension induced by trimethaphan.

During momentary cardiac arrest, prolongation of the arterial phase in both intracranial and extracranial arteries was seen in the entire 6 or 12 second course of the study, while the blood pressure was at baseline level. The duration of the arterial phase was 3–4 seconds when angiography was performed during the recovery period at a mean blood pressure of 40 mm. Hg, and prolongation of the arterial phase was not observed when the study was done at the pressure of approximately 50 mm. Hg.

In systemic hypotension in the presence of continuing cardiac function, the duration of the arterial phase lasted 4–5 seconds or longer at the mean blood pressure at or below 30 mm. Hg, and prolongation of the arterial phase was not present at the pressure of 40 mm. Hg.

The duration of prolongation of the arterial phase is well correlated with the degree of hypotension at mean arterial pressures below 40 mm. Hg in both conditions.

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REFERENCES

1. Ajmone-Marson, C., and Fuortes, M. G. F. Electrographic study of convulsant action of intravenously administered acetylcholine. *Electroencephalog. & Clin. Neurophysiol.*, 1949, *1*, 238–290.
2. Bayliss, W. M. On local reaction of arterial wall to changes of internal pressure. *J. Physiol.*, 1902, *28*, 220–231.
3. Brierley, J. B., Brown, A. W., Excell, B. J., and Meldrum, B. S. Brain damage in rhesus monkey resulting from profound arterial hypotension. I. Its nature, distribution and general physiological correlates. *Brain Res.*, 1969, *13*, 68–100.
4. Dewar, H. A., Owen, S. G., and Jenkins, A. R. Effect of hexamethonium bromide on cerebral circulation in hypertension. *Brit. M. J.*, 1953, *2*, 1017–1018.
5. Ekström-Jodal, B. On relation between blood pressure and blood flow in canine brain with particular regard to mechanism responsible for cerebral blood flow autoregulation. *Acta physiol. scandinav.*, 1970, Suppl. 350, 1–28.
6. Ekström-Jodal, B., Häggendal, E., and Nilsson, N. J. On relation between blood pressure and blood flow in cerebral cortex of dogs. *Acta physiol. scandinav.*, 1970, Suppl. 350, 29–42.
7. Ekström-Jodal, B., Häggendal, E., and Nilsson, N. J. Cerebral venous oxygen saturation during rapid changes in arterial blood pressure: oximetric study in dogs. *Acta physiol. scandinav.*, 1970, Suppl. 350, 43–61.
8. Finnerty, F. A., Jr., Witkin, L., and Fazekas, J. F. Cerebral hemodynamics during cerebral ischemia induced by acute hypotension. *J. Clin. Invest.*, 1954, *33*, 1227–1232.
9. Fog, M. Om piaarteriernes vasomotoriske reaktioner. Ejnar Munksgaard Forlag, Copenhagen, 1934, p. 183.
10. Folkow, B. Description of myogenic hypothesis. *Circulation Res.*, 1964, Suppl. 15, 279–287.
11. Forbes, H. S. Cerebral circulation I. Observation and measurement of pial vessels. *Arch. Neurol. & Psychiat.*, 1928, *19*, 751–761.
12. Gastaut, H., and Fischer-Williams, M. Electroencephalographic study of syncope: its differentiation from epilepsy. *Lancet*, 1957, *2*, 1018–1025.
13. Häggendal, E., and Johansson, B. Effects of arterial carbon dioxide tension and oxygen saturation on cerebral blood flow autoregulation in dogs. *Acta physiol. scandinav.*, 1965, Suppl. 258, *66*, 27–53.
14. Harper, A. M. Interrelationship between PCO_2 and blood pressure in regulation of blood flow through cerebral cortex. *Acta neurol. scandinav.*, 1965, Suppl. 14, 94–103.
15. Johnson, P. C. Review of previous studies and current theories of autoregulation. *Circulation Res.*, 1964, Suppl. 15, 1–9.
16. Kleinerman, J., Sancetta, S. M., and Hackel, D. B. Effects of high spinal anesthesia on cerebral circulation and metabolism in man. *J. Clin. Invest.*, 1958, *37*, 285–293.
17. Lassen, N. A. Autoregulation of cerebral blood flow. *Circulation Res.*, 1964, Suppl. 1, *15*, 201–204.
18. Lee, K. F., Lin, S. R., and Lee, J. J. Stand-still cerebral arteriogram secondary to heart block: case report with discussion of its mechanism. *Neuroradiol.*, 1973, *5*, 233–236.
19. Lin, S. R., Lee, K. F., Go, E. B., Biery, D. N., and Lin, Z. S. Cerebral angiography during acetylcholine chloride induced momentary cardiac arrest: experimental study in dogs. *Invest. Radiol.*, 1973, 413–417.
20. Lin, S. R. Angiographic study of cerebral circulation after various periods of cardiac arrest: preliminary study in dog. To be published.
21. McCubbin, J. W., and Page, I. G. Nature of hypotensive action of thiophanium derivative in dogs. *J. Pharmacol. & Exper. Therapy*, 1952, *105*, 437–442.
22. Meyer, J. S., and Marx, P. W. Pathogenesis of EEG changes during cerebral anoxia. Handbook *Electroencephalog. & Clin. Neurophysiol.*, 1972, *14-A*, 6-11.
23. Randall, L. O., Peterson, W. G., and Lehman, G. Ganglionic blocking action of thiophanium derivatives. *J. Pharmacol. & Exper. Therapy*, 1949, *97*, 48–57.
24. Rapela, C. E., and Green, H. D. Autoregulation of canine cerebral blood flow. *Circulation Res.*, 1964, Suppl. 1, *15*, 205–211.
25. Riley, R. L., Permutt, S., and Bromberger-Barnea, B. Elastic properties of tissues and autoregulation of blood flow. *Physiologist*, 1962, *5*, 203.
26. Sagawa, K., and Guyton, A. C. Pressure-flow relationship in isolated canine cerebral circulation. *Am. J. Physiol.*, 1961, *200*, 711–714.
27. Schallek, W., and Walz, D. Effect of drug-induced hypotension on electroencephalogram of dog. *Anesthesiology*, 1954, *15*, 673–680.
28. Wiederholt, W. C., Locke, G., and Yashon, D. Hypotension and its effect on EEG activity in dog. *Neurology*, 1972, *22*, 717–725.

VOL. 122, No. 3

INTRACRANIAL CALCIFICATIONS IN CHILDHOOD*

FREQUENCY OF OCCURRENCE AND SIGNIFICANCE

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THE significance of roentgenographically visible intracranial calcifications has been reported in numerous instances in both adults[20] and children.[2,16,18,24] Calcifications may be "physiologic" and therefore of no concern, or represent a change secondary to infection, degeneration, trauma, neoplasia, metabolic disease, familial disorders or medical treatment.[14] If one attempts to distinguish calcifications of importance from those of no consequence, it is imperative that the examiner be aware of the frequency with which intracranial calcifications are noted and the forms and locations of those calcifications. With that knowledge it may be assumed that the appropriate investigation can be instituted, where necessary.

An investigation was undertaken in an attempt to ascertain: (1) the frequency with which intracranial calcifications are noted in roentgenograms of children; (2) those entities in which the literature and our personal experiences indicate that calcifications do occur; (3) the appearance and location of those calcifications; and (4) the logical clinical approach to those patients in whom calcifications are noted.

MATERIAL AND METHOD

A number of patients having skull roentgenograms for specific but varied reasons had their roentgenograms reviewed for calcifications. These patients afforded the first 245 sets of skull roentgenograms analyzed in a 3 month period. No set of roentgenograms was purposely excluded. The patient's age, reason for obtaining the roentgenograms, and the presence or absence of intracranial calcifications were recorded (Table 1). The literature was then scanned for similar types of data; *i.e.*, those noting the frequency with which intracranial calcifications are noted.

The skull radiography reports of a second group of patients seen at the Dysfunctioning Child Center (D.C.C.) of Michael Reese Medical Center were reviewed for comments referable to intracranial calcifi-

TABLE I

"THE 245 SERIES"
Age Range: 1 day–17 years

	No. of Cases
Reason for Skull Roentgenography	
Trauma	93
Convulsions	42
Known or ?hydrocephalus	21
Failure to thrive	12
Cerebral palsy	11
Weakness, syncope or fainting	9
Headache	7
Microcephaly	7
Cranial asymmetry	6
Possible tumor	5
Possible metastases	5
Infection (meningitis; ?rubella, ?toxoplasmosis)	4
Mental or motor retardation	4
Others (3 patients or less): Hypoglycemia, coma, spine tumor, hypotonia, dermal sinus, skull transilluminatability, jaundice, renal disease, delayed speech, cyclic vomiting, craniostenosis, neurofibromatosis, battered baby, earache, vision problem, ataxia.	19
Patients with calcifications	
Trauma cases	3
Failure to thrive	1
Convulsions	1

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TABLE II

INTRACRANIAL CALCIFICATIONS IN THE TEACHING FILES OF MICHAEL REESE MEDICAL CENTER AND CHILDREN'S MEMORIAL HOSPITAL

1. Abscess
2. Cytomegalic inclusion disease
3. Hyperparathyroidism
4. Hypoparathyroidism
5. Hypervitaminosis D
6. Lead intoxication
7. Methotrexate (intrathecal)
8. Phakomatoses
9. Pseudohypoparathyroidism
10. Subdurals and other intracranial "bleeds"
11. Toxoplasmosis
12. Tuberculosis
13. Tuberous sclerosis
14. Tumors (all types)
15. The usual physiologic calcifications

cations. There were 1,283 different patients. Such patients are admitted to the D.C.C. if an attending physician determines that they are developing poorly physically and/or emotionally or in general "are not adjusting to the world well." All these patients were 6 years of age or younger. The most frequently seen problems were cerebral palsy, discoordinate motor function, language and/or speech problems, behavior problems and hyperkinesis. Patients with other complaints of "inappropriate adjustment" are also seen. All patients had skull roentgenograms. The literature was scanned for reports on other "select groups" in which skull roentgenogram findings were noted.

The teaching files at Children's Memorial Hospital in Chicago and Michael Reese Medical Center were searched for those skull roentgenograms indexed because of demonstrable calcifications. The etiologies of the calcifications are listed in Table II. That series and "others" documented in the literature were recorded.

RESULTS

FREQUENCY

The "245 Series." The age range of this series was "hours" to 17 years; the mean age was 4.6 years (4 years and 7 months). "Trauma" and "convulsion" cases accounted for 55 per cent of the entire series. Other large groups in the series consisted of patients with failure to thrive (12 patients), cerebral palsy (11), and hydrocephalic patients (21). Five children of 245 had calcifications demonstrated. One child with failure to thrive had pineal calcifications at age 6 months. One 11 year old had faint pineal calcifications and was being evaluated for a seizure disorder. Three children being evaluated for trauma had calcifications of the petroclinoid or interclinoid ligaments. They were 11, 14, and 16 years of age. Thus, 2 per cent of the group had calcifications. Only 2 patients with calcifications (0.9 per cent) were considered for evaluation for reasons other than trauma. Only 1 patient with calcifications was less than 6 years of age.

THE LITERATURE ON PHYSIOLOGIC CALCIFICATIONS

Willich *et al.*[24] in a review of over 34,000 children found calcifications in 0.83 per cent. In 25 per cent of those no cause was found. Peterson and Kieffer[16] stated that pineal calcifications are found in approximately 5.1 per cent of children. McRae[10] stated that habenular commissure calcifications are found almost as often as pineal calcifications. In the present series there is a frequency of less than 1 per cent. Palubinskas and Davies[14] indicated that physiologic calcifications are commonly found in children; these physiologic calcifications involve the pineal gland, the choroid plexus, and the falx. Varadarajan and Ramamurthi[23] reported that numerous structures in the skull often become partly or wholly calcified under normal circumstances (*i.e.*, physiologic). They also considered calcifications of the pineal gland, choroid plexus, dura, and the pacchionian bodies as physiologic.

THE DYSFUNCTIONING CHILD CENTER

The Dysfunctioning Child Center (D.C.C.) of Michael Reese Medical Center afforded roentgenograms of 1,283 children 6 years of age or younger. No intracranial

calcifications were noted in the reports of these roentgenograms.

THE LITERATURE ON "SPECIAL GROUPS"

In the literature on special groups with intracranial calcifications are references to patients with brain tumors, convulsions, psychiatric disorders, A-V malformations, "abnormalities" in a large series, and toxoplasmosis. In 123 children with brain tumors French[4] found calcifications in 20 per cent. These tumors included meningiomas, astrocytomas, angiomas, ependymomas, craniopharyngiomas, spongioblastomas, pinealomas, oligodendrogliomas, astroblastomas, tuberculomas, and metastatic neuroblastomas. Grossman *et al.*[5] found calcifications in 4 per cent of 50 consecutive children with brain tumors. Majd *et al.*[9] reviewed 31 children in early infancy with brain tumors. Three (10 per cent) had calcifications. These 3 patients had a craniopharyngioma, meningioma, or teratoma.

Hayes and Shopfner[6] reviewed 234 children with convulsions. One patient had a falx calcification. The remainder of the patients failed to reveal any evidence of intracranial calcifications.

Rumbaugh and Potts[19] evaluated 61 patients with A-V malformations. Thirty per cent had calcifications, but a significant number were not children.

Kraft *et al.*[8] reviewed 1,000 psychiatric patients (primarily older children and adults) and only 2 had calcifications. These 2 patients later were found to have brain tumors.

Varadarajan and Ramamurthi[23] reviewed a series of "abnormal" patients without describing the nature of the abnormality. Of 2,151 patients less than 10 years of age 0.04 per cent demonstrated pineal calcifications and no other intracranial calcifications.

Sutton[21] reported on 6 patients with toxoplasmosis all of whom had calcifications which on close inspection were strongly suggestive of toxoplasmosis. The literature according to Sutton reports that calcifications occur in toxoplasmosis at least 50 per cent of the time.

EVALUATION OF TEACHING FILES AND THE LITERATURE

The teaching files of the Children's Hospital and Michael Reese Medical Center were reviewed and revealed calcifications in the skull roentgenograms of children with the diagnoses as enumerated in Table II. Table III lists the additional diagnoses causing intracranial calcifications, according to published reports.

THE LOCATION AND MORPHOLOGY OF INTRACRANIAL CALCIFICATIONS

The morphologic variability of physiologic and pathologic calcifications as reported in the literature is great. Dural calcifications have been described as linear sheet-like, solid, flecks, etc. Pineal calcifications can be pin-point or greater than 1 cm. in diameter.[12] Calcifications can occur in any of the basal ganglia,[1] primarily the globus pallidus, and are described as mottled, solid, crescentic, faint, dense or "variable."[20] The habenular calcifications are usually described as crescentic[10] and the choroid plexus (glomus) calcifications are described as pop-corn kernel, circular[18] and stippled.[22] The teaching file roentgenograms of Michael Reese and Children's Memorial Hospitals reveal similar variability in the morphology of the calcifications.

The locations in which physiologic cal-

TABLE III

OTHER DOCUMENTED CAUSES OF INTRACRANIAL CALCIFICATIONS

1. "Abnormal" basal ganglia
2. "Abnormal" choroid plexus (with or without seizures)
3. Ecchinococcosis
4. Familial degenerative disease
5. Intracranial hemorrhage
6. Lipoproteinosis
8. Rubella
9. Tapeworm larvae
10. Torulosis

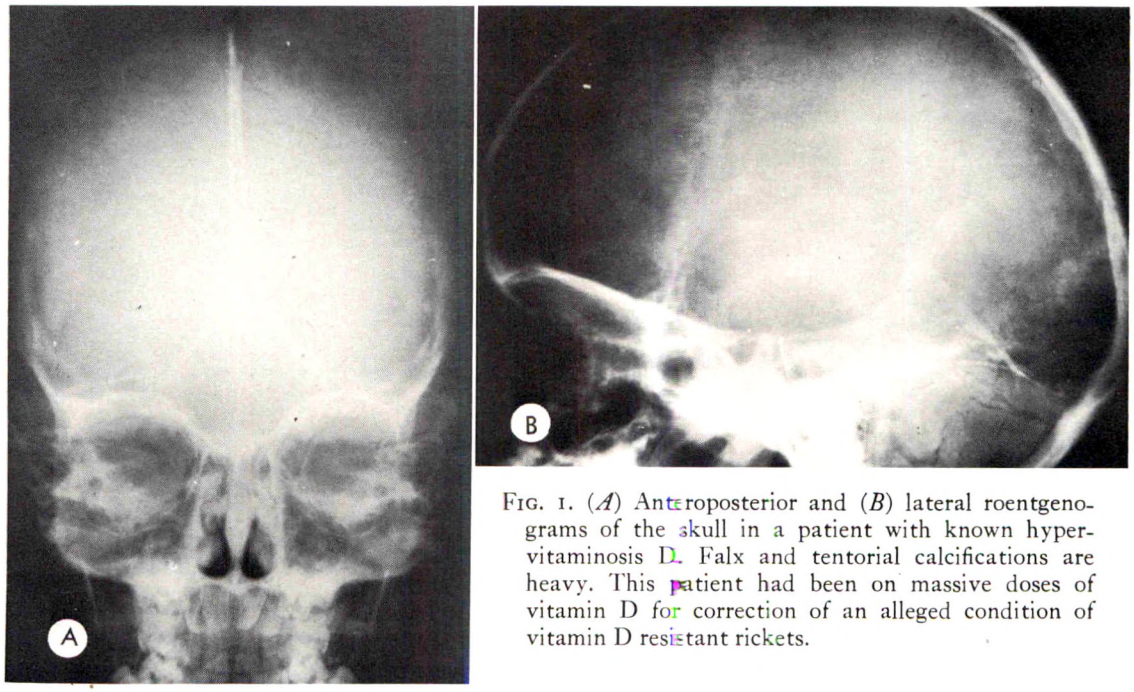


Fig. 1. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the skull in a patient with known hypervitaminosis D. Falx and tentorial calcifications are heavy. This patient had been on massive doses of vitamin D for correction of an alleged condition of vitamin D resistant rickets.

cifications occur are more specific than the morphologic characteristics.[10] Pathologic calcifications can occur in any intracranial site.

Morphologic similarities of pathologic calcifications do occur but variability in appearance is more frequent.

The analysis of our teaching file films demonstrated dural calcifications (usually considered as benign) in pathologic states such as hypervitaminosis D (Fig. 1) and lead intoxication (Fig. 2). Our films showed diffuse and dense paraventricular calcifications in 1 case of cytomegalic inclusion dis-

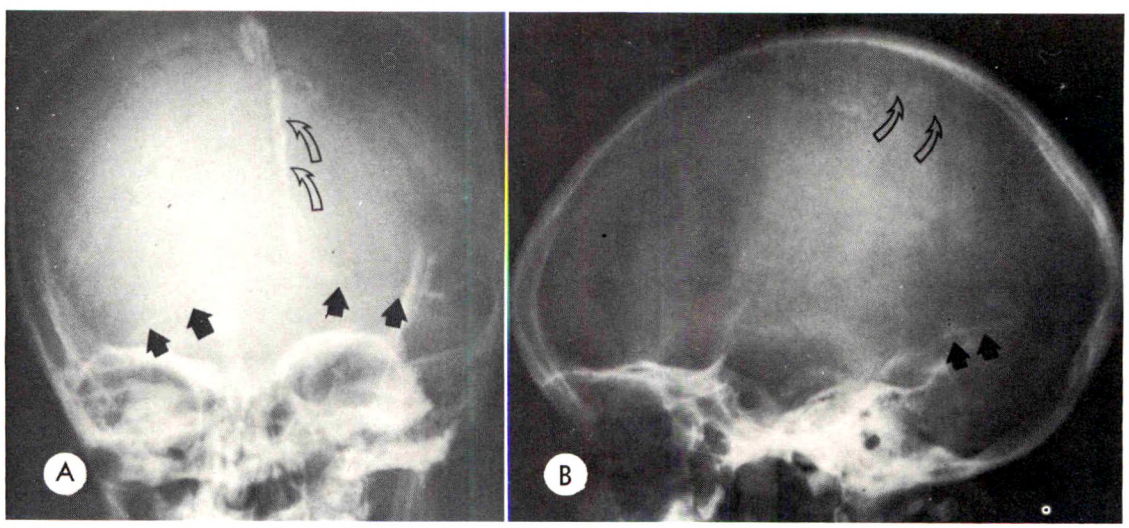


Fig. 2. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the skull in a patient with lead intoxication. Calcifications of the falx and tentorium are present and simulate the calcifications noted in the patient with hypervitaminosis D (Fig. 1). The specific explanation of extensive dural calcification in this patient with lead intoxication is unknown.

ease (Fig. 3) and a small deep crescentic calcification in a second (Fig. 4). In the cases of toxoplasmosis, some had diffuse concentric and linear calcifications (Fig. 5) and one had deep circular densities (Fig. 6).

In each of the disease entities noted in Table II there was significant variation in the morphologic characteristics of calcifications in those patients with identical diagnoses. The literature supports the finding that considerable variation is frequent.[2,19]

DISCUSSION

The frequency with which calcifications appear intracranially increases with age and is unrelated to sex. Physiologic calcifications are seen in the first 10 years of life but with a frequency of less than 1 per cent. In the first 6 years of life the rate is even lower. Physiologic calcifications are considered such when they are found in certain defined areas of the brain or meninges (pineal, choroid plexus, basal ganglia[1] or dura) and are apparently unrelated to the patient's general status.

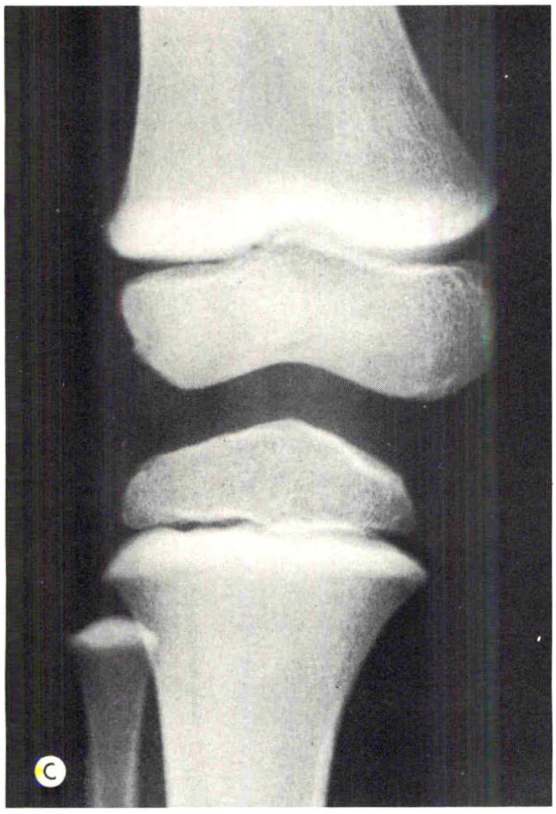


Fig. 2. (*C*) Anterior view of the patient's knee demonstrating heavy metaphyseal bands representing further evidence of heavy metal intoxication.

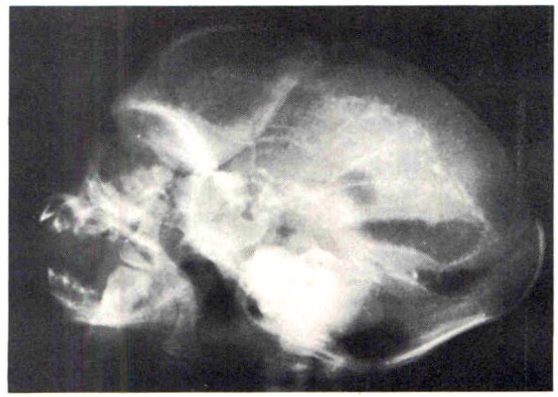


Fig. 3. Lateral view of a microcephalic patient with extensive paraventricular calcifications. The patient was a proved case of cytomegalic inclusion disease. There is residual air from previous ventricular studies.

It is unusual to find any calcifications in children being evaluated for head trauma in general hospitals. Of course, skull roentgenograms in referral institutions might be expected to have a higher yield. This would be so in neurologic institutions to which brain tumor patients are referred or

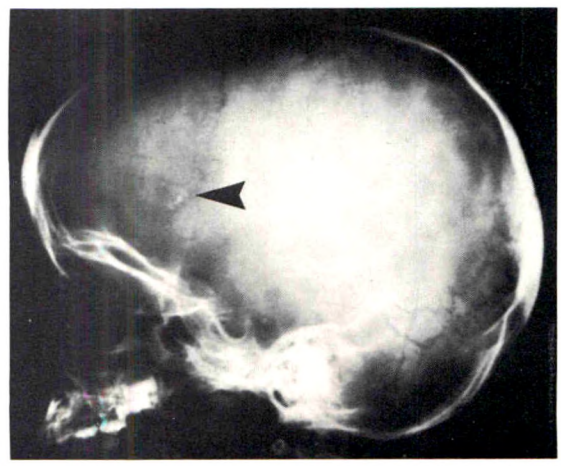


Fig. 4. Lateral roentgenogram of a patient with a normal or slightly enlarged skull and a single calcification (arrow) deep within a frontal lobe. This patient had cytomegalic inclusion disease and demonstrates manifestations different from those of the patient in Figure 3.

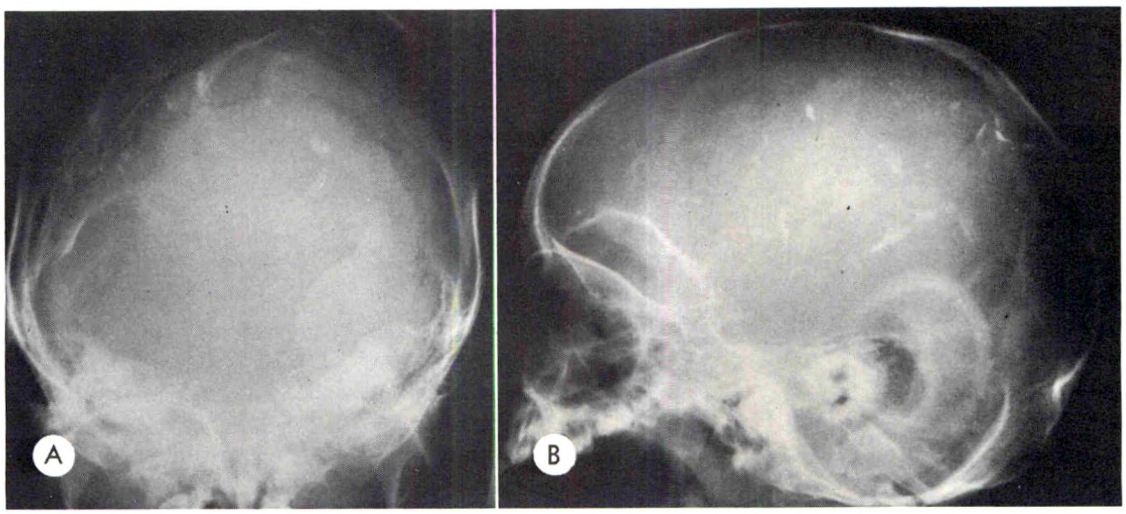


FIG. 5. (*A*) Anteroposterior and (*B*) lateral roentgenograms of a patient with toxoplasmosis. There are diffuse parenchymal and subcortical calcifications, some of which are curvilinear and others straight.

in institutions caring for children post inflammation of the central nervous system. Actually, many groups that would be expected to have high rates of calcifications do not. This includes the dysfunctioning child center patients and the reported series of seizure patients.[6]

The frequency of occurrence of pathologic calcifications is greatest in vascular abnormalities (A-V malformations,[19] Sturge-Weber and similar entities[24]), brain tumors, and as a result of cerebral inflammatory disease (cytomegalic inclusion disease, toxoplasmosis, tuberculosis, etc.[24]). Although considerably higher in these specific groups than others the frequency in any one group is still low, except for toxoplasmosis.

Numerous etiologies are responsible for

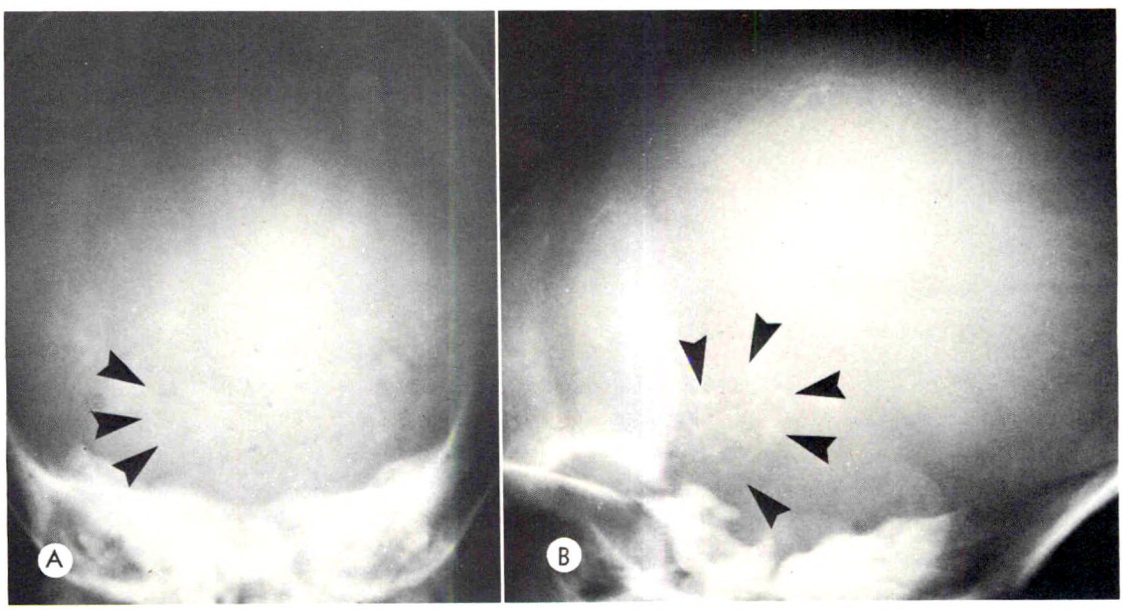


FIG. 6. (*A*) Anteroposterior and (*B*) lateral roentgenograms of the skull in a patient with toxoplasmosis. This patient has curvilinear and circular calcifications (arrows) deep within the parenchyma just above the skull base. There is a significant difference in appearance from that shown in Figure 5.

causing intracranial calcifications. We found calcifications in many instances (Tables II and III). Teaching files and personal collections could probably add a significant number of varying etiologies but most would be the result of a very unusual inflammatory disease (or infestation), tumor,[17] iatrogenic[3,13] or familial cause.[7,11,14]

Intracranial hemorrhage causing a chronic subdural hematoma or old intraventricular bleeding can account for intracranial calcifications.[24] Unusual inflammatory processes have also been reported and include listerosis and torulosis.[24] Some of the most interesting causes are reported in patients with familial and/or degenerative disease processes.[14,15,20] These patients may have "fits" or seizure disorders, ataxia, or physical and/or mental deterioration. Since calcifications in some of these patients are found in the basal ganglia, hypoparathyroidism or pseudohypoparathyroidism should be considered. The serum calcium and phosphorus levels are usually normal, however.[14] It is evident that basal ganglion calcifications can be found in normal patients, but since such occurrences are rare the finding of calcific deposits at that site should be viewed with considerable suspicion.

The morphologic characteristics of intracranial calcifications have been described by numerous authors. Some feel that calcifications in specific entities yield pathognomonic data. Most authors, including the writer, feel that the variability of the calcifications (number, size, quality, location) is so great that in most instances only a differential diagnosis should be proffered. Nevertheless, it behooves the roentgenologist to call attention to the findings and suggest a possible etiology.

In considering further medical evaluation of a patient with intracranial calcifications it probably is safe to say that further evaluation should be based heavily upon the history and clinical findings rather than on the specific finding of intracranial calcifications. Calcifications found intracranially in locations other than the usual physiologic locales will raise more questions but should not in and of themselves stimulate extensive, costly, painful and at times morbid examination.

CONCLUSIONS

1. Intracranial calcifications are infrequent in the childhood population. "Physiologic" calcifications are almost never seen under age 6 years. They are noted with increasing frequency, thereafter.

2. Calcifications in pathologic states are reported with varying rates of occurrence. Tumors, inflammatory disease and some vascular abnormalities account for the majority.

3. The morphology and location of intracranial calcifications can suggest a possible diagnosis but variations are too great to be unequivocal; a likely differential diagnosis is possible, however.

4. A clinical approach to the patient should include consideration of the findings of calcifications but that finding alone should not stimulate exhaustive studies.

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REFERENCES

1. Bennett, J. C., Maffly, R. H., and Steinbach, H. L. Significance of bilateral basal ganglia calcification. *Radiology*, 1959, *72*, 368–378.
2. Constantini, F. E., Merli, G. A., Pellone, M., and Marin, G. Sulle difficoltà di una diagnosi radiologica differenaziale in alcuni casi con calcificazioni endocraniche. *Chir. ital.*, 1960, *20*, 1817–1832.
3. Dolan, P. A. Tumor calcification following therapy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 166–174.
4. French, L. A. Brain tumors in children. *Minnesota Med.*, 1948, *31*, 817–874.
5. Grossman, H., Winchester, P. H., Deck, M.,

and Guistra, P. Brain tumors in children with normal skull roentgenograms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *112*, 329–331.

6. Hayes, W. G., and Shopfner, C. E. Plain skull roentgenographic findings in infants and children with convulsions. *A.M.A. Am. J. Dis. Child*, 1973, *126*, 785–787.
7. Kotscher, E. Familiares Aufreten von endokraniellen Verkalkungen bei Lipoproteinose. *Radiologia Austriaca*, 1960, *10*, 299–303.
8. Kraft, E., Finby, N., and Schillinger, A. Routine skull roentgenography of psychiatric hospital admissions. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 1212–1219.
9. Majd, M., Farkas, J., Lopresti, J. M., Chandra, R., Hung, W., and Lussenhop, A. J. Large calcified craniopharyngioma in newborn. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *99*, 399–400.
10. McRae, D. L. Habenular calcification as aid in diagnosis of intracranial lesions. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 541–546.
11. Melchoir, J. C., Benda, C. E., and Yakovlev, P. I. Familial idiopathic cerebral calcifications in childhood. *A.M.A. Am. J. Dis. Child*, 1960, *99*, 787–803.
12. Muraase, Y., Tanaka, S., Futamura, A., Ito, T., Imamura, K., and Sakata, K. New simple measurement of pineal calcification in lateral craniogram. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 92–95.
13. Norrell, H., Wilson, C. B., Slagel, D. E., and Clark, D. B. Leukoencephalopathy following administration of methotrexate into cerebrospinal fluid in treatment of primary brain tumors. *Cancer*, 1974, *33*, 923–932.
14. Palubinskas, A. J., and Davies, H. Calcification of basal ganglia of brain. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 806–822.
15. Palubinskas, A. J., and Davies, H. Familial basal ganglia calcification. *Radiology*, 1959, *72*, 426–429.
16. Peterson, H. O., and Kieffer, S. A. Introduction to Neuroradiology. Volume 1. Harper and Row, Hagerstown, Md., 1972, p. 10.
17. Potts, D. G., and Svare, G. T. Calcification in intracranial metastases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 1249–1251.
18. Rothstein, J. L. Calcification of choroid plexus. *Am. J. Dis. Child.*, 1936, *51*, 123–130.
19. Rumbaugh, C. L., and Potts, D. G. Skull changes associated with intracranial arteriovenous malformations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 525–534.
20. Strobos, R. R. J., De La Torre, E., and Martin, J. F. Symmetrical calcification of basal ganglia with familial ataxia and pigmentary macular degeneration. *Brain*, 1957, *80*, 313–317.
21. Sutton, D. Intracranial calcification in toxoplasmosis. *Brit. J. Radiol.*, 1951, *24*, 31–37.
22. Teplick, J. G., and Adelman, B. P. Unilateral calcification of choroid plexus in child. *Radiology*, 1956, *66*, 231–233.
23. Varadarajan, M. G., and Ramamurthi, B. Assessment of normal physiological intracranial calcification occurring in Indian subjects. *Mediscope*, 1964, *12*, 453–466.
24. Willich, E., Sellier, W., and Weigel, W. Die intrakraniellen Verkalkungen des Kindesalters. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1972, *116*, 735–750.

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INCIDENCE OF NORMAL PINEAL GLAND CALCIFICATION IN SKULL ROENTGENOGRAMS OF BLACK AND WHITE AMERICANS*

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HUMAN pineal gland calcification, which was reported in autopsy specimens about 300 years ago, was first described in the plain skull roentgenogram in 1918 by Schüller,[16] who remarked that the gland "very often shows a calcium deposit from the third decade and occasionally even in children."

Subsequent workers confirmed Schüller's observation and established the influence of age on the incidence of pineal gland calcification.[6,7,13,17,18] From a summary of several of these works, the estimated frequency of pineal shadow was found to be 2 per cent in those 3 to 12 years of age; 46 per cent from 13 to 40; and 69 per cent after the age of 40.[20] All the above studies were carried out mainly in Caucasians, and racial differences were not mentioned.

In recent years, a much lower incidence of pineal gland calcification has been reported in other races: 9.9 per cent in Japanese;[4] 15.6 per cent in Fijians;[11] and 19–24 per cent among Indians.[11,15] In Africa, one of the first hints of a low incidence of pineal shadow was made by Odeku and Janota[14] in their study of brain tumors at the University College Hospital, Ibadan, Nigeria. They mentioned a surprising rarity of calcified pineal gland on skull roentgenograms in West Africans. At the same hospital, where an average of about 2,000 skull roentgenographic examinations are made every year, Adeloye and Odeku[2] encountered less than 10 cases of roentgenologically visible calcified pineal gland in the neurosurgery unit during a period of 10 years. These impressions have been confirmed by Daramola and Olowu,[5] who, in their study of 952 skull roentgenograms obtained in Nigerians in Lagos, found a calcified pineal shadow in 5 per cent. In East Africa, Murphy,[12] a radiologist at the Mulago Hospital in Kampala, Uganda, found only 2 examples of calcified pineal gland in a series of 100 consecutive skull examinations obtained from 88 East Africans, 9 Asians, and 3 Europeans between 17 and 62 years of age. Although he did not identify the racial origin of the 2 patients, any mode of mathematical manipulation would still make the incidence of pineal gland calcification remarkably low in the East African.

Pineal gland calcification is not necessarily indicative of pineal gland atrophy, at least by biochemical and histologic evidence.[20] A recent pathologic study showed that microscopic pineal gland calcification occurs in Nigerian Africans in every decade from the first to the ninth.[2] The paradoxical rarity of gross, roentgenographically visible pineal gland calcification remains unexplained.

The purpose of the present study is to compare the incidence of the calcified pineal gland shadow between black and white populations living in a similar environment, to determine if there is truly a lower incidence of visible pineal gland calcification among blacks and if so, to seek an explanation for it.

MATERIAL AND METHOD

Five hundred consecutive normal plain skull roentgenographic examinations performed on patients in the Emergency Room of the Cincinnati General Hospital between

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February and May 1973 provided the material for this study.

There were 300 blacks and 200 whites in the series; of the blacks, 162 were males and 138 females; 127 males and 73 females comprised the white group. The age, sex, and racial distribution of the patients is shown in Table 1.

The Cincinnati General Hospital is a 691 bed teaching hospital in which all the medical specialties are represented. Patients in the humbler categories of the socioeconomic stratum of the population predominate among the patients of this hospital. Most patients in the city of Cincinnati who require emergency care are taken to the General Hospital.

Three standard views of the skull were routinely obtained: lateral; anteroposterior (Towne); and posteroanterior. In view of the frequent inability of these patients to cooperate, a number of films were less than optimal. Patients with technically inadequate films were rejected from the series. If the lateral projection was of good quality, however, the patient was included. If anything, then, the prevalence of visible pineal calcification was slightly underestimated.

The pineal shadow was recorded as visible when seen either on the lateral or one of the frontal projections. Calcification in the falx cerebri and choroid plexus was also recorded. Habenular calcification, when recognized, was tabulated separately and is not included in the pineal statistics. Skull thickness was also recorded on all the roentgenograms used in this series. These will form the basis of a separate report.[1]

FINDINGS

Of the 500 patients in the entire series, 61 (12.2 per cent) showed a calcified pineal gland (Table 1). None of the 125 patients in the first decade of life showed pineal gland calcification.

RACIAL VARIATION

There were 29 blacks and 32 whites with a calcified pineal gland shadow—a racial incidence of 9.7 and 16.0 per cent, respectively. The ratio of visible pineal shadow in white and black Americans is thus about 1.6 to 1, a difference which is statistically significant (chi square, 4.49; $p < .05$).

This racial difference is also seen if the comparisons are limited to adult age groups: 16.1 per cent of the blacks and 26.4 per cent of the whites from the third decade on.

AGE

The youngest black with a calcified pineal shadow was a boy of 16; the youngest white was a woman of 21. Among both blacks and whites, the frequency of calcified pineal gland shadow increased with age, but this was more striking among

TABLE I

AGE, SEX, AND RACIAL INCIDENCE OF PINEAL CALCIFICATION

Age Groups (yr.)	Under 10		10–19		20–29		30–39		40–49		50–59		60–69		70+	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
No. of black subjects	51	33	28	20	28	31	16	14	14	16	11	5	9	8	5	11
No. with calcified pineal gland	0	0	2	0	5	4	2	0	2	3	0	2	2	1	2	4
No. of white subjects	26	15	23	15	26	17	16	8	17	9	9	4	8	3	2	2
No. with calcified pineal gland	0	0	0	0	0	3	4	1	4	4	6	0	6	3	1	0

TABLE II

RACE AND AGE DISTRIBUTION OF CALCIFIED PINEAL GLAND

Age (yr.)	Black Subjects			White Subjects		
	No. of Subjects	Calcified Pineal Glands		No. of Subjects	Calcified Pineal Glands	
		No.	Per cent		No.	Per cent
Under 10	84	0	0	41	0	0
10–19	48	2	4.2	38	0	0
20–29	59	9	15.3	43	3	7
30–39	30	2	6.7	24	5	20.8
40–49	30	5	16.7	26	8	30.8
50–59	16	2	12.5	13	6	46.2
60–69	17	3	17.4	11	9	81.8
70+	16	6	37.5	4	1	25.0
Total	300	29	9.8	200	32	16.0

whites (Table II). Under the age of 40, a calcified pineal gland was encountered with more or less equal frequency among black and white populations; thereafter, the predominance among whites was clearly shown (Table III). It was also noted that the calcifications were generally larger and more inclined to be multiple in the older patients.

SEX

Among the blacks, the pineal shadow was visible in 15 (9.3 per cent) of the 162 males and 14 (10.1 per cent) of the 138 females. The sex ratio of the patients with calcified pineal gland did not appear to be influenced by age. Among the whites, pineal gland calcification was seen in 21 (16.5 per cent) of the 127 males and in 11 (15.1 per cent) of the 73 females. Thus, no significant sex differences in incidence of pineal gland calcification were found in either the whites or blacks. Beyond age 40 in the white group, pineal gland calcification was seen in 47 per cent of the males and 38 per cent of the females. Corresponding figures among the blacks were 15 and 25 per cent, respectively.

OTHER INTRACRANIAL CALCIFICATIONS

A striking preponderance of *calcified falx cerebri* was noted in the blacks. There were 32 examples (16 per cent), with 18 females and 14 males. In the whites, only 6 instances of this were seen (3 per cent), 3 in men and 3 in women. *Choroid plexus calcification* was found in 7 blacks (4 men and 3 women) aged 40 to 70, and in 4 white men between 24 and 60. There were 6 examples of *habenular calcification*, with equal racial distribution.

DISCUSSION

In addition to its racial aspects, this study provides an opportunity to take another look at some of the viewpoints concerning the calcified pineal shadow that have been discussed in the literature. One of these is the increase in incidence of calcification with age. Our percentage age figures shown in Table II agree fairly closely with those which Wurtman *et al.*[20] constructed from the several studies compiled earlier by Kitay and Altschule.[7] The age effect was maintained up to age 70, after which most series (including ours) were limited by the paucity of subjects. Almost all reports indicate that the pineal

TABLE III

PERCENTAGE INCIDENCE OF PINEAL SHADOW IN BLACK AND WHITE SUBJECTS

Age Groups (yr.)	Black Subjects	White Subjects
10–19	4.2	0
20–39	12.4	11.9
Over 40	20.3	44.4

shadow can be seen as early as the second decade of life. Its occurrence in the first decade is quite rare.[7,9]

Most reports on calcified pineal gland have underlined a male preponderance. Vastine and Kinney[18] found that in every decade from the second on, males predominated, with an over-all incidence of 58 per cent. Dyke[6] reported a male incidence of 54 per cent. A higher male incidence was also found among the Japanese[4] and Indians,[11,15] whereas among Fijians[11] and Nigerians in Lagos,[5] slightly more females were found to have calcified pineal glands. The sex distribution was about even in the present study.

The incidence of pineal gland calcification in our series is not as high as that reported in the literature. Our over-all incidence was 12.2 per cent. Over the age of 20 we found an incidence of 20.4 per cent. In studies reported before 1940, the incidence varied between 33 and 76 per cent among subjects of all ages;[7] in persons over 20, the incidence was found to be about 60 per cent.[6,10,13,17,18] Wakeley[19] predicted that this percentage would rise even higher with improved techniques in radiology. The high figures recorded in these early studies might be explained in part by inclusion of habenular calcification, but the contribution of this factor must have been small, judging by the fact that we found only 6 examples among 500 subjects. The inconsistency is more likely the result of differences in age range. Many of our patients were under 20, an age group in which the incidence of pineal gland calcification is known to be low. Thus, two-thirds were in the first 3 decades as compared with one-third in the series of Vastine and Kinney[18] and of Dyke;[6] 25 per cent of our patients were below the age of 10, in striking contrast to 1.5 per cent in the series of Vastine and Kinney and 6.5 per cent in the series of Dyke. However, decade by decade, our figures are clearly lower than theirs.

The most interesting aspect of this study is the significant difference in the frequency of pineal gland calcification between black and white Americans. This observation has not been made before. The subjects in our study were all Americans who live under similar environmental conditions and who belong to more or less comparable socioeconomic groups. Thus, environmental factors are probably not as important as constitutional ones in explaining the differences noted. We have not explored the biochemical factors which might control calcification in our patients (*e.g.*, serum calcium, phosphorus, and protein). In a few patients so examined in their Nigerian series, Daramola and Olowu[5] found that these values were normal.

An intriguing possibility has been raised that might relate to the racial differences in pineal gland calcification. Melatonin, the most potent skin lightening agent presently known, is elaborated only by the pineal gland.[8] Increase in daylight inhibits melatonin synthesis, while a decrease of light has the opposite effect.[3,5] Could increase in this hormone in the pineal gland be related to calcium formation in that organ as well as to the whiteness of the human skin?

When our figures are compared with those obtained among Africans living in Uganda[12] and in Lagos, Nigeria,[5] it is seen that the incidence of pineal gland calcification is higher in the American black than in the indigenous African. Since the American black and the African indigen are ethnologically related, it seems reasonable to look toward environmental factors to explain the difference in the incidence of calcified pineal shadow between them. Perhaps the better nutritional status of the American black accounts in some way for the discrepancy. Another possibility is the difference in age distribution between the 2 groups. In Nigeria today, as in most African countries, a considerable proportion of hospital patients are children and young people; older patients habitually shun the hospitals. Since age groups are not mentioned in Murphy's East African series,[12] this might weight his figures, but could hardly account for the 2 per cent level. On the other hand, age by decades

was recorded in the Nigerian series of Daramola and Olowu,[5] and their age groupings were similar to our own.

It thus appears that possibly the most important factor of all that might explain these differences is neither environmental nor socioeconomic. One wonders how black the American black of today really is. Racial admixture has probably created constitutional differences between the American black and his African cousin, of which a discrepancy in the incidence of pineal gland calcification is perhaps an expression.

SUMMARY

The incidence of roentgenologically visible pineal gland calcification is approximately twice as common in American whites as in blacks, a difference that is very striking after age 40. Comparison of this finding with reports in the literature shows that the incidence of pineal gland calcification is slightly higher in American blacks than in indigenous Africans, probably due to racial mixture among the American blacks we studied.

It appears that the low incidence of calcified pineal shadow already observed in the African has a constitutional basis.

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REFERENCES

1. Adeloye, A., and Kattan, K. R. Thickness of normal skull in American blacks and whites. To be published.
2. Adeloye, A., and Odeku, E. L. Preliminary study of pineal gland in Nigerian African. *Rev. neuro-psiquiat.*, 1972, *1*, 54–77.
3. Axelrod, J., Wurtman, R. J., and Winget, C. M. Melatonin synthesis in hen pineal gland and its control by light. *Nature*, 1964, *201*, 1134.
4. Chiba, M., and Yamada, M. About calcification of pineal gland in Japanese. *Folia psychiat. neurol. Jap.*, 1948, *2*, 301–303.
5. Daramola, G. F., and Olowu, A. O. Physiological and radiological implications of low incidence of pineal calcification in Nigeria. *Neuroendocrinology*, 1972, *9*, 41–57.
6. Dyke, C. G. Indirect signs of brain tumor as noted in routine roentgen examinations: displacement of pineal shadow: survey of 3,000 consecutive skull examinations. Am. J. Roentgenol. & Rad. Therapy, 1930, *23*, 598–606.
7. Kitay, J. I., and Altschule, M. D. The Pineal Gland: A Review of the Physiological Literature. Commonwealth Fund, Cambridge, 1954, pp. 77–78.
8. Lerner, A. B., Case, J. D., and Takahashi, C. Y. Isolation of melatonin and 5-methoxyindole 3-acetic acid from bovine pineal glands. *J. Biol. Chem.*, 1960, *235*, 1992–1997.
9. Lorenz, R. Zur Lagebestimmung der verkalkten Glandula pinealis im Röntgenbild. *Fortschr. a.d. Geb. d. Röntgenstrahlen*, 1940, *61*, 338–348.
10. Mascherpa, F., and Valentino, V. Intracranial Calcification. Charles C Thomas, Publisher, Springfield, Ill., 1959, pp. 3–27.
11. McKay, R. T. Pineal calcification in Indians and Fijians. *Tr. Roy. Soc. Trop. Med. & Hyg.*, 1973, *67*, 214–216.
12. Murphy, N. B. Carotid cerebral angiography in Uganda: review of 100 consecutive cases. *East African M. J.*, 1968, *45*, 47–60.
13. Naffziger, H. C. Method for localization of brain tumors: pineal shift. *Surg., Gynec. & Obst.*, 1925, *40*, 481–484.
14. Odeku, E. I., and Janota, I. Intracranial masses —Ibadan. *West African M. J.*, 1967, *16*, 31–42.
15. Pillai, K. M. Pineal gland calcification. *Neurol., India*, 1968, *16*, 35–36.
16. Schüller, A. Roentgen Diagnosis of Diseases of the Head. C. V. Mosby Company, St. Louis, 1918, p. 156.
17. Sosman, M. C. Radiology as aid in diagnosis of skull and intracranial lesion. *Radiology*, 1927, *9*, 396–407.
18. Vastine, J. H., and Kinney, K. K., Pineal shadow as aid in localization of brain tumors. Am. J. Roentgenol. & Rad. Therapy, 1927, *17*, 320–324.
19. Wakeley, C. P. G. Surgery of pineal organ. *Brit. J. Surg.*, 1938, *25*, 561–591.
20. Wurtman, R. J., Axelrod, J., and Barchas, J. D. Age and enzyme activity in human pineal. Letter to the editor. *J. Clin. Endocrinol. & Metabol.*, 1964, *24*, 299–301.

CO-EXISTING PITUITARY ADENOMA AND INTRASELLAR ARACHNOID INVAGINATION*

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ALTHOUGH there is an increasing awareness in the literature of the existence of intrasellar arachnoid invagination ("empty" sella), no report has appeared concerning its specific relationship to intrasellar tumors.

We propose to contribute our experience of pituitary tumors associated with an empty sella and present the import of this finding.

MATERIAL AND METHOD

In the last 5 years we have observed 3 patients who presented sellar modifications, and clinical or endocrinologic findings indicative of a pituitary tumor. None of these patients had previous surgical or radiation treatment. At pneumoencephalography, including lateral tomography, an unexpected subarachnoid invagination into the sella turcica was demonstrated (Fig. 1; 2, *A* and *B*; and 3).

Their clinical features, namely an acromegalic syndrome in 2 patients and an amenorrhea-galactorrhea syndrome in 1 (Table 1), still forced us to consider the surprising co-existence of a pituitary tumor with the "empty" sella. For this reason, trans-sphenoidal surgical exploration of the sella was performed and in all cases this co-existence was confirmed.

DISCUSSION

Numerous reports of "empty" sella have appeared since the initial pathologic description by Busch in 1951,[2] and the subsequent radiologic demonstration.[11]

Nearly all articles give accounts of an "empty" sella occurring possibly either after pituitary surgery or radiotherapy.[3,6,8,10] In these cases the surgically created vacuum or the necrotic shrinking of the tumor induced by radiotherapy allow subarachnoid invagination into the sella turcica through a patent diaphragma sellae.

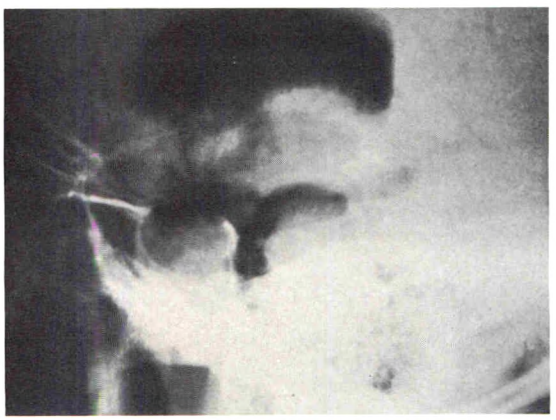


FIG. 1. Pneumoencephalogram, brow-up lateral projection, of an untreated acromegalic patient showing enlargement of the sella and co-existence of a pituitary adenoma and an intrasellar subarachnoid invagination.

On the other hand, the co-existence of this condition with untreated pituitary adenomas has not been emphasized. Only 5 patients with untreated pituitary adenoma and associated "empty" sella have been reported.[7,9,12] Four of these cases were demonstrated roentgenologically, the fifth being proven at autopsy.

We can offer no definite explanation for this association in untreated cases. It may well be, as Kaufman and Chamberlain[7] suggest, that the frequency of "empty" sellae is great enough in the adult population so they can be found in association or concomitantly with many disease states. An equally valid explanation may be the shrinkage of the tumor following spontaneous apoplexy,[1] with subsequent aspiration

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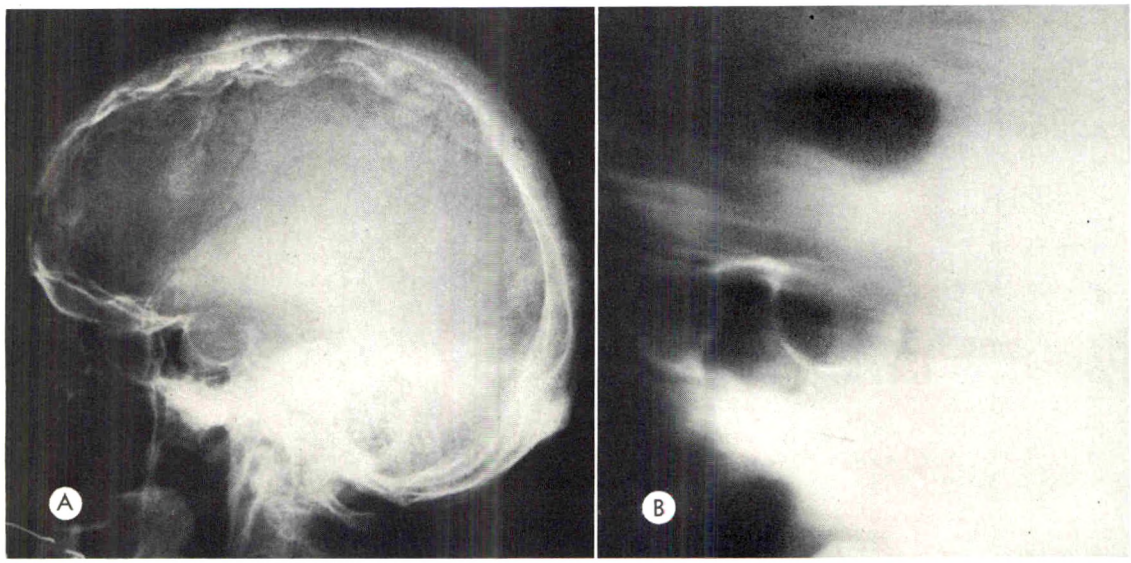


FIG. 2. (*A*) Lateral roentgenogram of the skull showing enlargement of the sella, marked thickening of the cranial vault, prominent external occipital protuberance and hypertrophy of the paranasal sinuses. (*B*) Lateral pneumoencephalotomogram, brow-up projection, revealing large arachnoidocele coexisting with shrunken pituitary gland.

of the subarachnoid space into the sella through an incompetent diaphragm. This apoplexy need not necessarily compromise the hormonal function of a secreting pituitary adenoma as is shown in 2 of our patients. Therefore, in the presence of endocrinologic abnormalities, the finding of an empty sella during pneumoencephalography in an untreated patient must not lead one to invariably exclude an underlying pituitary adenoma.

CONCLUSION

If an untreated patient presents a clinical syndrome suggesting a secreting pituitary tumor, the finding of an "empty" sella at pneumoencephalography must not lead one to conclude to the invariable absence of

TABLE I

SUMMARY OF 3 CASES

Case No.	Clinical Findings	Surgical Findings
I A.L.—M Age 45 yr. *Fig. 1*	Active acromegaly for more than 4 years. Serum prolactin (PRL): 31 ng./ml. No previous radiotherapy or surgery	Large pituitary adenoma, co-existing with a sizeable subarachnoid invagination. Selective total removal of the adenoma. Histology: typical acidophilic cells
II A.R.—F Age 26 yr. *Fig. 2, A and B*	Rhinorrhea. Arrested acromegaly—characteristic facies, large hands. No previous radiotherapy or surgery	Large arachnoid invagination occupying nearly entirely the sellar cavity and eroding its floor with rupture into the sphenoid sinus
III D.C.—F Age 26 yr. *Fig. 3*	Amenorrhea and galactorrhea for 6 years. One previous pregnancy. Elevated serum prolactin (PRL: 105 ng./ml.)	5 mm. pituitary cystic adenoma, selectively and totally removed with preservation of the normal hypophyseal tissue



FIG. 3. Pneumoencephalotomogram, lateral brow-up projection. Air-filled chiasmatic cistern, outlining the optic chiasm and showing intrasellar invagination of the subarachnoid space, ballooning of the sella, undercutting of the tuberculum, and thinned cortex.

an associated pituitary adenoma, since the subarachnoid invagination is not responsible for the endocrinologic manifestations.

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REFERENCES

1. BROUGHAM, M., HEUSNER, A. P., and ADAMS, R. D. Acute degenerative changes in adenomas of pituitary body: with special reference to pituitary apoplexy. *J. Neurosurg.*, 1960, *7*, 421–439.
2. BUSCH, W. Die Morphologie der Sella Turcica und ihre Beziehungen zur Hypophyse. *Arch. path. Anat.*, 1951, *320*, 437–458.
3. CAPLAN, R. H., and DOBBEN, G. D. Endocrine studies in patients with empty sella syndrome. *A.M.A. Arch. Int. Med.*, 1969, *123*, 611–619.
4. EL GAMMAL, T., and ALLEN, M. B., JR. Intrasellar subarachnoid recess: some clinical and radiologic observations. *Acta radiol.* (Diag.), 1972, *13*, 401–412.
5. HODGSON, S. F., RANDALL, R. V., HOLMAN, C. B., and MACCARTY, C. S. Empty sella syndrome. *Med. Clin. North America*, 1972, *56*, 897–907.
6. KAUFMAN, B. Empty sella turcica—manifestations of intrasellar subarachnoid space. *Radiology*, 1968, *90*, 931–941.
7. KAUFMAN, B., and CHAMBERLAIN, W. B., JR. Ubiquitous empty sella turcica. *Acta radiol.* (Diag.), 1972, *13*, 413–425.
8. MORTARA, R., and NORRELL, H. Consequence of deficient sellar diaphragm. *J. Neurosurg.*, 1970, *32*, 565–573.
9. NEELON, F. A., GOREE, J. A., and LEBOVITZ, H. E. Primary empty sella: clinical and radiographic characteristics and endocrine function. *Medicine*, 1973, *52*, 73–92.
10. OBRADOR, S. Empty sella and some related syndromes. *J. Neurosurg.*, 1972, *36*, 162–168.
11. ROBERTSON, E. G. Pneumoencephalography. Charles C Thomas, Publisher, Springfield, Ill., 1957, pp. 230–231.
12. ZATZ, L. M., JANON, E. A., and NEWTON, T. H. Enlarged sella and intrasellar cistern. *Radiology*, 1969, *93*, 1085–1091.

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ANGIOGRAPHIC DIAGNOSIS OF A GIANT THIRD VENTRICLE*

INVERSION OF THE VENOUS ANGLE

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ALTERATIONS in the configuration of the striothalamic-internal cerebral vein junction, the venous angle of Krayenbühl, are valuable in the localization of intracranial mass lesions. The significance of displacement, opening, and closing of this region is well known.[4,9] An inverted venous angle is an unusual angiographic finding and has received little attention in the roentgenologic literature.[5,10]

The purpose of this paper is to describe this angiographic abnormality and to discuss its pathologic significance.

REPORT OF CASES

CASE I. *Agenesis of the corpus callosum* (Fig. 1, *A–D*). A 5 year old male presented with headache and lethargy. A right carotid arteriogram demonstrated a mass of the right cerebral hemisphere. The pericallosal artery and the internal cerebral vein were at the same level and the normal curve of the pericallosal artery above the genu of the corpus callosum was absent (Fig. 1, *A* and *B*). The striothalamic vein entered the internal cerebral vein from below forming an inverted venous angle (Fig. 1*B*). Pneumoencephalography (Fig. 1, *C* and *D*) revealed separation of the lateral ventricles and a large third ventricle which extended dorsally between the lateral ventricles. There was also a cystic hemispheric mass which communicated with the right lateral ventricle. Agenesis of the corpus callosum and a porencephalic cyst were confirmed at surgery.

CASE II. *Cystic dilatation of the third ventricle associated with benign aqueductal stenosis* (Fig. 2, *A–E*). A 10 year old male presented with a history of increasing seizures, mental retardation, headache and mild gait disturbances. A left carotid arteriogram revealed a hydrocephalic sweep of the pericallosal artery (Fig. 2*A*). The internal cerebral vein was flattened and the striothalamic vein formed an inverted venous angle (Fig. 2*B*). The internal cerebral vein was not as inferiorly displaced as might be expected with marked hydrocephalus. In the frontal projection (Fig. 2*C*), the striothalamic vein reflected ventricular dilatation. The medial half of this vein coursed upward to enter the internal cerebral vein. Ventriculography (Fig. 2, *D* and *E*) demonstrated a giant third ventricle which extended cephalad with its roof above the foramen of Monro. Subsequent roentgenograms revealed benign aqueductal stenosis.

CASE III. *Cholesteatoma of the third ventricle* (Fig. 3, *A–D*). A 40 year old male was evaluated for several years of deteriorating mental function. A right carotid arteriogram demonstrated hydrocephalus (Fig. 3*A*). The internal cerebral vein was bowed upward and the striothalamic vein entered this vessel from below, forming an inverted venous angle (Fig. 3*B*).

Ventriculography revealed a large cholesteatoma of the third ventricle. The roof of this structure was markedly elevated above the level of the foramen of Monro (Fig. 3*D*). The classic "cauliflower" appearance was present with irregular margins and deep fissures (Fig. 3, *C* and *D*). The diagnosis was confirmed at surgery.

DISCUSSION

The venous angle is formed at the junction of the internal cerebral vein and the striothalamic vein. Unless there is an anomalous deep venous system, this angle demonstrates the location of the foramen of Monro. The internal cerebral vein runs posteriorly from the foramen of Monro in the tela choroidea of the third ventricle. The striothalamic vein extends anteriorly

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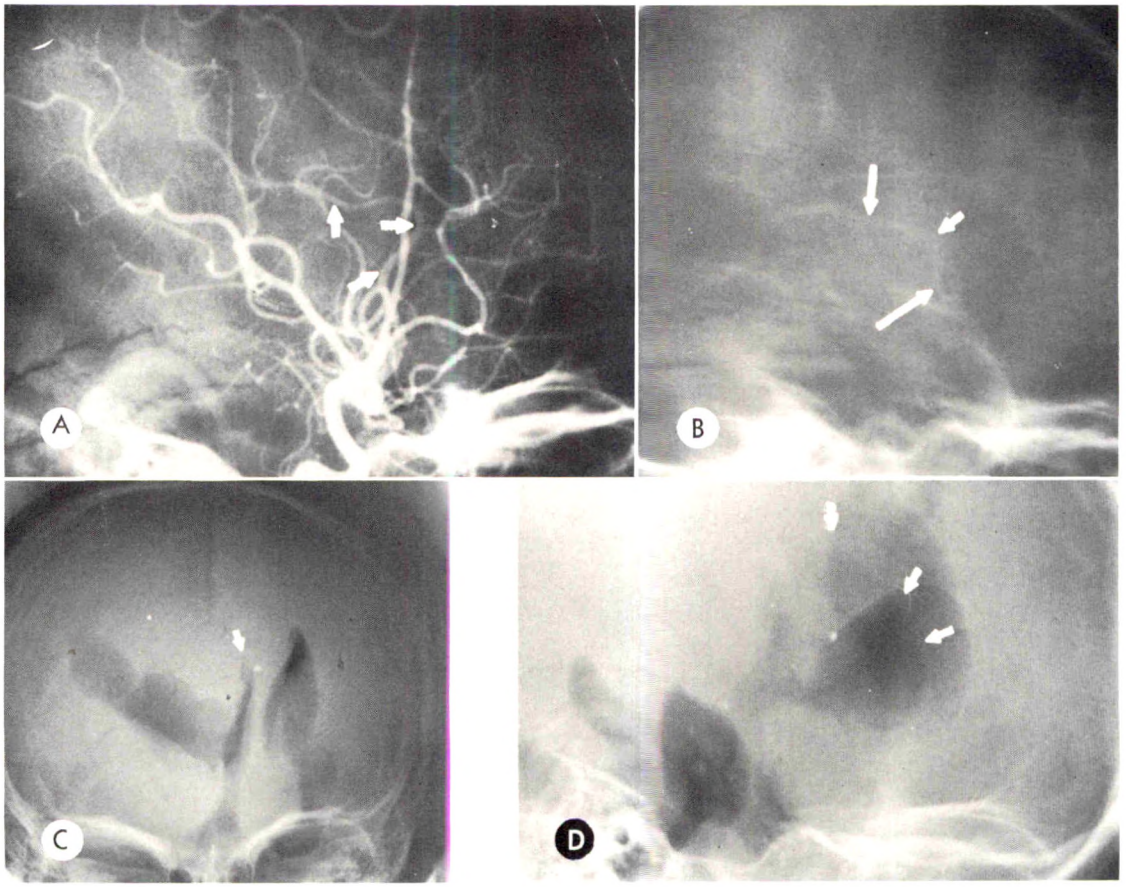


FIG. 1. Case 1. *Agenesis of the corpus callosum with porencephalic cyst.*

(*A*) Carotid angiogram. The pericallosal artery lacks the normal curve around the genu of the corpus callosum (arrows). (*B*) Venous phase, lateral angioautotomogram. The striothalamic vein (long arrow) enters the internal cerebral vein (medium arrow) from below, forming an inverted venous angle (short arrow). (*C*) Pneumoencephalogram, anteroposterior projection. The third ventricle (arrow) extends dorsally between the separated lateral ventricles. There is a midline shift produced by a porencephalic cyst which communicates with the right lateral ventricle. (*D*) Pneumoencephalogram, lateral projection. The third ventricle is enlarged and its roof (arrows) is above the level of the foramen of Monro.

in the groove between the thalamus and the caudate nucleus. It crosses the floor of the lateral ventricle, passes through the interventricular foramen, and enters the internal cerebral vein.[3] This classic description is illustrated in Figure 4*A*.

A review of the roentgenologic literature reveals little on the significance of an inverted venous angle. Wolf *et. al.*[10] described "reversal" of the relationship between the striothalamic vein and the internal cerebral vein in a third ventricular tumor of unspecified type. Larsen[5] illustrated this abnormality in agenesis of the corpus callosum. He stated that this could only occur "with separation of the lateral ventricles as seen with agenesis of the corpus callosum."

Agenesis of the corpus callosum may vary from a small defect to complete absence of the corpus callosum and related midline structure.[7] This is reflected anatomically, and at pneumoencephalography, as separation of the lateral ventricles with extension of the third ventricle upwards. There may also be angular dorsal margins

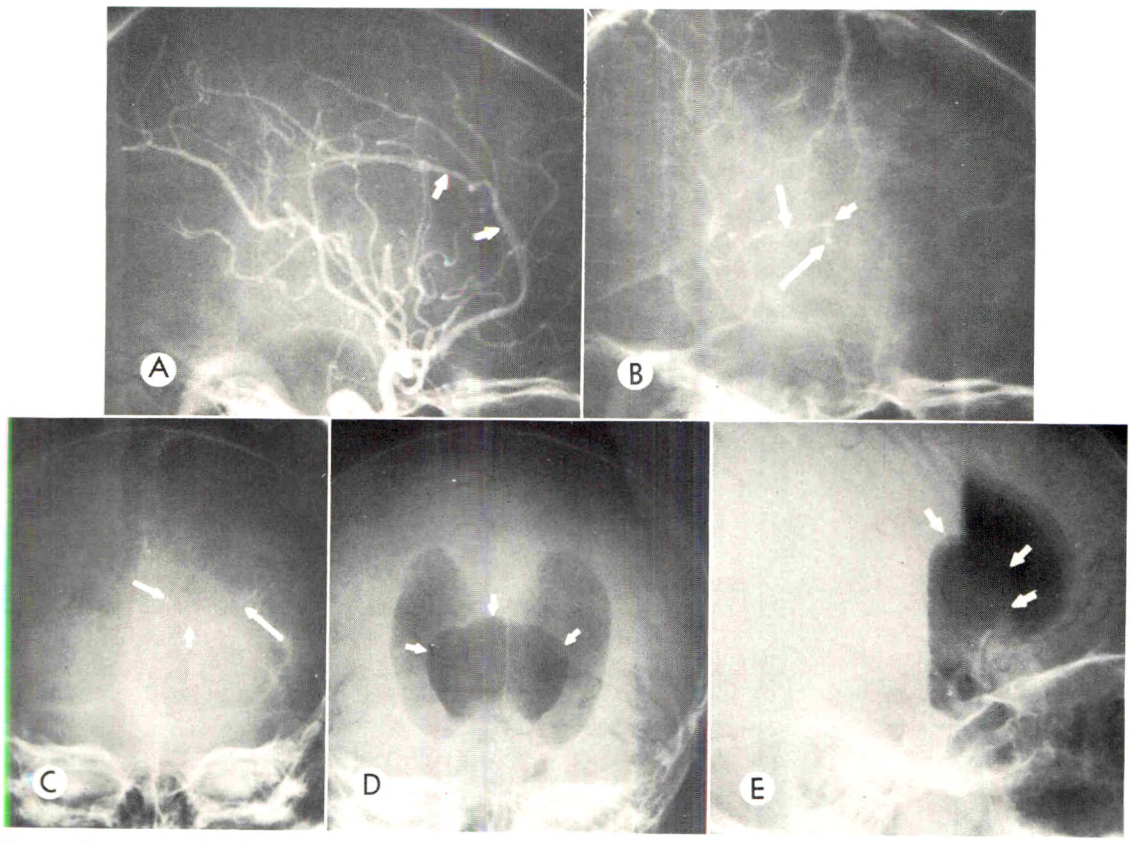


Fig. 2. Case II. *Cystic dilatation of the third ventricle associated with benign aqueductal stenosis.*

(*A*) Carotid angiogram. There is a hydrocephalic sweep of the pericallosal artery (arrows). (*B*) Venous phase, lateral angioautotomogram. The striothalamic vein (long arrow) enters the internal cerebral vein (medium arrow) from below, forming an inverted venous angle (short arrow). (*C*) Venous phase, frontal projection. The striothalamic vein (long arrow) reflects hydrocephalus. Medially, it courses upward (short arrow) to enter the internal cerebral vein (medium arrow) from below. (*D*) Ventriculogram, frontal projection. There is a giant third ventricle (arrows) extending between the dilated lateral ventricles. (*E*) Ventriculogram, lateral projection. The roof of the giant third ventricle (arrows) extends above the level of the foramen of Monro.

of the lateral ventricle and concave medial walls.[1]

The angiographic features of agenesis of the corpus callosum have been discussed by numerous authors.[2,5,6,8,11] The most important diagnostic change is the reduced distance between the pericallosal artery and the internal cerebral vein. This reflects the absence of the corpus callosum. There may be an inverted venous angle, and the pericallosal artery may lose its normal curve around the genu of the corpus callosum. These changes are present in Case I. The inverted venous angle is formed because the roof of the third ventricle is above the foramen of Monro, and the striothalamic vein must course upward to join the internal cerebral vein (Fig. 4*B*).

A similar situation is present in the other cases presented here. In both instances, the third ventricle is enlarged, and its roof is above the level of the foramen of Monro (Fig. 4*C*). Both pathologic processes are congenital and have resulted in a defect of the midline structures which allows elevation of the roof of the third ventricle.

Tumors of the third ventricle usually obstruct the ventricular system before they

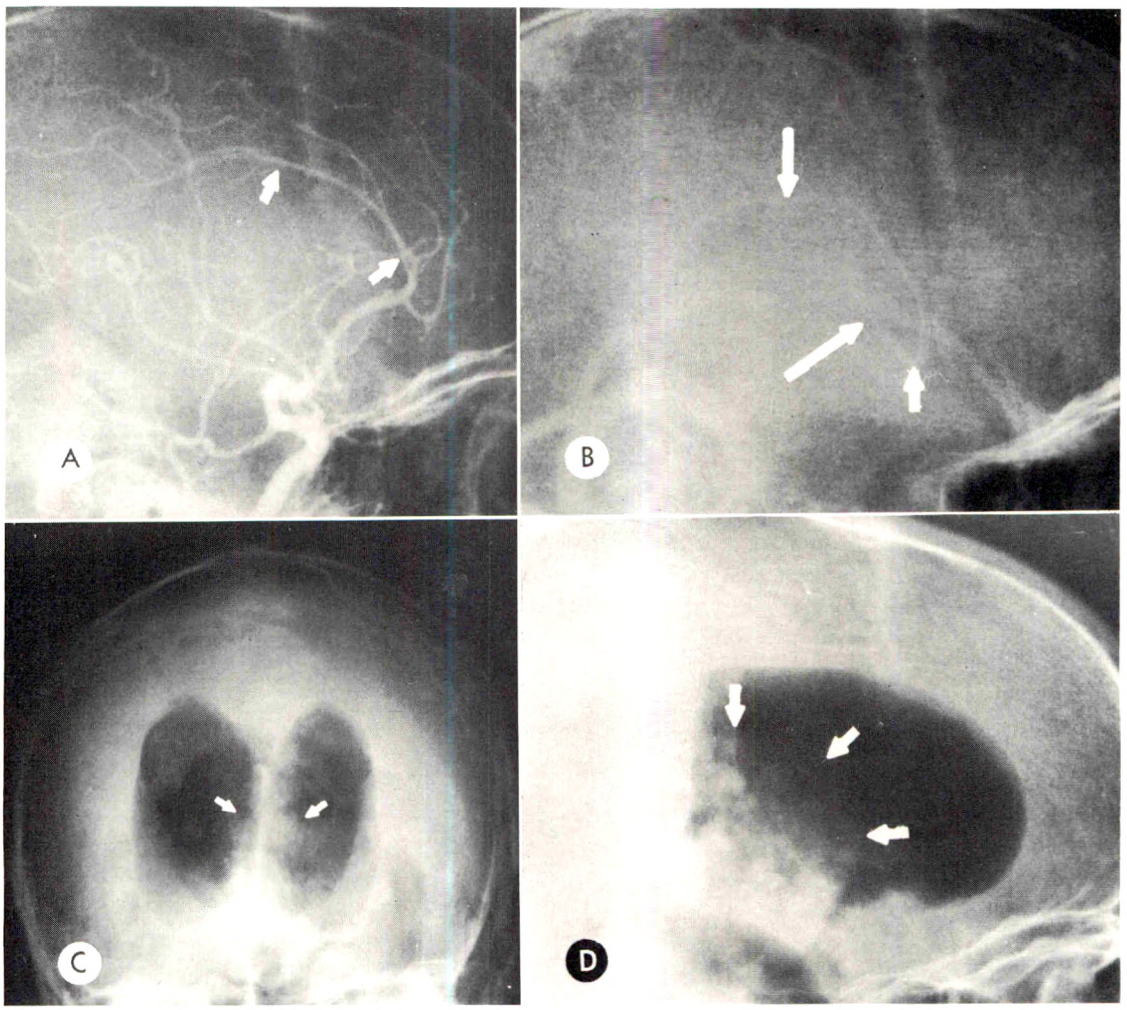


FIG. 3. Case III. *Cholesteatoma of the third ventricle.*

(*A*) Carotid angiogram. There is a hydrocephalic sweep of the pericallosal artery (arrows). (*B*) Venous phase, lateral projection. The internal cerebral vein is bowed upwards (upper arrow). The venous angle is reversed (short arrow) as the striothalamic vein (long arrow) enters from below. (*C*) Ventriculogram, frontal projection. The lateral ventricles are dilated. A cauliflower like mass fills the third ventricle (arrows) and extends into the lateral ventricle. (*D*) Ventriculogram, lateral projection. The enlarged third ventricle is filled with tumor and its roof (arrows) is elevated well above the level of the foramen of Monro.

are large enough to elevate the roof. A cholesteatoma can grow to a considerable size without complete obstruction, due to its irregular surface and deep fissures which allow escape of cerebrospinal fluid.

A review of our experience with colloid cyst of the third ventricle did not reveal an inverted venous angle. The largest cyst measured $4\frac{1}{2}$ cm. in diameter, but did not elevate the roof of the third ventricle because it protruded through the foramen of Monro. However, due to the chronic intermittent obstruction of the ventricular system, its developmental origin and the occasional large dimensions of a colloid cyst, an inverted venous angle could conceivably be produced by this lesion.

Suprasellar tumors are remote from the venous angle, and if they produce a change on this structure, it is usually displacement

of the entire complex. Hydrocephalus produces flattening of the internal cerebral vein, while the striothalamic vein is stretched superiorly and laterally.

If an inverted venous angle is discovered at cerebral angiography, the abnormality is almost certainly congenital. Only a developmental process can produce the defect in the midline structures that allows significant elevation of the third ventricle. A giant third ventricle is present which may be on the basis of agenesis of the corpus callosum, a congenital neoplasm, or some other developmental abnormality.

If the internal cerebral vein and the pericallosal artery are at the same level in the lateral projection, and the anterior cerebral artery does not show a normal curve around the genu of the corpus callosum, the diagnosis of agenesis of the corpus callosum is certain.

If there is a hydrocephalic curve of the pericallosal artery, or there is some distance between the pericallosal artery and the internal vein, ventriculography will be required to determine the etiology of the giant third ventricle. A congenital third ventricle tumor or cystic dilatation of the third ventricle will almost certainly be present.

CONCLUSION

The angiographic sign of an inverted venous angle indicates the presence of a congenital abnormality involving the third ventricle. This is commonly seen in agenesis of the corpus callosum.

An associated hydrocephalic sweep of the pericallosal artery indicates the presence of a primary congenital lesion of the third ventricle.

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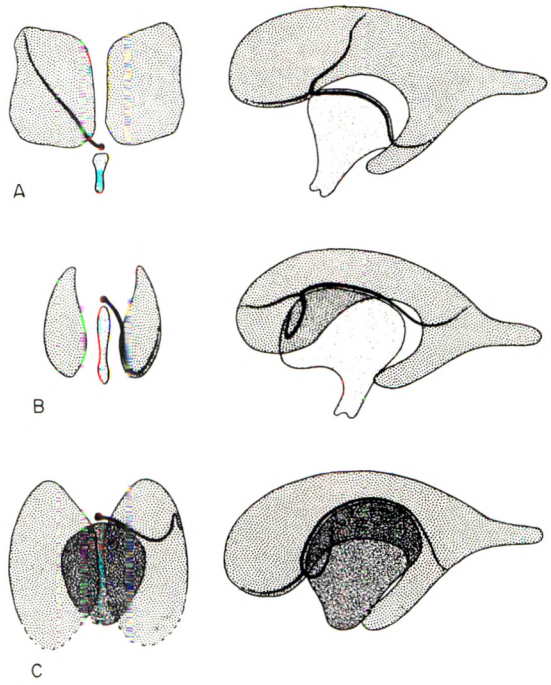


Fig. 4. *Drawings to illustrate the relationship of the striothalamic-internal cerebral veins to the ventricular system.* The lateral ventricles are shaded with small dots and the third ventricle is in fine stipple. (*A*) Normal. The striothalamic vein enters the internal cerebral vein from above forming a normal venous angle. The internal cerebral vein courses posteriorly in the roof of the third ventricle. (*B*) Agenesis of the corpus callosum. The lateral ventricles are separated and the large third ventricle extends upwards. The internal cerebral vein is elevated above the foramen of Monro and the striothalamic vein courses upward to form an inverted venous angle. (*C*) Giant third ventricle. This could be on the basis of cystic dilatation of the third ventricle (Case II) or obstruction due to a cholesteatoma (Case III). The third ventricle is enlarged and extends upwards, elevating the internal cerebral vein. The striothalamic vein enters from below forming an inverted venous angle.

REFERENCES

1. Davidoff, L. M., and Dyke, C. G. Agenesis of corpus callosum: its diagnosis by encephalography. Am. J. Roentgenol. & Rad. Therapy, 1934, *32*, 1–10.
2. Holman, C. B., and MacCarty, C. S. Cerebral angiography in agenesis of corpus callosum. *Radiology*, 1959, *72*, 317–321.
3. Johanson, C. Central veins and deep dural sinuses of brain. *Acta radiol.*, 1954, Suppl. 107, 38–52.
4. Krayenbühl, H. A., and Yašargil, M. G. Cerebral Angiography. J. B. Lippincott Com-

pany, Philadelphia, 1968, pp. 154–156, 242–247.

5. LARSEN, J. L. Angiographic findings in agenesis of corpus callosum. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 579–582.
6. NOBLER, M. P., SHAPIRO, J. H., and FINE, I. M. Cerebral angiogram in agenesis of corpus callosum. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *90*, 522–527.
7. PROBST, F. P. Congenital defects of corpus callosum. *Acta. radiol.*, 1973, Suppl. 331, 1–48.
8. SHELDON, P. W. E., and PEYMAN, A. Radiological appearances of agenesis of corpus callosum. *J. Neurol., Neurosurg. & Psychiat.*, 1953, *16*, 117–123.
9. TAVERAS, J. M., and WOOD, E. H. Diagnostic Neuroradiology. Williams & Wilkins Company, Baltimore, 1964, I. 541, I. 600, I. 624, I. 627, I. 639–I. 643.
10. WOLF, B. S., NEWMAN, C. M., and SCHLESINGER, B. Diagnostic value of deep cerebral veins in cerebral angiography. *Radiology*, 1955, *64*, 161–177.
11. ZELLWEGER, H. Agenesis corporis callosi. *Helvet. paediat. acta.*, 1952, *7*, 136–155.

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DOUBLE MIDLINE INTRACRANIAL ATYPICAL TERATOMAS*

A RECOGNIZABLE NEUROENDOCRINOLOGIC SYNDROME

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THE simultaneous occurrence of 2 midline intracranial atypical teratomas and their attending neuroendocrinologic syndrome is not widely appreciated. However, the typical clinicoroentgenographic findings virtually assure the diagnosis, and indeed, allow treatment without surgical exploration or histologic confirmation of the tumor.

Four patients with remarkably similar clinical and roentgenographic findings, and a review of the literature are presented in this report.

REPORT OF CASES

CASE 1. R.D. A 13 year old male noticed increased thirst for 2 months and nausea and vomiting for 2 weeks prior to his admission to our hospital in September, 1969. He also had nocturia (3 to 4 times a night) and had noticed that his urine was pale. Physical examination, including neurologic and ophthalmologic examinations, were negative. Laboratory studies revealed neurogenic diabetes insipidus and panhypopituitarism.

An electroencephalogram (EEG) and brain scan were normal, but plain skull roentgenograms showed a small, midline calcification in the area of the pineal gland (Fig. 1). A positive contrast ventriculogram revealed a mass deforming the anterior third ventricle (Fig. 2). The appearance and location of this lesion led to a presumptive diagnosis of craniopharyngioma. A smaller mass surrounding the pineal calcification was also noted (Fig. 2), but the possibility that it represented a second lesion was not universally entertained. Consequently an anterior craniotomy was performed and as much as possible of the anterior lesion was removed. It was encapsulated and located posterior and inferior to the optic chiasm and below the hypothalamus. Histologic diagnosis was craniopharyngioma.

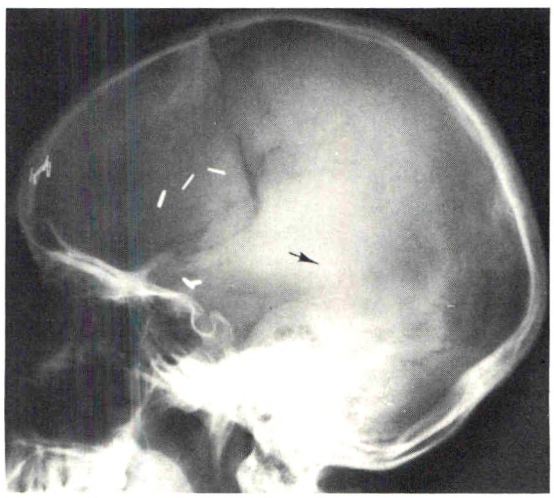


FIG. 1. Case 1. Note pineal calcification (arrow) on this postoperative roentgenogram which demonstrated the calcification to better advantage than did the preoperative roentgenogram. In frontal view the calcification was in the midline.

Following surgery the patient was maintained on pitressin and other replacement therapy for his panhypopituitarism. One year after surgery, however, he developed blurred vision, and physical examination revealed paralysis of upward gaze (Parinaud's syndrome), pupils nonreactive to light, and a right upper outer quadrant field defect. A brain scan showed an area of increased tracer activity in the region of the pineal gland, and a ventriculogram demonstrated a large mass in this area (Fig. 3). The third ventricle was grossly deformed and the aqueduct obstructed. A presumptive diagnosis of pinealoma was made and the tumor was treated with radiation therapy (5,000 r). After treatment the patient improved and his visual findings returned to normal.

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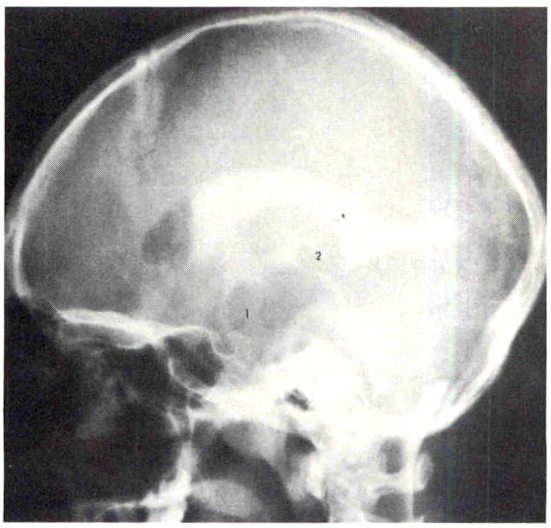


FIG. 2. Case 1. Positive contrast ventriculogram demonstrates the larger anterior tumor (1) and the small, almost inconspicuous, posterior tumor (2). Both tumors protrude into the third ventricle. Note that the anterior lesion is suprasellar, but also somewhat posterior in position.

The patient did well for approximately another year but then returned with weakness and paresthesias of his lower extremities. Eventually, this progressed to involve his trunk and upper extremities. A lumbar puncture demonstrated a cerebrospinal fluid protein content of 206 gm. per cent, and cytologic examination of the cerebrospinal fluid showed neoplastic cells. These were judged to be of epithelial origin, and because of this, tissue from the original anterior third ventricular lesion was re-examined. The tumor was then reclassified as "atypical teratoma" and the abnormal cells in the spinal fluid were believed to be seedings from it.

The patient expired within a few days, but no postmortem examination was permitted.

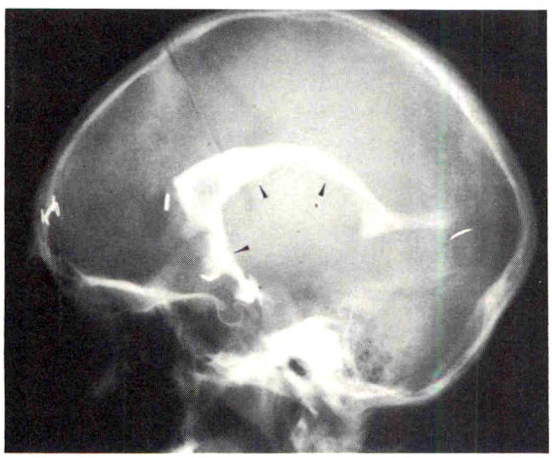


FIG. 3. Case 1. Note how large the posterior lesion has become 1 year after initial treatment and how it completely deforms the posterior third ventricle and lateral ventricles. The aqueduct is also deformed and obstructed and hydrocephalus is present. Note that the anterior lesion, although still present, is smaller.

CASE II. J.V. A 12 year old male had a 2 year history of increased water consumption and nocturia (3 to 4 times a night), and a 3 month history of lethargy, ataxia, blurred vision, and headache. Physical examination revealed loss of upward gaze (Parinaud's syndrome) and dilated pupils, slowly reactive to light. Papilledema was also present and laboratory tests led to a diagnosis of neurogenic diabetes insipidus, but not panhypopituitarism.

The brain scan was normal, but plain skull roentgenograms showed a midline calcification in the region of the pineal gland (Fig. 4). A carotid angiogram showed mild dilatation (hydrocephalus) of the lateral ventricles, while a positive contrast ventriculogram demonstrated a mass deforming the anterior third ventricle and a second, separate mass in the region of the pineal gland (Fig. 5).

An anterior craniotomy was performed and the anterior tumor was found to extend around the optic chiasm and to invade the adjacent

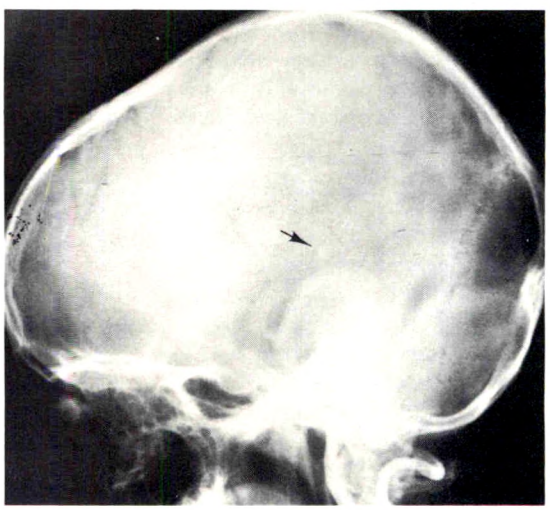


FIG. 4. Case II. Note calcification in the region of the pineal gland (arrow). This calcification was midline. The pituitary fossa is generous in size.

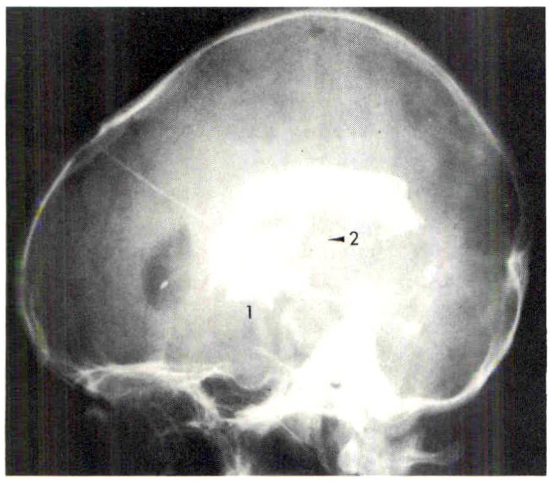


Fig. 5. Case II. Positive contrast ventriculogram demonstrates the anterior (1) and posterior (2) lesions. The concave truncated deformity of the anterior third ventricle is similar to that seen in Case I. The calcification in the center of the posterior lesion is seen again, and the posterior tumor is producing deformity and truncation of the posterior third ventricle and aqueduct.

brain stem. Histologic diagnosis was atypical teratoma (Fig. 6). The patient was shunted and treated with radiation therapy (5,000 r) to both tumor sites. The entire neural axis was not prophylactically irradiated.

Two years after the initial diagnosis the patient, on pitressin therapy, is doing well.

Case III. S.L. A 12 year old male presented with vomiting in April, 1972. He was seen by

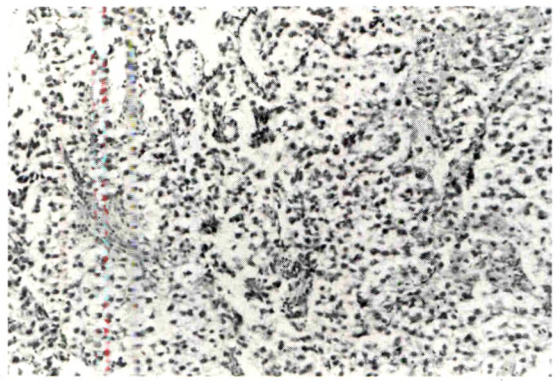


Fig. 6. Case II. Histologic section: atypical teratoma. Note that the tumor is composed of cells with fairly abundant cytoplasm. Vacuolated cells suggest early degenerative changes. Also note the elongated fibrous trabeculae containing small lymphocytes. The findings are consistent with those of atypical teratoma.[24] (H and E×100.)

his family physician, who also noted that his urine specific gravity was low. In September, 1972, because of persistent difficulties, he was referred to our hospital, and at this time, in addition to the problem of vomiting, he also related a history of nocturia (2 to 3 times a night). Physical examination was normal, but laboratory tests led to a diagnosis of neurogenic diabetes insipidus and mild panhypopituitarism. A brain scan was normal.

Plain skull roentgenograms showed a calcification in the region of the pineal gland (Fig. 7, *A* and *B*), and a pneumoencephalogram demonstrated 2 tumors, one in the region of the

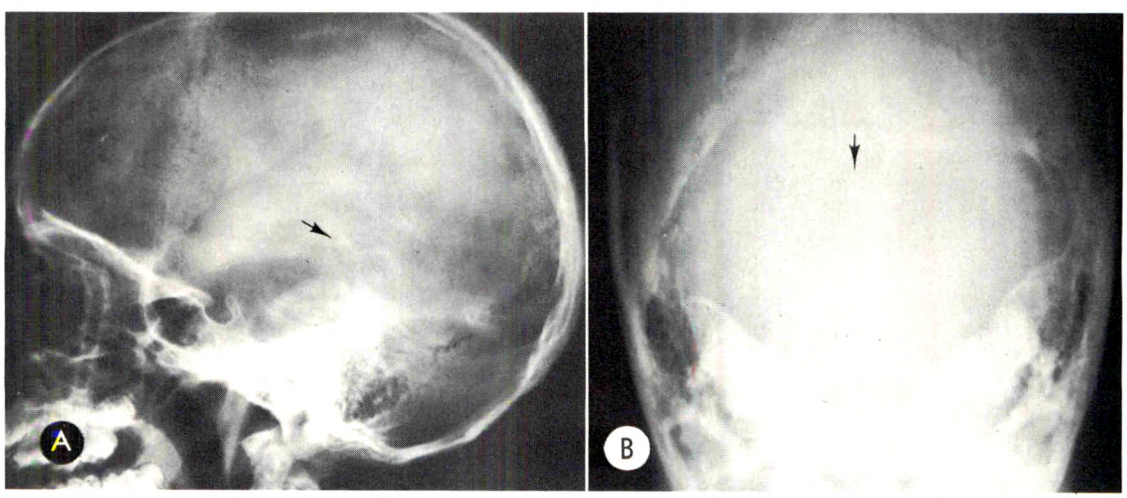


Fig. 7. Case III. (*A*) Note small calcification in the region of the pineal gland (arrow). The sella is normal. (*B*) Towne's projection shows the calcification to be just to the right of the midline (arrow).

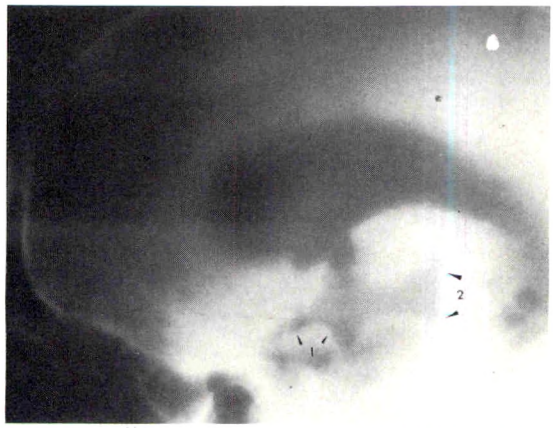


FIG. 8. Case III. Pneumoencephalogram demonstrates both the anterior (1) and posterior (2) lesions. Calcification in the posterior lesion is noted again and the tumor has produced complete obliteration of the pineal and suprapineal recesses (arrows). In addition, there is partial compression of the aqueduct. The anterior lesion is producing a concave, truncated deformity of the third ventricle (arrows), identical to that seen in Cases I and II.

anterior third ventricle and another in the area of the pineal gland (Fig. 8). A presumptive diagnosis of atypical teratoma was made, and no operative procedure was attempted.

The patient was treated with radiation therapy (5,000 r) to both lesions, and was subsequently discharged on pitressin and other appropriate replacement hormonal therapy for his hypopituitarism.

One year after treatment the patient is doing well.

CASE IV. J.R. This case was discovered in our autopsy files some time after the first 3 were studied. This 13 year old white male presented in August, 1962, with a 1 year history of nocturia (5 to 6 times a night), sleeplessness, and weight loss. Vomiting and polydipsia were present for 6 months, and dizziness and stumbling for 2 or 3 months. Physical examination demonstrated generalized weakness, pupils which were dilated and not reactive to light, and a lack of upward gaze (Parinaud's syndrome).

Roentgenograms of the skull revealed 2 intracranial calcifications (Fig. 9). One was located in the region of the pineal gland, and the other in the suprasellar region. Unfortunately, the suprasellar calcification was missed on initial inspection in 1962, and on later inspections was felt to represent an area of superimposed normal inner table sclerosis. A brain scan was also obtained at this time and was interpreted as normal, but in retrospect it is felt that an area of increased tracer activity was probably present in the suprasellar region.

A pneumoencephalogram showed a small tumor mass around the pineal gland area, protruding into the third ventricle and obliterating the pineal and suprapineal recesses (Fig. 10). The area of the anterior third ventricle was not adequately visualized, but in retrospect the anterior recesses were felt to be somewhat truncated. However, at the time of the study the area of the anterior third ventricle was considered normal and a provisional diagnosis of pinealoma was made. Radiation therapy (6,000 r over 6 weeks) was administered and the boy was discharged on replacement therapy for his endocrinologic deficits.

He returned some 9 months later with weakness of the right arm and leg, hyperactive deep tendon reflexes, and bilaterally positive Babinski responses. At this time the brain scan revealed an area of increased uptake in the frontal region, just slightly to the left of midline (Fig. 11, *A* and *B*). Radiation therapy was administered to this lesion, but improvement was only temporary. Within 4 months the patient expired.

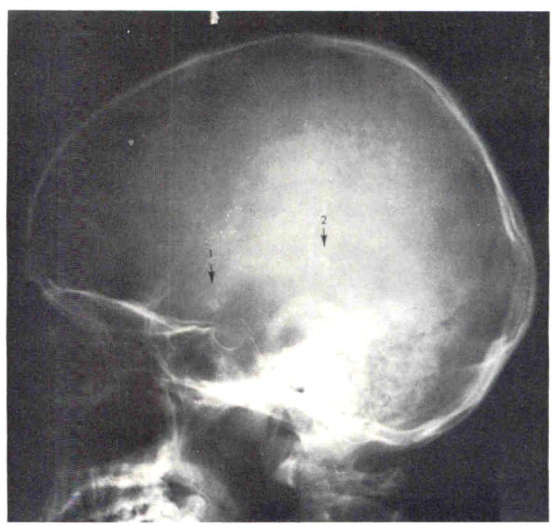


FIG. 9. Case IV. Note 2 intracranial calcifications; one located in the suprasellar region (1) and one in the region of the pineal gland (2). The sella is normal and the anterior calcification, if seen alone, could be taken to be that of a craniopharyngioma. The pineal calcification was in the midline.

At postmortem examination a 5 mm. pineal tumor, with central calcification, was identified. No specific comment was made regarding the anterior tumor site, except that the posterior lobe of the pituitary gland was atrophic. Even at this time the presence of the anterior tumor seemed to elude all concerned, and yet on the basis of the clinical findings, plain film calcification, and brain scan it was clearly present. Radiation therapy probably contributed to some decrease in its size, but it is also possible that the tumor was more infiltrative than mass-like.[20]

In the brain stem and upper cervical spinal cord a large tumor mass, measuring 5 cm., was identified. It was somewhat cystic in appearance, and on histologic examination was an atypical teratoma (Fig. 12). It was believed to be metastatic. The diagnosis was corroborated by the Armed Forces Institute of Pathology.



FIG. 10. Case IV. Pneumoencephalogram demonstrates the posterior lesion (2) to be producing truncation and a concave deformity of the posterior third ventricle. The pineal and suprapineal recesses are obliterated. The anterior calcification is just vaguely identified on this study (1), but a discrete tumor is not seen. The anterior third ventricle is poorly visualized.

DISCUSSION

Atypical teratomas of the central nervous system most frequently occur as single lesions and most are located in the suprasellar or pineal areas.[9–11,15,23,24] The occurrence of these tumors at both sites simultaneously is less well appreciated. Furthermore, the fact that they might represent a definite neuroendocrinologic syndrome has not been overly stressed, and indeed, when such a case is cited, it is usually done in passing only.[3,5,13,14,16,18,20,21,26,28]

Typically these tumors occur in males between the ages of 5 and 25 years,[20,22,25] and symptoms referable to either the anterior or posterior lesion may cause the patient to seek medical attention. However, most often it is the insidious, but progressive, onset of diabetes insipidus that heralds the syndrome. Some patients may also show signs of decreased anterior pituitary lobe function, but these changes are often subtle.[1,2,12,20,25] Most often it is the gonads that are involved first, but the adrenal and thyroid glands can also be involved, and indeed hypoadrenalism may lead to adrenal

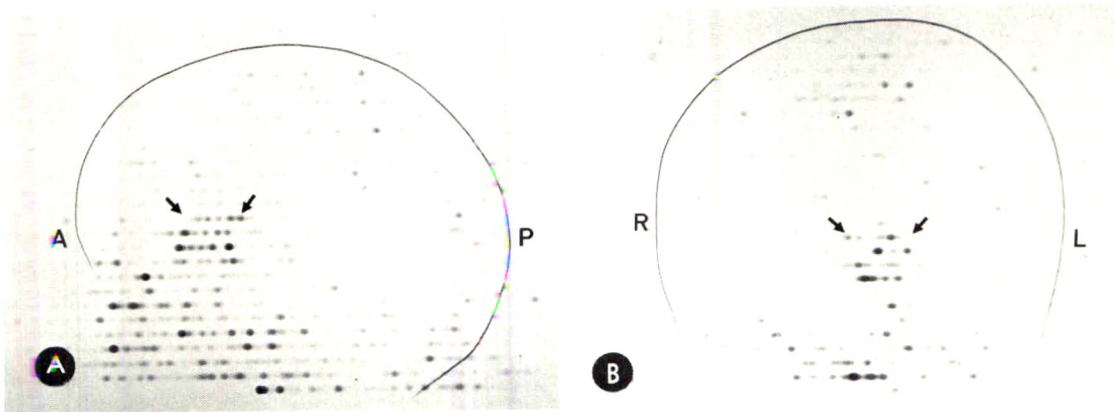


FIG. 11. Case IV. (*A* and *B*) Brain scans obtained 5 months after initial examination show a large area of increased tracer activity (arrows) in the suprasellar region. In *B* this area of increased activity is noted to lie just to the left of midline (arrows).

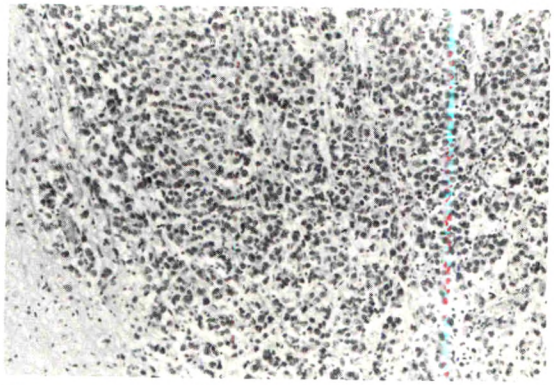


FIG. 12. Case IV. Histologic section: atypical teratoma. Metastatic tumor in the brain stem (left lower corner) consists of cells with abundant cytoplasm and linear collections of small lymphocytes which are within fibrous trabeculae. The findings are consistent with atypical teratoma[24] invading the brain stem. (H and E×100.)

crisis during pneumoencephalography or surgical exploration.

Neurologic deficit is usually limited to findings in the eyes, and is not always present from the onset.[20] Field deficits from the anterior lesion may be noted in some patients, while the posterior lesion may produce Parinaud's syndrome and loss of the pupillary light reflex. If the aqueduct is obstructed, hydrocephalus with headaches, lethargy, and vomiting may be noted.

Electroencephalograms and brain scans are usually normal in the early stages.[20] Calcification in the region of the pineal gland, however, is almost always present and is a most important roentgenographic finding. Indeed, in the presence of the proper clinical setting, such a calcification, especially in a child, should lead to the proper diagnosis. Calcification in the anterior lesion occurs less frequently and can be confused with that of craniopharyngioma. Sellar changes are usually absent or minimal.[20]

Just why these patients tend to present with symptoms referable to the anterior lesion first is not definitely known. Perhaps it is due to the large size of the tumor in some of these children. But it may also be that the anterior lesion tends to encroach upon more critical areas sooner (*i.e.*, the various hypothalamic-pituitary tracts and the hypothalamus itself). Direct extension or pressure upon the pituitary gland itself is less likely, as most of these tumors do not extend into the pituitary fossa.[20]

Ventriculography or pneumoencephalography usually demonstrate these tumors best. The anterior lesion appears as a rounded mass projecting into the anterior third ventricle, while the posterior lesion obliterates the pineal and suprapineal recesses, and if large enough, displaces and obstructs the aqueduct.

The histologic identification and classification of these tumors is often controversial, and one may be presented with a multiplicity of terms for the same lesion (*i.e.*, ectopic pinealoma, germinoma, dysgerminoma, testicular dysgerminoma, seminoma, etc.). However, most authorities consider these lesions to be atypical teratomas[3–11,19,24,25] and concur with Russell's initial impressions in 1944.[23] Atypical teratomas have long been known to metastasize, especially within the confines of the subarachnoid space,[9] and reported metastatic sites include the cerebellum, lateral ventricles, fourth ventricle, cerebral hemispheres, and spinal cord. However, when two intracranial lesions are seen from the beginning, one might justifiably ask, whether they need necessarily be considered metastatic. Indeed, there is the possibility that both tumors might be separate, that is, autochthonous in origin. This question is of more than academic interest, for the postulated origin of these tumors is important in prescribing treatment. Unfortunately, the problem is not completely resolved, although most would favor a metastatic origin.

Radiation therapy is the treatment of choice, and good results can often be obtained.[1,3,4,6,7,9,13,14,16,20,22,29] In fact, these tumors can be considered radiocurable.[22] Because of this, and because of the fact that the anterior or posterior lesion may be roentgenographically elusive in the initial stages, treatment to both sites is advisable. Our Cases I and IV speak to this

point, but the real problem is whether to prophylactically treat the entire neural axis or to simply treat both areas of the third ventricle. If one favors the concept that the 2 tumors are autochthonous, radiation of the third ventricle alone would probably suffice. If, on the other hand, a metastatic process is invoked to explain the 2 tumors, it would be reasonable and desirable to irradiate the entire neural axis. However, even with this orientation, there are some who feel that such extensive radiation therapy is not always necessary, especially when there is a negative millipore filter examination of the cerebrospinal fluid.[3,7,9,10]

In terms of differential diagnosis, we know of no other lesion which characteristically and simultaneously occurs at these 2 sites, and it is for this reason that we feel a presumptive diagnosis of atypical teratoma can be made without surgical exploration or biopsy. Although craniopharyngiomas, gliomas, adenomas, sarcoidosis, etc.[20] can occur at either site individually, it is unusual to encounter any of these lesions at the 2 sites simultaneously.

CONCLUSIONS AND SUMMARY

Although we report only 4 cases, the striking similarity of clinical presentation and roentgenographic findings in these cases, and those reported by others, causes us to strongly consider this entity a distinct neuroendocrinologic and roentgenographic syndrome.

These patients suffer from double, possibly simultaneously arising, atypical teratomas and may present with symptoms referable to one or other of the lesions. However, the anterior lesion usually produces symptoms first and characteristically there is insidious, but progressive, onset of diabetes insipidus and hypopituitarism. The posterior lesion characteristically leads to abnormal eye signs consisting of loss of pupillary light reflex and loss of upward gaze (Parinaud's syndrome).

In addition, if the lesion is large enough, aqueduct obstruction and hydrocephalus occur. The anterior lesion, if large enough, may encroach upon the optic nerves and produce visual field deficits.

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REFERENCES

1. BAUER, H. G. Endocrine and metabolic conditions related to pathology in hypothalamus: review. *J. Nerv. & Ment. Dis.*, 1959, *128*, 323–338.
2. BOOTH, C. B., SCHWARTZ, E., JANIS, R., and KRUEGER, E. G. Atypical teratoma (pinealoma): meningoencephalitic and endocrine manifestations. *Neurology*, 1963, *13*, 999–1010.
3. BRADFIELD, J. S., and PEREZ, C. A. Pineal tumors and ectopic pinealomas. *Radiology*, 1972, *103*, 399–406.
4. CUMMINS, F. M., TAVERIS, J. M., and SCHLESINGER, E. B. Treatment of gliomas of third ventricle and pinealomas: with special reference to value of radiotherapy. *Neurology*, 1960, *10*, 1031–1036.
5. CUNEO, H. M. Ectopic pinealomas. *J. Neurosurg.*, 1960, *17*, 161–165.
6. DAVIDOFF, L. M. Some considerations in therapy of pineal tumors. *Bull. New York Acad. Med.*, 1967, *43*, 537–560.
7. EL-MAHDI, A. M., PHILLIPS, E., and LOTT, S. Role of radiation therapy in pinealoma. *Radiology*, 1972, *103*, 407–412.
8. FOLKES, R. F. Pineal gland and its tumors with notes on three verified cases. *Bull. Los Angeles Neurol. Soc.*, 1964, *29*, 208–214.
9. FOWLER, F. D., ALEXANDER, E., and DAVIS, C. H. Pinealoma with metastasis in central nervous system. *J. Neurosurg.*, 1956, *13*, 271–288.
10. FRIEDMAN, N. B. Germinoma of pineal. *Cancer Res.*, 1947, *7*, 363–368.
11. GHATAK, N. R., HIRANO, A., and ZIMMERMAN, H. M. Intrasellar germinomas: form of ectopic pinealoma. *J. Neurosurg.*, 1969, *31*, 670–675.
12. HORRAX, G. Role of pinealomas in causation of diabetes insipidus. *Ann. Surg.*, 1947, *126*, 725–739.
13. HORRAX, G. Treatment of tumors of pineal body. *Arch. Neurol. & Psychiat.*, 1950, *64*, 227–242.

14. HORRAX, G., and WYATT, J. P. Ectopic pinealomas in chiasmal region. *J. Neurosurg.*, 1947, *4*, 309–326.
15. HOSOI, K. Teratoma and teratoid tumors of brain. *Arch. Path.*, 1930, *9*, 1207–1219.
16. KAGEYAMA, N. Ectopic pinealoma in region of optic chiasm. *J. Neurosurg.*, 1971, *35*, 755–759.
17. KUNICKI, A. Operative experiences in 8 cases of pineal tumor. *J. Neurosurg.*, 1960, *17*, 815–823.
18. LEWIS, I., BAXTER, D. W., and STRATFORD, J. G. Atypical teratomas of pineal. *Canad. M. A. J.*, 1963, *89*, 103–110.
19. MCGOVERN, V. J. Tumours of epiphysis cerebri. *J. Path. & Bact.*, 1949, *61*, 1–9.
20. PUSCHETT, J. B., and GOLDBERG, M. Endocrinopathy associated with pineal tumor. *Ann. Int. Med.*, 1968, *69*, 203–219.
21. RAND, R. W., and LEMMEN, L. J. Tumors of posterior portion of third ventricle. *J. Neurosurg.*, 1953, *10*, 1–18.
22. RUBIN, P., and KRAMER, S. Ectopic pinealoma: radiocurable neuroendocrinologic entity. *Radiology*, 1965, *85*, 512–523.
23. RUSSELL, D. S. Pinealoma: its relationship to teratoma. *J. Path. & Bact.*, 1944, *56*, 145–150.
24. RUSSELL, D. S. Ectopic pinealoma; its kinship to atypical teratoma of pineal gland: report of case. *J. Path. & Bact.*, 1954, *68*, 125–129.
25. RUSSELL, W. O., and SACHS, E. Pinealoma: clinicopathologic study of seven cases with review of literature. *Arch. Path.*, 1943, *35*, 859–888.
26. SIMSON, L. R., LAMPE, I., and ABELL, M. R. Suprasellar germinomas. *Cancer*, 1968, *22*, 533–544.
27. SMITH, R. A. Pineal tumors. *Univ. Mich. M. Bull.*, 1961, *27*, 33–43.
28. STRINGER, S. W. Diabetes insipidus associated with pinealoma transplant in tuber cinereum. *Yale J. Biol. & Med.*, 1934, *6*, 375–383.
29. SUZUKI, J., WADA, T., and KOWADA, M. Clinical observations on tumors of pineal region. *J. Neurosurg.*, 1962, *19*, 441–445.

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FRONTAL EPIDURAL HEMATOMA*

THE ANGIOGRAPHIC DIAGNOSIS WITH A NEW FINDING

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IN individuals who have sustained anterior polar brain injury, the differentiation of brain contusion or "pulping," subdural hematoma, and epidural hematoma can be difficult clinically and angiographically.[1–3,5,6,12]

It is the purpose of this presentation to report 2 instances of frontal head trauma in which an angiographic observation by one of us (D.D.) led to the correct preoperative diagnosis of frontal epidural hematoma. From our review of the literature, this angiographic finding has not previously been recorded. In both patients a posterior displacement of a meningeal branch of one of the ethmoidal arteries of the ophthalmic artery system was observed. Definitive angiographic diagnosis led to prompt surgical exploration and evacuation of an epidural hematoma in both cases. The absence of displacement of this artery in a third patient with otherwise similar clinical presentation and angiographic findings contributed to a diagnosis of frontal lobe contusion, a conservative approach in clinical management, and complete recovery without craniotomy.

REPORTS OF CASES

CASE 1. A 15 year old Mexican-American male was admitted to the White Memorial Medical Center following head trauma. At the time of admission he was lethargic, showed a short attention span, but did respond to commands. The right pupil was larger than the left and bilateral Babinski reflexes were elicited. Skull roentgenograms disclosed no fracture. An echoencephalogram (A-mode) showed a 2 mm. shift of the mid-line structures to the left. On the third hospital day he began to show some signs of circumorbital hematoma but the neurologic findings remained unchanged. Because of neurologic stability and the initial clinical impression that the patient had sustained cerebral contusion and concussion, no surgical intervention was instituted at that time. On the fifth hospital day a cerebral angiography was performed with imaging in the anteroposterior, lateral and oblique projections.

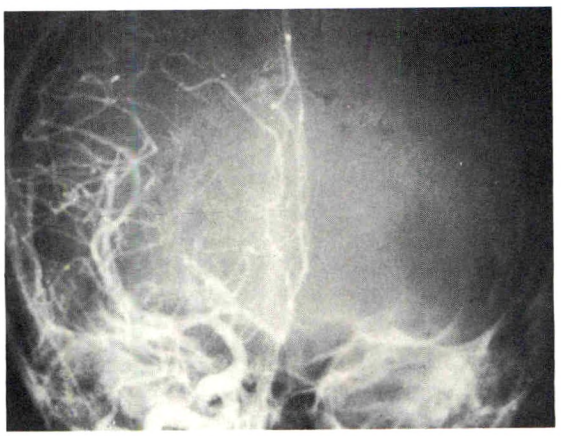


FIG. 1. Case 1. The pericallosal arteries are shifted 4 mm. to the left.

The cerebral angiogram revealed a 4 mm. shift of the pericallosal arteries to the left (Fig. 1), a stretching of the frontopolar branches of the right anterior cerebral artery on lateral projection (Fig. 2), and associated posterior displacement of the sylvian triangle and pericallosal artery. In addition a posterior displacement of a meningeal branch of the right ophthalmic artery system was noted (Fig. 3). The demonstration of this displaced vessel was significantly improved on a magnified subtraction view (Fig. 4). The right posterior oblique projection also disclosed an avascular extra-axial space at the anterior pole of the right frontal lobe. The avascular extra-axial mantle was interpreted to be an epidural hematoma rather than a subdural hematoma because of the associated displacement of the meningeal

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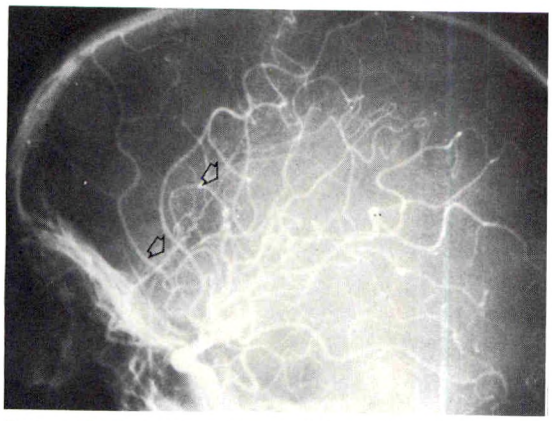


FIG. 2. Case 1. There is stretching of the frontopolar branches of the right anterior cerebral artery with associated posterior displacement of the pericallosal artery and sylvian triangle vessels consistent with an anterior polar mass effect. Posterior displacement of a meningeal branch of the right ophthalmic artery system is observed (open arrowheads).



FIG. 3. Case 1. A subtraction angiogram in the right posterior oblique projection reveals an avascular extra-axial space at the anterior pole of the right frontal lobe. The open arrowhead identifies the same meningeal branch observed in Figure 2.

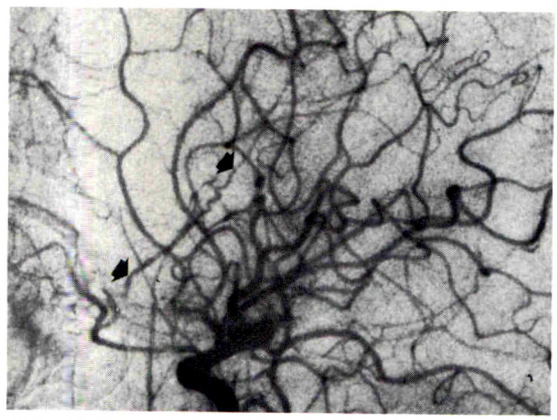


FIG. 4. Case 1. Magnified subtraction view better demonstrates the posteriorly displaced meningeal branch of the ophthalmic artery system (closed arrowheads). The vessel can be traced back to the ophthalmic artery and is probably a branch of the posterior ethmoidal artery.

branch of the ophthalmic artery system.

At surgery, a large epidural hematoma of approximately 175 cc. was evacuated from the anterior fossa. A fracture line (not visible on the roentgenograms) across the distribution of a small meningeal artery involving only the internal table of the skull was observed. This was believed to be the source of the epidural bleeding.

The postoperative course was uneventful and the patient recovered without neurologic deficit.

CASE II. A 13 year old Caucasian female was brought to the White Memorial Medical Center Emergency Room following an automobile accident. She was stuporous and agitated and exhibited bilateral Babinski reflexes, but was able to move all extremities. She also had multiple lacerations and abrasions about the head. She was admitted to the hospital for observation and on the second day demonstrated signs of decerebration on the left side with a partial third cranial nerve palsy.

Bilateral carotid arteriography was performed with imaging in the anteroposterior, lateral, and left posterior oblique projections. A 1.2 cm. round shift of the anterior cerebral and pericallosal arteries from left to right (Fig. 5) was observed in the anteroposterior view. The lateral projection (Fig. 6) revealed posterior displacement of the pericallosal artery and frontopolar branches of the anterior cerebral artery as well as similar displacement of

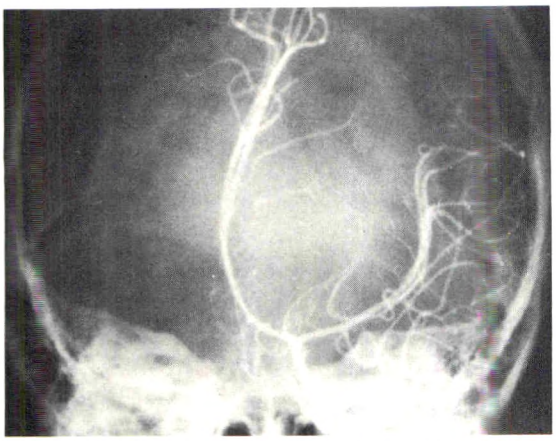


Fig. 5. Case II. There is a round shift of the anterior cerebral and pericallosal arteries from left to right.

the sylvian triangle vessels. An avascular space was observed to be an extra-axial mantle on the left posterior oblique projection (Fig. 7). Again (as in Case I) the depression, posterior displacement and arching of a meningeal branch of the ophthalmic artery system, observed best in lateral projection, resulted in a definitive angiographic diagnosis of acute epidural hematoma.

Trephines were placed in the frontal bone and a relatively large volume of epidural blood was evacuated.

Although the patient experienced a stormy postoperative course and later spent some time in the rehabilitation unit, she exhibited virtually no demonstrable neurologic deficits 1 year following surgery.



Fig. 6. Case II. Posterior displacement of the pericallosal artery and frontopolar branches of the anterior cerebral artery indicates the presence of an anterior polar mass lesion. Closed arrowheads outline the course of a posteriorly displaced meningeal branch of the ophthalmic artery system. This vessel is traced back to the ophthalmic artery (open arrowhead).

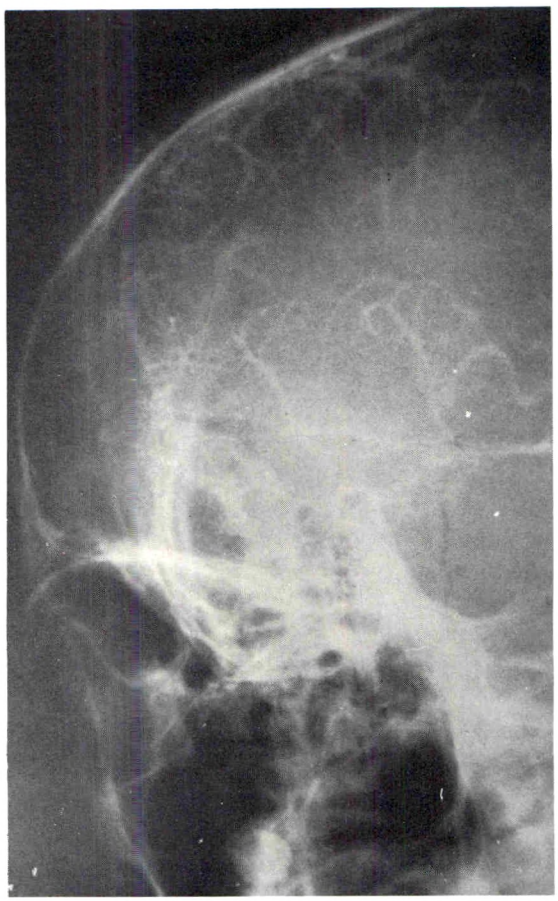


Fig. 7. Case II. An avascular extra-axial mantle in the left posterior oblique projection suggests either a subdural or epidural hematoma. Because of the displaced meningeal branch of the ophthalmic artery system (Fig. 6), a definitive angiographic diagnosis of epidural hematoma was made.

DISCUSSION

The angiographic findings in frontal epidural hematoma mimic those observed in intracerebral hemorrhage or "pulping" as described by Davis and Coxe.[2] It is of interest to note that the anteroposterior and lateral illustrations of our Case I are almost identical to many of the illustrations presented by those 2 authors. Others have stressed that additional information is provided by oblique projections.[8] The demonstration of an avascular extra-axial mantle provides evidence of a subdural or epidural

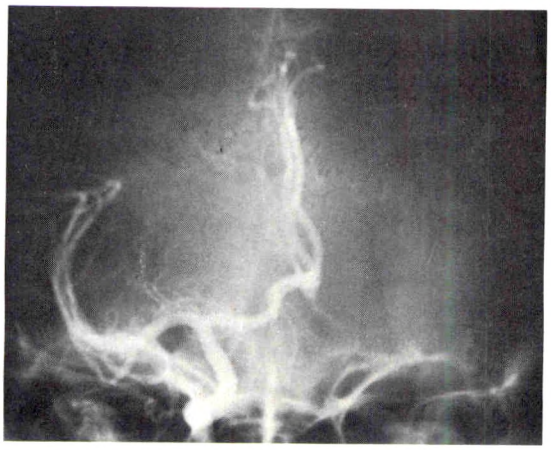


FIG. 8. Displacement of the pericallosal arteries from right to left was observed in a patient with cerebral edema in the right anterior polar aspect of the brain following concussion.

hematoma, but does not differentiate well between these 2 diagnoses in the frontal area. We believe that the displacement of the meningeal branches of the anterior or posterior ethmoidal arteries of the ophthalmic artery system, such as occurred in the 2 cases presented here, may be an important clue favoring the diagnosis of epidural hematoma. Although we have not been afforded the opportunity as yet of testing this hypothesis with the absence of the finding in a suitable case of frontal subdural hematoma, we have noted the absence of such displacement in a patient with clinical and angiographic findings of cerebral edema in the anterior polar aspect of the brain following concussion (Fig. 8; and 9). In this instance the absence of the sign supported the clinical decision for a conservative approach to management of the patient. Craniotomy was not performed and the patient has returned to the pretrauma neurologic state with a 1 year follow-up.

Epidural hematoma has its difficult and treacherous facets clinically as well. It may occur following a trivial blow or a minor fall, resulting in a tearing away of the dura from the internal table of the skull.[5] In the early course, the patient may seem to be recovering from a concussion and then quite suddenly exhibit signs of deepening cerebral depression leading to death if not treated. The mortality rate of extradural hematoma has been reported from 27 to 50 per cent in most series and as high as 86 per cent in one series.[6] In about one-third of the cases of extradural hematoma, the pathology is not in the middle fossa, but in atypical locations including the anterior polar areas.[5] McKissock *et al.*,[6] in reporting on 126 cases of epidural hematomas, found 9 in the frontal area. Two of the deaths in this series were in patients with frontal epidural hematoma, who had multiple bilateral exploratory trephines in the parietal and temporal regions without discovery and evacuation of the blood which was in the anterior fossa. Also noteworthy is the fact that the majority of their patients were young, over one-half being under the age of 15 years. It can be postulated that one primary reason for the high mortality associated with extradural hematoma is that it is not infrequently missed when occurring in one of the atypical sites. Fracture may involve the internal table of the

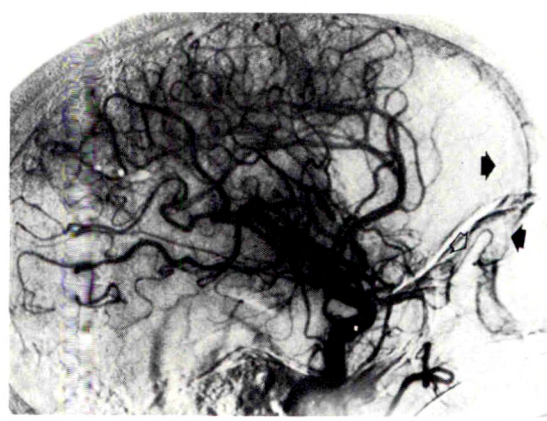


FIG. 9. The subtraction angiogram of the same patient as in Figure 8 reveals posterior displacement of the pericallosal, frontopolar, and sylvian triangle vessels. However, a meningeal branch of the ophthalmic artery system (closed arrowheads) is not displaced posteriorly. This is probably a branch of the anterior ethmoidal artery and can be traced back to the ophthalmic artery (open arrowhead). The patient survived without craniotomy with a clinical and angiographic diagnosis of concussion and cerebral edema.

skull, as in 1 of our cases, and not be visualized on skull roentgenograms. Such fractures not uncommonly occur in association with epidural hematoma.

The anatomy of the meningeal branches of the ophthalmic artery system has been described in some detail by Kuru.[4] The anterior meningeal and the anterior falcial arteries are quite commonly described vessels.[7,9] The vessels that are demonstrated and exemplified by our cases are apparently meningeal branches of the ophthalmic artery complex[11] and probably actually branches of one of the ethmoidal arteries. In our cases they have been traced back on magnification subtraction angiograms to an ophthalmic artery origin. For that reason we refer to them as meningeal branches of the ophthalmic artery system. They apparently arise from either or both the anterior and posterior ethmoidal arteries which arise from the ophthalmic artery.[10] They supply the dura mater in the anterior polar area.

Angiographic demonstration of the meningeal branches of the ophthalmic artery system in the anterior fossa is not always possible. Meticulous technique enhanced by magnification subtraction efforts are helpful. The examples presented in this article were in patients in their second decade of life. Attempts at demonstrating these vessels in elderly patients have been less satisfactory. However, they are quite consistently recognized in elderly patients with occlusive disease of the internal carotid artery proximal to the origin of the ophthalmic artery. This is presumably due to the development of a rich collateral circulation. Similarly, they have been reported as prominent in patients with subfrontal meningioma, falx meningioma, frontopolar arteriovenous malformation, epidural lymphoma, Paget's disease of the frontal bone, and metastatic tumor invasion of the frontal dura.[7]

In conclusion, we recommend that when selective carotid angiographies are performed on patients who have sustained frontal head injury, imaging be carried out in the anteroposterior, lateral and appropriate oblique projections. The study should include subtraction roentgenograms, particularly in the lateral projection, on which a search for displacement of the meningeal branches of the ophthalmic artery system may be made. If such displacement is documented, a diagnosis of epidural hematoma is favored and immediate surgical intervention may be life saving. The visualization of the meningeal branches of the ophthalmic artery is more likely in young patients.

SUMMARY

The angiographic recognition of displacement of the meningeal branch of the anterior or posterior ethmoidal artery of the ophthalmic artery system—a sign not previously reported in the literature—led to the correct preoperative diagnosis of frontal epidural hematoma in 2 patients with frontal head injury.

Furthermore, the validity of the sign is strengthened by its absence in a third patient, who also sustained anterior polar brain trauma, otherwise demonstrated classical angiographic manifestations of post-traumatic frontopolar cerebral edema, and recovered without craniotomy.

The differential diagnosis of anterior polar space occupying lesions of traumatic origin is reviewed.

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REFERENCES

1. Cronqvist, S., and Köhler, R. Angiography in epidural haematomas. *Acta radiol.* (Diag.), 1961, *1*, 42–52.
2. Davis, D. O., and Coxe, W. S. Angiographic evaluation of anterior polar brain injury. *Radiology*, 1969, *93*, 1061–1066.
3. Higazi, I., and El-Banhawy, A. Value of angiography in diagnosis of extradural hematoma of anterior fossa. *J. Neurosurg.*, 1966, *24*, 765–771.
4. Kuru, Y. Meningeal branches of ophthalmic artery. *Acta radiol.* (Diag.), 1967, *6*, 241–251.
5. Lewin, W. The Management of Head Injuries.

Baillière, Tindall & Cassell, London, 1966, p. 85.

6. McKISSOCK, W., TAYLOR, J. C., BLOOM, W. H., and TILL, K. Extradural haematoma: observations on 125 cases. *Lancet*, 1960, *2*, 167–172.
7. POLLOCK, J. A., and NEWTON, T. H. Anterior falx artery: normal and pathologic anatomy. *Radiology*, 1968, *91*, 1089–1095.
8. SCHECHTER, M. M., and ZINGESSER, L. H. Special procedures in management of traumatic lesions of head and neck. *Radiol. Clin. North America*, 1966, *4*, 53–74.
9. STATTIN, S. Meningeal vessels of internal carotid artery and their angiographic significance. *Acta radiol.*, 1961, *55*, 329–336.
10. WARWICK, R., and WILLIAMS, P. L. Gray's Anatomy. Thirty-fifth edition. W. B. Saunders Company, Philadelphia, 1973, pp. 635–637; 986.
11. WHEELER, E. C., and BAKER, H. L., JR. Ophthalmic arterial complex in angiographic diagnosis. *Radiology*, 1964, *83*, 26–35.
12. WORTZMAN, G. Roentgenologic aspects of extradural hematoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *90*, 462–471.

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ULTRASONIC DIAGNOSIS OF SUBDURAL HEMATOMAS, EPIDURAL HEMATOMAS, BRAIN EDEMA AND BRAIN ATROPHY*

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A PREVIOUS paper[2] reviewed the literature on ultrasonic diagnosis of subdural hematomas. The technique outlined in this review and another study of subdural hematomas utilized nonfocused transducers.

In this previous paper[2] visualization of the lateral surface of the brain with adjacent meninges and bone by a focused transducer revealed that a subdural hematoma caused an extra or third echo sandwiched between the normally visualized "inner table cerebrospinal fluid brain interface complex" and gray-white matter interface. It was stated at the conclusion that with more sophisticated instrumentation a better picture could be obtained.

This is now provided by the Hoffrel 21A ultrasonoscope used in conjunction with a 2 megacycle transducer having a 2.3 cm. focal distance. This equipment provides great near side resolution, giving an anatomic picture of the lateral surface of the brain and adjacent structures. The "main bang" artifact, a machine artifact which obscures visualization immediately beneath the sound transducer, is virtually eliminated by this equipment.

TECHNIQUE

Our method of procedure in examining is the same for all lesions. We will refer henceforth to the procedure as the procedure for subdural hematomas. All of the studies in this current series were done in the A mode of ultrasound. Echoes produced at tissue interfaces appear as parallel vertical lines of varying intensity. The greater the acoustical impedance mismatch at a particular interface (defined as density times velocity on either side of the interface), the greater the reflected energy, resulting in a brighter echo.

The position of the transducer for the subdural examination is as follows: As the subdural fluid collection might be present only in small localized areas, we examine each patient at a minimum of 5 positions on either side of the head. Additional positions are added when indicated by the clinical history. Our choice of positions is somewhat arbitrary and we occasionally vary them from patient to patient; however, it is important to note the location of each examining site on a given patient in order to be able to return to these positions in subsequent follow-up examinations. We use the following 2 lines as standard reference for all examinations and measure the examining sites from them for easy reproducibility. The first is the canthomeatal line. Line 2 is drawn through the external auditory meatus and perpendicular to the canthomeatal line. The first 3 points are on Line 2, at approximately 6, 8 and 10 cm. above the external auditory meatus. In infants, the 10 cm. position is eliminated and one at 4 cm. is used. Anterior and posterior points are generally located 6 to 10 cm. above the canthomeatal line and 3 to 5 cm. anterior or posterior from Line 2. The exact choice of these positions (Points 4 and 5) will vary slightly depending on the size of the patient's head, and in the case of trauma patients, the location of the trauma.

Once the site is chosen, the hair is pushed

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aside and a sufficient amount of ultrasonic coupling gel is applied. The transducer is then firmly applied to the head and a slight wiggling motion is used to see which echoes are the strongest; at the same time care must be taken so that the transducer does not shift position. The clipping and fine gain are off. The near gain control and the damping are manipulated. Before the near gain is adjusted in order to bring out the near field echo complex consisting of outer and inner tables of the skull, brain surface and gray-white matter interface, we have found it necessary to first measure the inner table to skin-air interface distance on the far side as well as on the near side (by palpating the skin on the far side, the identification of the skin-air echo is facilitated). This measurement on the far side is made initially and the value obtained (ranging from 0.8 cm. to 1.3 cm. in adults, less in children) is used as a check in identifying the inner table echo in the near side complex. While it is true that this value will vary slightly from position to position, even on the same patient, we have found that this single determination is sufficient (the outer table is also located on the near side). The correct identification of the inner table echo is important in this examination in order to differentiate it from a ghost generated by reverberation between the outer table and the transducer which sometimes appears near it with almost equal intensity. The physical properties of ultrasonic waves are such that ghosts are often produced when 2 or more parallel interfaces are present. The examination for subdural fluid collections requires the delineation of several virtually parallel tissue interfaces and is complicated by the presence of these ghost echoes which under certain conditions are almost as intense as the inner table echo as well as the echo from the brain surface. With larger subdural fluid collections, above 8 mm. in width, we have not found the presence of ghost echoes to be as much of a problem. As with most ultrasound examinations, the echo pattern is both pressure and angle dependent. A very slight wiggling motion as mentioned previously has been found to be useful in reducing some of the artifacts caused by these 2 factors.

The near gain needed to bring out the full near field complex will vary from patient to patient, depending on skull thickness, age, the amount of overlying tissue and the presence of any pathologic processes in the area examined.

In addition to the near gain control, the damping control on this unit is also utilized in this procedure. If the near gain is increased without using the damping control at the same time, it becomes very difficult to separate the various echoes. Increasing the damping control minimizes the main bang artifact which would interfere with interpretation if the near gain alone was used. Care must be taken not to over-damp as echoes emanating from tissue interfaces of interest could accidently be eliminated. As the near gain is slowly increased, the first echo to the right of the "main bang," usually seen is the one corresponding to the outer table of the skull. In adults it occurs most often between .5 cm. and .7 cm. from the skin. Next, the inner table of the skull will be visualized between .8 cm. and 1.3 cm. Immediately adjacent to the inner table echo one generally encounters the first ghost echo. This is the previously mentioned ghost caused by reverberation between the outer table and the transducer surface and, while usually of lower intensity than the inner table echo, it can sometimes appear as bright depending on transducer pressure and angle. The location of this ghost can be predetermined, however, by doubling the distance from the skin to the outer table echo and most often is between 1.0 and 1.3 cm. from the skin. Knowing this, and having previously approximated where the inner table echo will occur by the technique described earlier, the separation of these 2 echoes is possible.

As the near gain is increased further, the brain surface echo is generally the next to appear. This is also the region, between 1.4 and 1.6 cm. from the skin, where the second

ghost echo is also found. This echo is generated by a reverberation between the inner and outer tables of the skull and can be accurately located by adding the distance between the outer and inner table echoes to the distance from the skin to the inner table echo. When no subdural hematoma is present this ghost usually is to the right of the brain surface echo. When a subdural hematoma is present, the brain surface echo is pushed further to the right and this ghost will be found between it and the inner table echo. This second ghost tends to be of lower intensity than the brain surface echo it often accompanies, although echo intensity as mentioned above is pressure and angle dependent and one should not rely on echo intensity alone when eliminating these artifacts.

With a further increase of the near gain, the echo from the gray-white matter interface will appear between 1.5 and 4 mm. to the right of the brain surface echo. The outer table echo at this point is usually obliterated in a main bang reverberation which sometimes extends as far as the inner table echo. With utilization of the damping control present on this instrument, taking care not to over-damp and thereby distorting the inner table and brain surface echoes, this main bang artifact can be minimized. In general the gain should be raised only the amount necessary to visualize the brain surface echo and the gray-white matter interface. In this way the intensity of the ghost echoes is minimized making them even easier to identify.

The third and last major ghost echo can be located by doubling the distance from the skin to the inner table echo.

The procedure used when examining infants and small children (up to approximately 7–8 years) is slightly different from that used during the examination of adults. Because of the thin layer of overlying skin and muscle as well as the thinness of the skull, it is somewhat more difficult to separate the echoes emanating from the interfaces in question. The echoes corresponding to the inner table and brain surface are only 2–3 mm. apart, that is, except when a subdural hematoma or hygroma is present. This has led us to utilize a modified scanning procedure. With sufficient coupling gel the transducer is moved around the area under investigation and the echo pattern is observed for any unusual separation of the echoes. If a suspicious area is noted the transducer is kept on this spot and the distance between the inner table and brain surface echoes is quantitatively measured. This approach enables one to cover a greater surface area of the head in a shorter period of time.

Movement sometimes is helpful in identification of echoes. As the transducer is moved from position to position during the course of the examination, some of the echoes will shift their locations slightly. While moving, careful observation and identification of the inner and outer table echoes and where their ghosts should appear will be invaluable in clarifying the picture obtained. If the thickness of tissue overlying the skull varies, particularly due to localized edema, this will shift the position of the outer and inner table echoes and will also cause the relative position of their associated ghosts to change. In conjunction with examination over nonedematous scalp areas, this has been found to be of help in some cases where difficulty in clearly identifying the brain surface echo was encountered, since the position of the ghosts changes their position relative to the brain surface. Conversely, should the outer and inner table echoes remain fixed, a shifting echo will more than likely be that corresponding to the brain surface, indicating the presence of a localized lesion.

Too high a gain setting sometimes results in the appearance of a few low intensity echoes bracketing the brain surface echo. It is felt that they correspond to the curvature of the sulci and gyri which appear superimposed due to the width of the ultrasonic beam. Generally no problem has been found with these echoes as careful adjustments of the gain settings will usually eliminate them completely from the picture

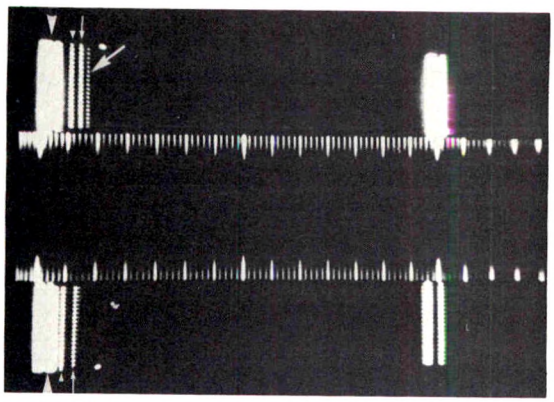


FIG. 1. Normal trace. The distance between each large marker is 1 cm. and between each small marker is 2 mm. The upper trace shows echoes from the main bang (large arrowhead), the inner table and dura of the skull (small arrowhead), the gyral brain surface (small arrow) and the gray-white matter interface (large arrow). The lower trace shows echoes from the main bang (large arrowhead), the inner table and dura of the skull (small arrowhead) and the gyral brain surface (small arrow).

without affecting the echoes from the interfaces under study.

DIAGNOSIS

The difference between the findings now and in the previous report is that now all the interfaces at or adjacent to the lateral surface of the brain are seen in their anatomic position. For example, in the normal, 4, rather than 2, echoes are demonstrated. Ultrasound shows an outer table, inner table dural echo, a gyral brain surface echo and a gray-white matter echo. These echoes show the normal anatomic distance between each other (Fig. 1 and 2). The normal for the distance between the inner table dural echo and gyral brain surface echo which we shall call the SD space is .3–.4 cm. in adults and .2–.3 cm. in children. Abnormality has only appeared above these values, therefore they are significant values. In adults values of .4 and .4–.5 cm. may or may not be abnormal. In children values of .3, .3–.4, and .4 cm. may or may not be abnormal. In addition it has been shown that a subdural hematoma of 4 mm. width will give a value of 4 mm. by echo as indicated by autopsy and angiogram correlation. Abnormality consists of the following:

1. An increased or decreased distance between the inner table dural echo and the gyral brain surface echo
2. An altered gray matter width
3. Decrease in the intensity of the gyral brain surface echo
4. An increase in the intensity of the gray-white matter echo
5. Presence of a very strong blood dural brain echo.

An increased distance between the inner table dural echo and gyral brain surface echo may indicate a unilateral subdural hematoma (Fig. 3 and 4), if found on one side and bilateral subdural hematomas if on both sides (Fig. 5). It also may indicate atrophy. If there is difficulty in deciding between the presence of a subdural hematoma and brain atrophy, the following ultrasound criteria may be of help. The midline shift may be helpful. If there is an increased subdural space on one side or if the subdural space is increased more on one side than another with a midline shift away from the larger space, a subdural hematoma is suspected. Sinking of the brain is also of help sometimes to distinguish

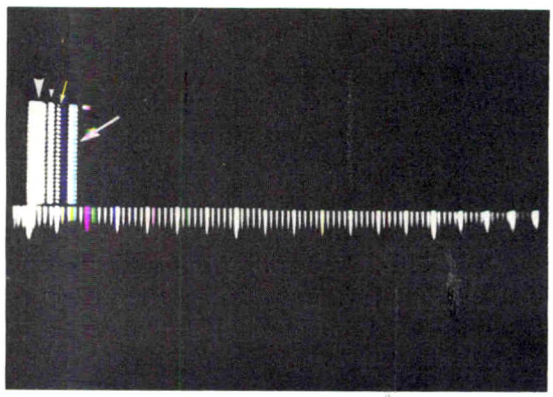


FIG. 2. Normal trace at lower sensitivity. The trace shows echoes from the main bang (large arrowhead), outer table of the skull (small arrowhead), inner table and dura of the skull (small arrow) and the gyral brain surface (large arrow).

atrophy from a subdural hematoma. With the patient lying supine when the head is turned to the side so that a sagittal section of the head is parallel to the floor, the brain sinks to the atrophic side. In cortical edema (Fig. 6) the subdural space is decreased, the gray matter width is increased, the intensity of the gyral brain surface echo is decreased (due to the fact that this interface approaches a water-cerebrospinal fluid interface) and the gray-white matter echo is increased in intensity.

An epidural hematoma has a different picture (Fig. 7). This is the only instance when the dura is visualized. A blood dural brain interface is visualized on the near side. This echo is so strong it appears even before the inner table echo at lower sensitivity.

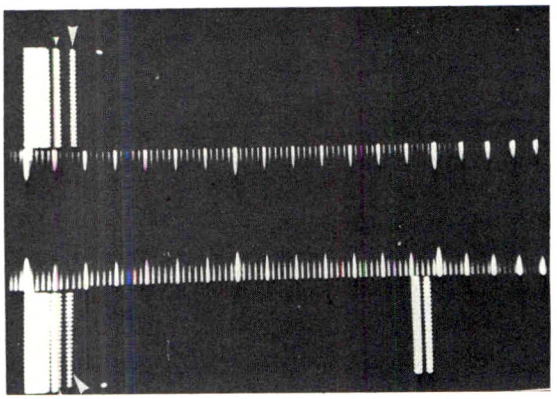


Fig. 4. Unilateral subdural hematoma. The upper trace shows the distance between the inner table dural echo (small arrowhead) and gyral brain surface echo (large arrowhead) to be increased to 6 mm. This space represents the subdural hematoma. The bottom trace shows the distance between the inner table dural echo (small arrowhead) and gyral brain surface echo (large arrowhead) to be normal (4 mm.).

CLINICAL FINDINGS

One hundred and seventy patients with head trauma, suspected subdural or epidural fluid collections, reaccumulation of subdural fluid, suspected brain atrophy, suspected brain edema due to contusion or those with abnormal midline shifts were studied. Of these, 13 subdural hematomas in all stages were correctly diagnosed. Subdural hematomas were diagnosed in both adults and children. The diagnosis was confirmed by angiography and/or surgery. Seven of these were bilateral subdural

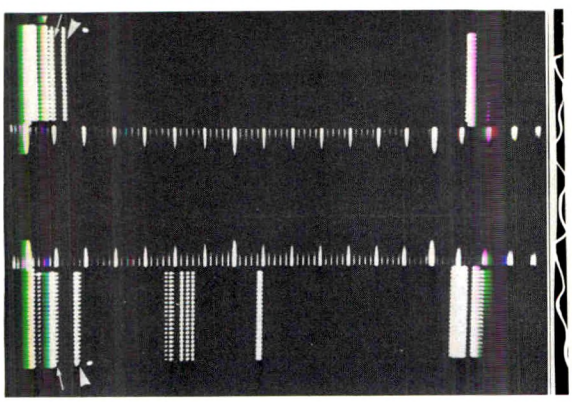


Fig. 3. Unilateral subdural hematoma. The top trace shows the distance between the inner table dural echo (small arrow) and gyral brain surface echo (large arrowhead) to be normal (4 mm.). The echo from the outer table of the skull (small arrowhead) is also seen on the top trace. The lower trace shows the distance between the inner table dural echo (small arrow) and gyral brain surface echo (large arrowhead) to be increased to 8 mm. This space represents the subdural hematoma.

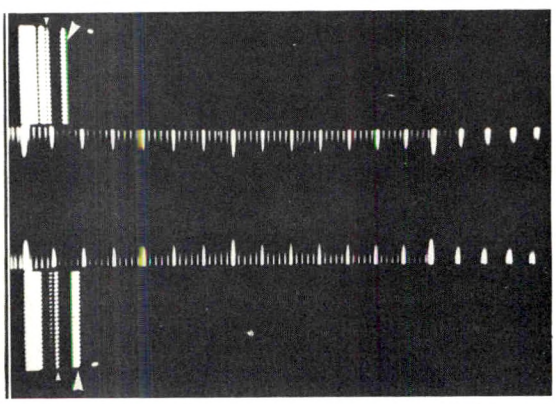


Fig. 5. Bilateral subdural hematomas. The top trace shows the distance between the inner table dural echo (small arrowhead) and gyral brain surface echo (large arrowhead) to be increased to 5 mm. This space represents the subdural hematoma. The lower trace shows the distance between the inner table dural echo (small arrowhead) and gyral brain surface echo (large arrowhead) to be increased to 6 mm. This space represents the subdural hematoma.

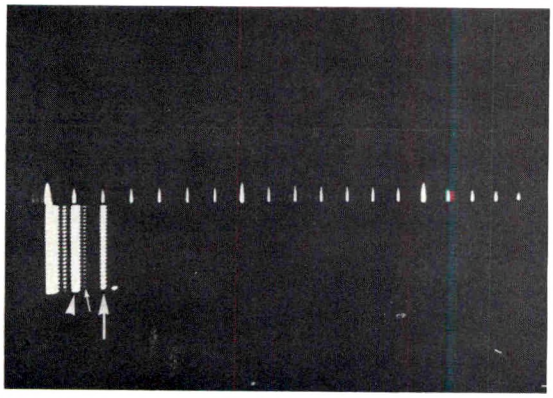


FIG. 6. Cortical edema. The trace shows the gyral brain surface echo (small arrow) near the inner table dural echo (large arrowhead). The gyral brain surface echo is weak. The gray-white matter interface echo (large arrow) is strong and the distance between it and the gyral brain surface echo is increased.

hematomas. Five cases were followed for reaccumulation of fluid giving a total of 21 positive subdural examinations. There was 1 epidural hematoma confirmed by surgery. Thirteen cases of brain atrophy determined by clinical impression (in 11 cases) and special study (in 2 cases) were diagnosed. There were 4 cases of cortical edema. One case was verified at surgery and 3 were consistent with the clinical impression.

There were 3 false positives and 1 false negative. There was 1 false positive subdural hematoma in a marked hydrocephalic due to the echo from the lateral border of the ventricle adjacent to the dura. There was a false positive epidural in a case of brain edema. In this case the strong gray-white echo was mistaken for a blood dural brain interface, since it was early in our study and the importance of a weak gyral brain surface echo very close to the inner table dural echo was not appreciated. Another case of brain edema was also missed because the weak brain surface echo was not noted and the strong gray-white echo was mistaken for the brain surface echo. This was mistakenly diagnosed as a subdural hematoma. There was one false negative before it was appreciated that .3 cm. may be an abnormal value in a child.

The width of the subdural hematomas and hygromas, the amount of brain atrophy and the width of the one epidural hematoma could be estimated as being small, moderate and large. This estimate correlated with the size found on special study.

Perhaps of great importance is the observation that small to moderate and moderate sized subdural hematomas which do not necessarily cause an abnormal shift cause mental aberrations.

SUMMARY

Use of the Hoffrel 21A ultrasonoscope in conjunction with a 2 megacycle transducer having a focal distance of 2.8 cm. gives great near side resolution.

Because of this an anatomic ultrasound demonstration of the lateral surface of the brain and adjacent interfaces is seen in the normal. Altered interface position and interface intensity can be seen in the abnormal giving a diagnostic picture.

In the abnormal as well as the normal the distances measured are the actual distances found in life, or a good approximation of those distances. Therefore, lesions such as subdural and epidural collections,

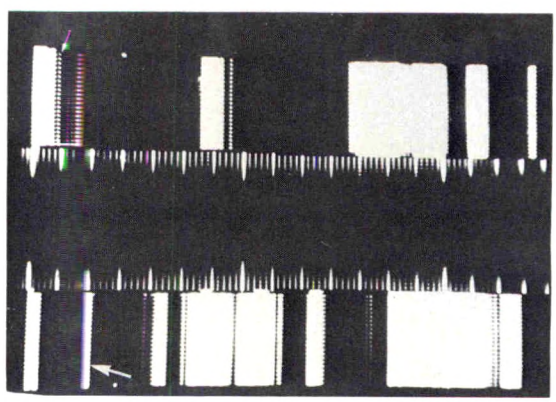


FIG. 7. Epidural hematoma. The bottom trace shows a strong blood dural brain echo (large arrow). At this sensitivity the inner table dural echo is not seen. It is seen in the upper trace (small arrow), but a strong blood dural brain echo is not seen in the upper trace.

cortical edema and brain atrophy may be diagnosed. This method has been found to be extremely accurate and sensitive.

The examination is useful for follow-up postoperative reaccumulation of fluid and resolution of brain edema.

Further work is needed on sinking of the brain as well as the width of the motor cortex in cases of brain atrophy.

The measurement of the gray matter in the motor cortex may be valuable in childhood mental retardation.

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REFERENCES

1. FOROGLOU, G., and ZANDER, E. Echoencephalography in cases of subdural hematomas. *Rev. méd. Suisse Rom.*, 1970, *90*, 131–152.
2. ROTHMAN, J., SHATSKY, S., KRICHEFF, I., and CHASE, N. Ultrasonic diagnosis of subdural hematomas. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *105*, 413–427.

ROENTGENOGRAPHIC ANALYSIS OF ARTERIOVENOUS MALFORMATIONS OF THE OCCIPITAL LOBE*

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THE blood supply of cerebral arteriovenous malformations is often extensive and depends on the cranial or intracranial location as well as the size of the lesion.[8] No previously reported study has analyzed the roentgenologic and clinical features of malformations which primarily involve the occipital lobe.

This report discusses the roentgenographic anatomy and summarizes the clinical features in 25 patients with arteriovenous malformation of the occipital lobe.

MATERIAL

A review of the angiographic files at the University of California Medical Center, San Francisco, produced 25 examples of arteriovenous malformations of the occipital lobe. The majority of the patients had been clinically evaluated by the staff of the Neuro-Ophthalmology Unit. The angiograms and clinical records were analyzed to determine if any anatomic findings could be correlated with the clinical problems caused by these lesions. The patients in the study ranged in age from 15 to 60 years with an average age of 37 years. Ten patients were women and 15 men. Follow-up periods varied from a few weeks to 10 years. Two patients died from complications after surgical procedures, and 1 patient died from an intracerebral hemorrhage 8 years after the diagnosis was established.

RESULTS

The clinical features of these cases are presented in a separate report.[11] Thirteen patients experienced visual phenomena such as flashes of light and dimming of vision: Seven had nonmigrainous, transient, cortical photopsias or scotomas and 7* described partial loss of vision. Nine patients had epilepsy and 6 of these 9 patients experienced visual aurae. Headaches were particularly common and were both acute and chronic. The headaches were predominantly unilateral and frontal—occurring on the side of the occipital lesion in all but 1 patient. Severe, "explosive" headaches signified intracranial bleeding. Nine patients had severe headache followed shortly by complete homonymous hemianopsia—the syndrome of occipital apoplexy. The most common neurologic abnormality of the initial examination was homonymous hemianopsia, present in 11 patients. The nonvisual signs included paralysis of the third nerve in 2 patients (the result of an accompanying aneurysm), hemiparesis in 5 patients, and a vascular bruit in 5 patients.

Roentgenographic diagnosis depended mainly on angiographic studies. The occipital lesions were right-sided in 11 instances and left-sided in 14. The angioma was confined to the occipital lobe in 13 patients, the occipitoparietal area in 9 patients, and the occipitotemporal region in 3 patients. The size of the malformation was classified according to the largest dimension into 3 grades: *small* (1 cm. or less), 7 patients; *medium* (2 to 4 cm.), 12 patients; and *large* (5 cm. or more), 6 patients. The roentgenographic features are listed in Table 1.

On plain roentgenograms, abnormalities of the skull were noted in only 3 patients. These abnormalities consisted of an enlarge-

* One patient had both phenomena.

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TABLE I

ROENTGENOGRAPHIC FEATURES OF OCCIPITAL LESIONS IN 25 PATIENTS*

Case No.	Location	Size (cm.)	Hematoma	Arterial Supply				Venous Drainage	Associated Aneurysm
				MCA	PCA	ECA	PERIC		
1	Occipital (R)	2×2×2	+	+	+			Superficial veins	
2	Occipital (L)	1×1×1	+		+			Superficial veins	
3	Occipitotemporal (R)	5×4×5		+	+			Dural sinus, torcula	
4	Occipital (R)	5×3×4		+	+	+		Torcula, transverse sinus	
5	Occipital (R)	3×3×4		+	+	+		Torcula, superior sagittal sinus	
6	Occipitotemporal (L)	4×3×3		+	+	+		Superficial veins	
7	Occipital (L)	2×3×2			+	+		Superficial veins	RICA
8	Occipitoparietal (R)	7×6×5		+	+			Vein of Galen, superior sagittal sinus	Basilar
9	Occipitoparietal (L)	7×4×4		+	+	+	+	Superficial veins	
10	Occipital (L)	1.5×1×1		+	+			Superficial veins	
11	Occipital (L)	4×4×4		+	+	+		Torcula, superior sagittal sinus	
12	Occipitoparietal (R)	3×3×2		+	?†	+		Superior, lateral, and straight sinuses	
13	Occipital (R)	1×1×1	+	+	+	+		Torcula	
14	Occipital (R)	3×2×2			+			Vein of Galen	
15	Occipitoparietal (L)	3×4×2		+	+		+	Superficial veins, superior sagittal sinus	Multiple
16	Occipitoparietal (L)	1×1×1	+	+	+			Superficial veins	
17	Occipitoparietal (L)	3×4×2	+	+	?†			Superficial veins	
18	Occipitoparietal (L)	3×3×3		+	+			Superior sagittal and transverse sinuses	
19	Occipital (L)	4×2×2		+	+			Straight and superior sagittal sinus	
20	Occipitoparietal (L)	1½×1½×1½	+	+	+			Superior sagittal sinus	
21	Occipitotemporal (R)	4×4×5		+	+			Superficial veins, transverse sinus	
22	Occipital (L)	1×1×1			+			Superior sagittal sinus	
23	Occipitoparietal (L)	4×6×7	+	+	+	+	+	Vein of Galen, superficial veins, dural sinuses	
24	Occipital (R)	½×½×½	+		+			Superior sagittal sinus	
25	Occipital (R)	2×2×2		+	+			Superior sagittal and transverse sinuses	
Total 25	(R) 11 (L) 14		8	20	23	9	3		3

* R=right; L=left; MCA=middle cerebral artery; PCA=posterior cerebral artery; ECA=external carotid artery; PERIC=pericallosal artery; RICA=right internal carotid artery.

† No vertebral arteriography performed.

ment of the groove of the posterior branch of the middle meningeal artery in all 3 instances, local thinning of the cranial vault in 2, and calcification within the malformation in 1 (Fig. 1, *A* and *B*). Multiple arteries supplied all but 2 of the arteriovenous malformations. The parieto-occipital, calcarine, and posterior temporal branches of the posterior cerebral artery as well as the angular branch of the middle cerebral artery were the most common contributing vessels. In each patient in whom vertebral arteriography was performed (23 of the 25 patients), it was found that branches of the posterior cerebral artery supplied the malformation (Fig. 2, *A* and *B*; 3, *A* and *B*; and 4, *A–D*). The distal branches of the middle cerebral artery contributed to the malformation in 20 patients (Fig. 3, *A* and *B*), whereas the pericallosal artery helped to supply the angioma in 3 instances. Dural contributions *via* meningeal branches of the external carotid or vertebral arteries were noted in 9 patients (Fig. 4, *C* and *D*).

Venous drainage from the malformation extended into the dural sinuses (*e.g.*, supe-

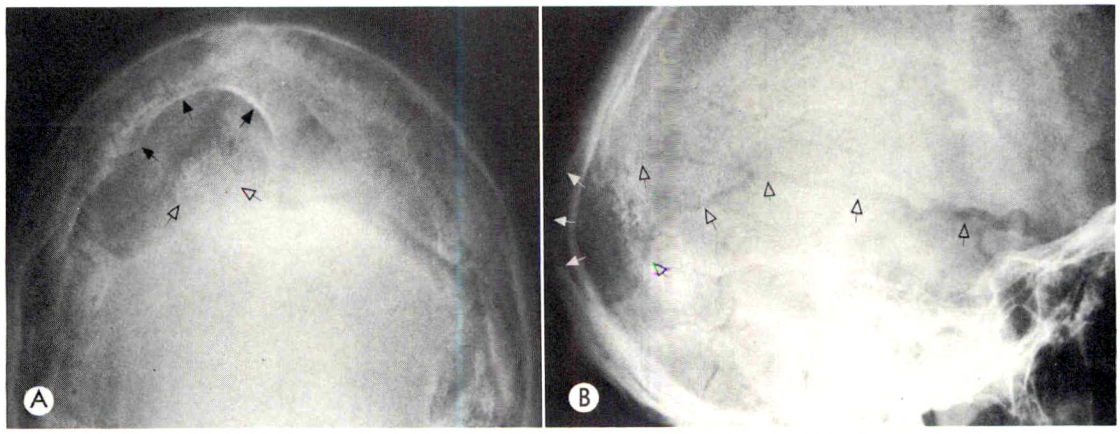


FIG. 1. Roentgenograms of the skull. (*A*) Roentgenogram in half-axial projection shows a localized thinning of the skull (closed arrows) with adjacent punctate calcifications (open arrows). (*B*) Lateral projection. The groove for the posterior branch of the middle meningeal artery is enlarged (open arrows). The localized posterior bulging and thinning of the skull vault are well shown (closed arrows).

rior sagittal sinus and transverse sinus) in 16 patients. The superficial veins drained the malformation in 11 patients. Three of the malformations drained deep into the cerebral venous system. Angiographic evidence of an intracerebral hematoma, with displacement of adjacent vessels, and an avascular zone around the arteriovenous malformation was observed in 8 of the 25 patients (Fig. 5, *A–E*).

DISCUSSION

The roentgenographic diagnosis of arteriovenous malformation depends pri-

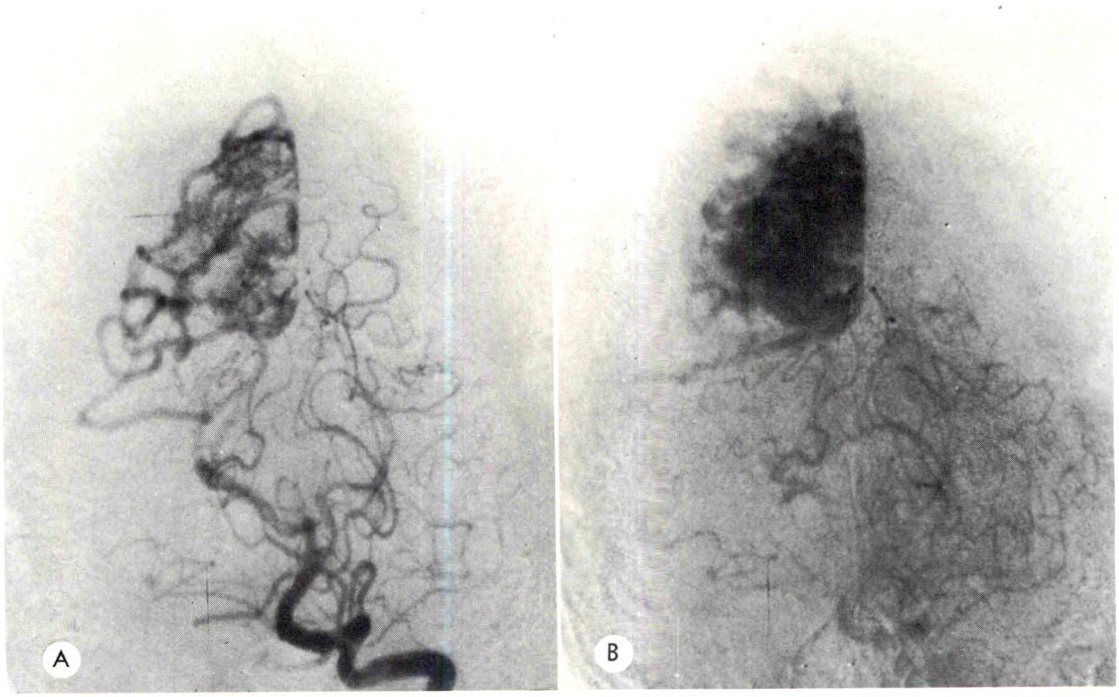


FIG. 2. Left vertebral arteriogram, frontal projection. (*A*) Arterial phase. (*B*) Capillary phase. A large arteriovenous malformation of the medial occipital lobe is supplied by enlarged calcarine and parieto-occipital branches of the right posterior cerebral artery.

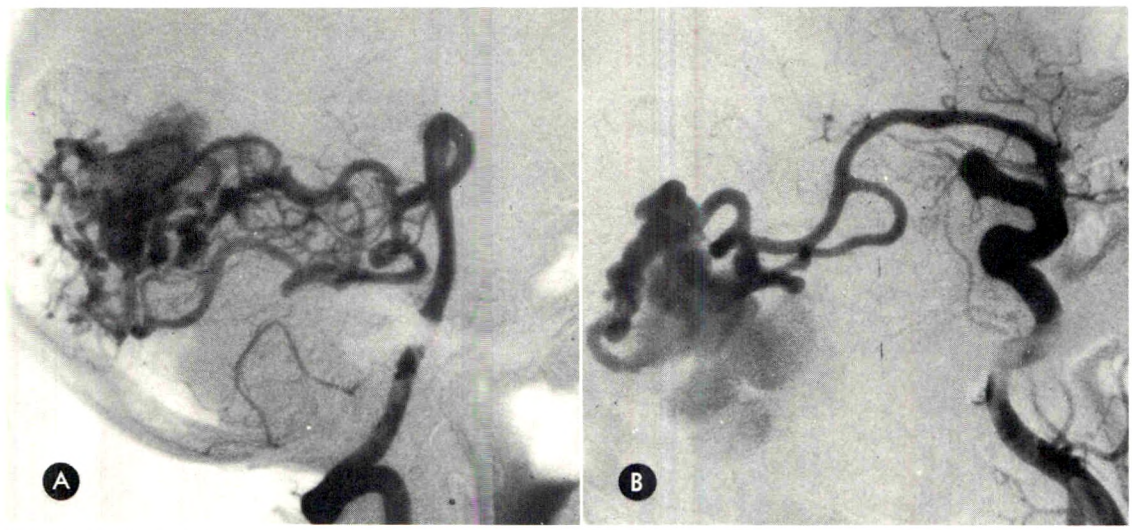


Fig. 3. (*A*) Vertebral arteriogram, lateral projection. (*B*) Carotid arteriogram, lateral projection. The large arteriovenous malformation of the occipital lobe is supplied by enlarged branches of both the posterior and middle cerebral arteries. No field defects were seen.

marily on cerebral angiography. Occasionally, however, plain roentgenograms of the skull may show signs of enlargement of the feeding arteries, calcification within the malformation, or localized erosion and bulging of the skull vault.[4,5,10] Angiography is crucial in the diagnosis and management of intracranial arteriovenous malformations that involve the occipital lobe. Rapid serial angiography is necessary to define the origin of the supplying arteries and the course of the draining veins. Subtraction and magnification techniques further aid in angiographic definition of these malformations. All arteries, as well as the draining veins that contribute to the malformation, must be carefully delineated if an accurate anatomic evaluation of the lesion is to be obtained. The angiographic study, therefore, should include opacification of all arteries which may supply the malformation. The exact nature and extent of the arterial supply to these malformations can be appreciated only by means of separate selective injection of contrast material into each vessel. In arteriovenous malformations of the occipital lobe, roentgenographic investigation must include selective vertebral as well as internal and external carotid arteriography. The dural contribution to these malformations *via* the external carotid artery is well demonstrated in Figure 4, *C* and *D*. In this patient the malformation of the occipital pole was supplied primarily by meningeal branches of the external carotid artery. This area of the lesion is shown as a relatively "avascular" region in the early phases following opacification of the posterior cerebral artery.

The presence of an associated intracerebral hematoma is recognized by the displacement of adjacent vessels as well as by an area of avascularity surrounding the malformation (Fig. 5, *A–E*). Occasionally, the hematoma may destroy portions of the malformation. This destruction is recognized by residual enlargement of afferent arteries and draining veins (Fig. 5, *A–E*). The association of aneurysms and arteriovenous malformations is well known.[1–3] Three of the patients studied had aneurysms. One aneurysm involved the distal basilar artery and one the internal carotid artery. One patient (Case 15) had 3 aneurysms on the same side as the arteriovenous malformation.

In all cases reviewed, the side of the malformation correlated with the contralateral visual field defect, when present. All patients with an intracerebral hematoma had

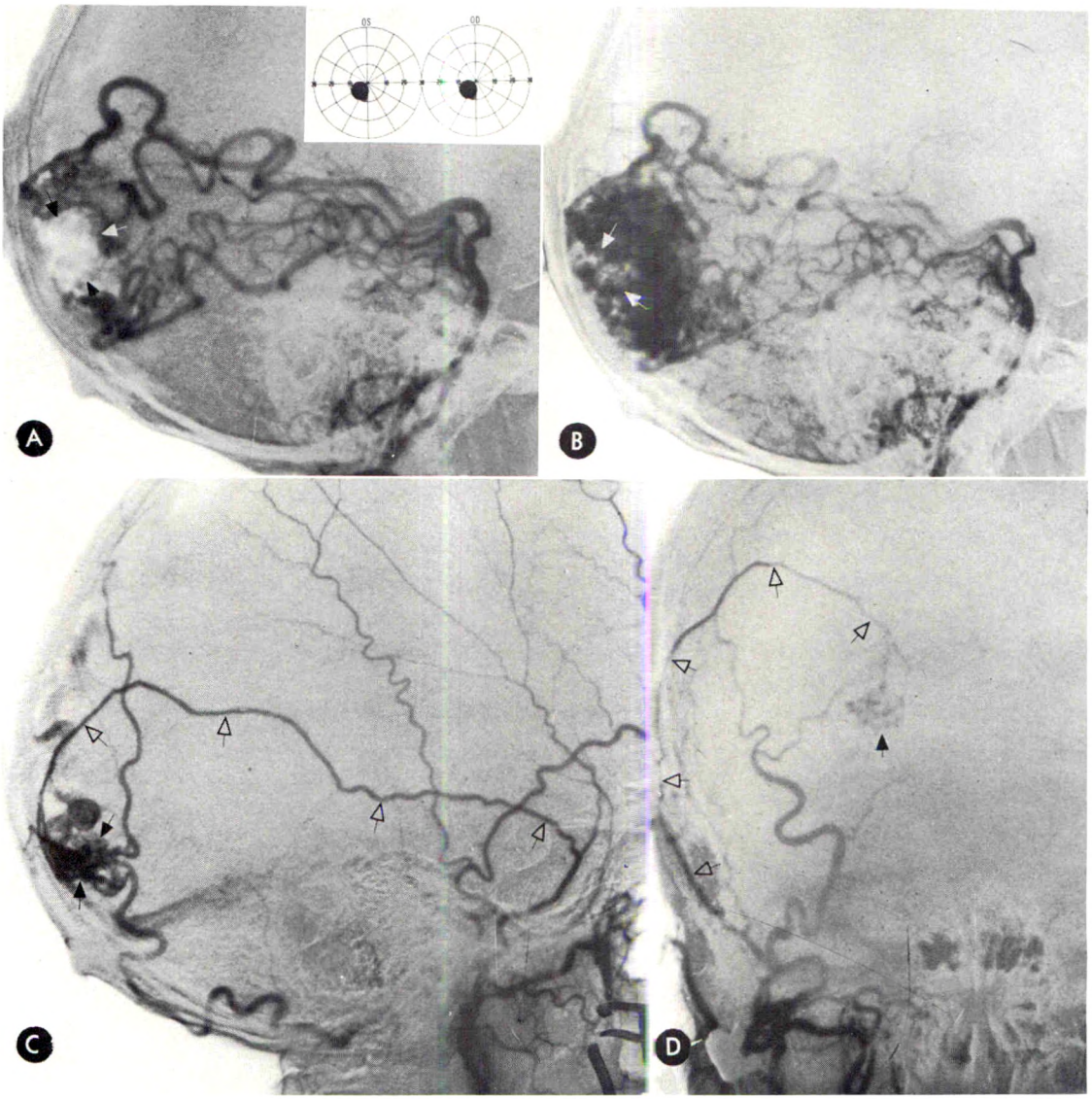


FIG. 4. (*A* and *B*) Vertebral arteriograms, lateral projection. The parieto-occipital, calcarine, and posterior temporal branches of the right posterior cerebral artery are enlarged and supply an arteriovenous malformation situated at the posterior pole of the right occipital lobe. Note area of relative avascularity in the region of the calcarine fissure (arrows) and the accompanying visual field. (*C*) Selective right external carotid arteriogram, lateral projection, and (*D*) frontal projection. The posterior branch of the middle meningeal artery is enlarged (open arrows) and supplies a small portion of the occipital lobe formation (closed arrows).

a complete visual field defect. The size of the malformation and the extent of the field loss, however, had no consistent relationship. Malformations of the occipital lobe comprise from 10 per cent[7,9] to 30 per cent[6] of the arteriovenous anomalies of the brain and usually have characteristic clinical and roentgenographic features.

SUMMARY

Roentgenographic and clinical features of 25 cases of arteriovenous malformations of the occipital lobe are summarized.

Occipital apoplexy and occipital epilepsy

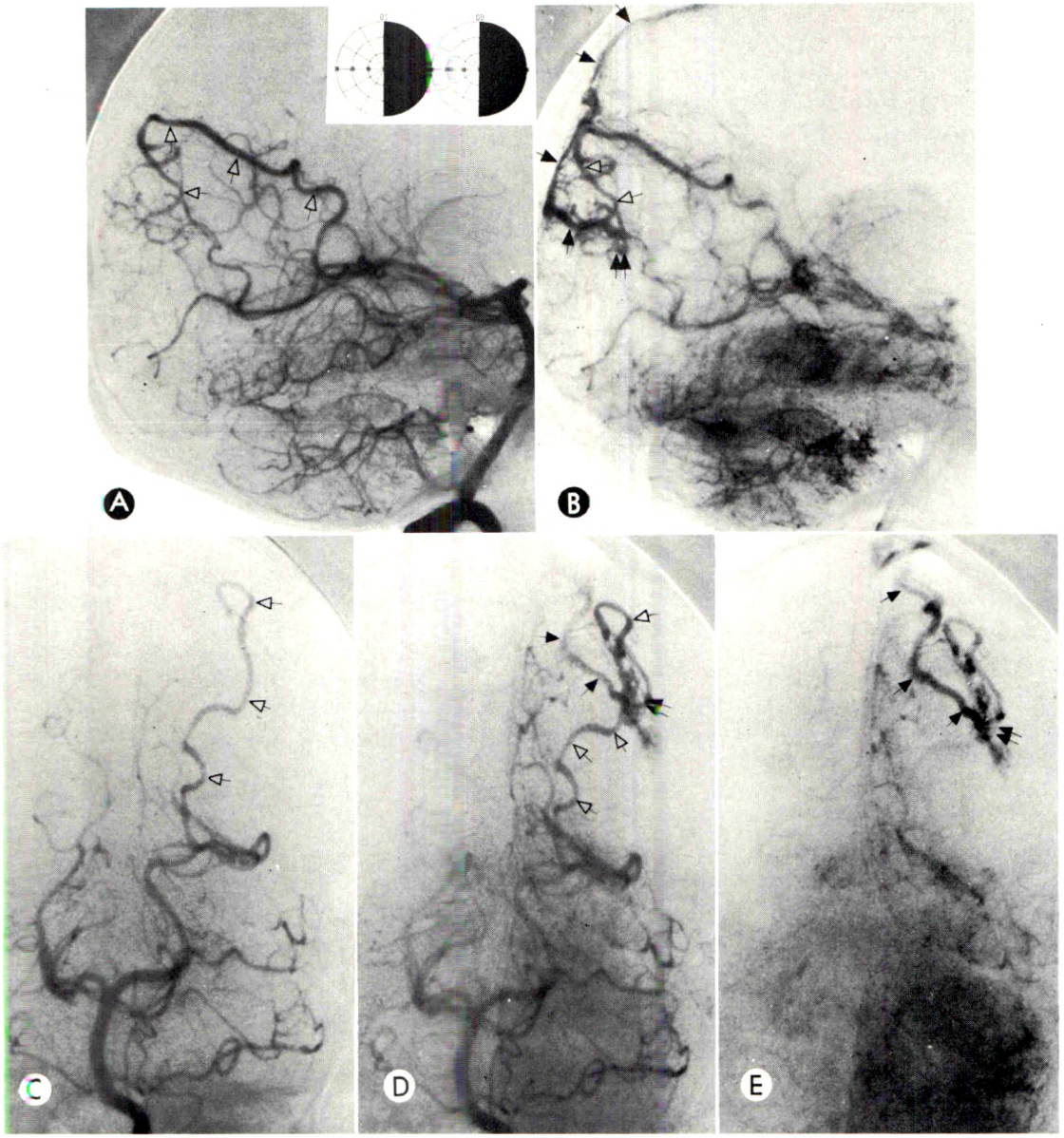


Fig. 5. (*A*) Selective vertebral arteriogram, lateral projection, arterial phase. (*B*) Venous phase. (*C–E*) Frontal projections. (*C*) Early arterial phase. (*D*) Late arterial phase. (*E*) Capillary phase. The parieto-occipital branch of the posterior cerebral artery is slightly enlarged and displaced superiorly (open arrows). The small arteriovenous malformation (double arrow) has been partially destroyed by the intracerebral hematoma and drains into veins that empty into the adjacent superior sagittal sinus (closed arrows).

are the cardinal syndromes of occipital arteriovenous malformations. The most common presenting sign, however, is a homonymous visual field defect. Size of the malformation does not correlate with presence of visual field defect.

Arteriovenous anomalies of the occipital lobe are supplied by multiple arterial sources, including distal branches of the middle cerebral artery, the posterior cerebral, external carotid, and pericallosal arteries.

Because early excision of occipital hematomas promotes recovery from visual field defects, selective arterial opacification is recommended to delineate these vascular malformations.

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REFERENCES

1. ANDERS, R. McD., and BLACKWOOD, W. Association of arteriovenous angioma and saccular aneurysm of arteries of brain. *J. Pathol. & Bact.*, 1959, *77*, 101–110.
2. BOYD-WILSON, J. S. Association of cerebral angiomas with intracranial aneurysms. *J. Neurol., Neurosurg. & Psychiat.*, 1959, *22*, 218–223.
3. CRONQVIST, S., and TROUPP, H. Intracranial arteriovenous malformation and arterial aneurysm in same patient. *Acta neurol. scandinav.*, 1966, *42*, 307–316.
4. CUSHING, H. W., and BAILEY, P. Tumors Arising from the Blood-Vessels of the Brain: Angiomatous Malformations and Hemangioblastomas. Charles C Thomas, Publisher, Springfield, Ill., 1928.
5. DANDY, W. E. Arteriovenous aneurysm of brain. *Arch. Surg.*, 1928, *17*, 190–243.
6. DIMSDALE, H. Discussion on neuro-ophthalmological aspects of cerebral angiomas. *Proc. Roy. Soc. Med.*, 1957, *50*, 85–88.
7. ENOKSSON, P., and BYNKE, H. Visual field defects in arteriovenous aneurysms of brain. *Acta ophth.*, 1958, *36*, 586–600.
8. NEWTON, T. H., and TROOST, B. T. Arteriovenous communications. In: Radiology of the Skull. Edited by T. H. Newton, and D. G. Potts. To be published.
9. PERRET, G., and NISHIOKA, H. Report on cooperative study of intracranial aneurysms and subarachnoid hemorrhage. Section VI. Arteriovenous malformations: analysis of 545 cases of cranio-cerebral arteriovenous malformations and fistulae reported to cooperative study. *J. Neurosurg.*, 1966, *25*, 467–490.
10. POETER, A. J., and BULL, J. Some aspects of natural history of cerebral arteriovenous malformation. *Brit. J. Radiol.*, 1969, *42*, 667–675.
11. TROOST, B. T., and NEWTON, T. H. Migraine of arteriovenous malformation (AVM)?: clinical and roentgenographic features in 25 cases of occipital lobe AVM. *Neurology*, 1973, *23*, 398 (Abstr.).

Vol. 122, No. 3

ANTERIOR MENINGEAL BRANCH OF THE VERTEBRAL ARTERY IN EXTRA-AXIAL POSTERIOR FOSSA LESIONS*

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THE anterior meningeal branch of the vertebral artery has been shown to be enlarged in various posterior fossa extra-axial tumors. These include glomus jugulare, meningioma, hemangioblastoma, metastatic hypernephroma, and plasmacytoma.[1,3,4]

We would like to report its enlargement in chordoma, schwannoma, arteriovenous malformation, and arteriovenous fistula—lesions in which its enlargement or presence has not been previously reported. It should be noted that the latter 2 are not tru tumors, but rather, vascular lesions.

The anterior meningeal branch of the vertebral artery is extremely small. When visualized in normal patients, its caliber is less than 0.5 mm. in diameter,[4] and subtraction technique is usually necessary for adequate visualization. The Caldwell view is the preferred anteroposterior projection for visualization of this vessel. Enlargement of this vessel is an indicator of posterior fossa extra-axial lesions, and this should be excluded on all vertebral angiograms

The detailed anatomy and roentgen features of the normal anterior meningeal branch of the vertebral artery are adequately described by Newton.[4] Briefly, it arises from the medial aspect of the vertebral artery at the level of C-2, and passes medially through the intervertebral foramen. It then runs superiorly and slightly medially in the spinal canal, following the posterior aspect of the vertebral arteries. It is projected anteriorly to the anterior spinal artery. At the level of the foramen magnum, it ends as small twigs to the dura[1,4] (Fig. 1, *A* and *B*).

Newton visualized the vessel in 24 of 100 selective vertebral arteriograms. He noted that it measured less than 0.5 cm. in diameter, and that, usually, only its proximal 1.0 to 1.5 cm. could be shown.

ILLUSTRATIVE CASES

CHORDOMA

A 30 year old white male presented with a 3 year history of frequent right-sided headaches and a 3 month history of diplopia. Several weeks before admission he developed slurred speech. On admission there was a sixth nerve palsy, decrease in gag reflex, and atrophy of the right half of the tongue. Plain skull film roentgenograms and tomograms demonstrated a nasopharyngeal mass with destruction of the clivus and the right medial border of the petrous ridge. Brain scan was negative. A right carotid arteriogram showed mildly dilated lateral ventricles with no evidence of midline shift. There was evidence of neovascularity from branches of the ascending pharyngeal artery. A vertebral angiography was performed (Fig. 2, *A* and *B*).

CHEMODECTOMA

A 42 year old female had a left carotid body tumor removed 17 years previously. The following year she had a right glomus jugulare tumor removed. This was followed with some palatal and vocal cord difficulty, along with double vision. Eight years later a right carotid body tumor was removed.

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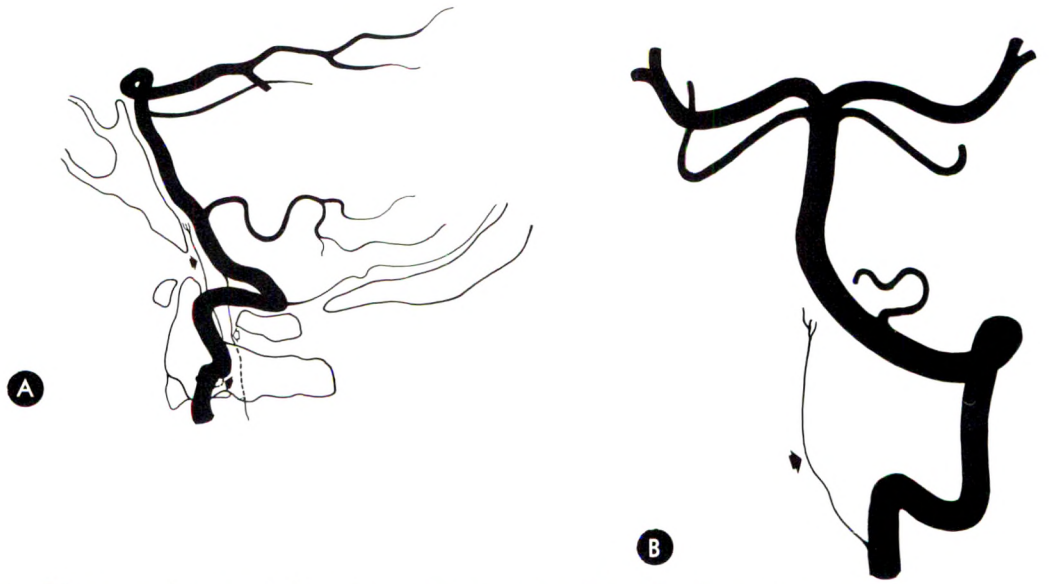


FIG. 1. *Diagrammatic representation of the anterior meningeal branch of the vertebral artery.*
(*A*) Lateral and (*B*) Caldwell projections. The vessel (solid arrow) is projected anterior to the anterior spinal artery (open arrow) in the lateral view.

Five years later she noticed increasingly unsteady gait with light-headedness and frontal and occipital headaches. She had a buzzing and resonating sound in the left ear and had decreased concentration. Swallowing was impaired on the right side, and her speech was noted to worsen. On physical examination the Romberg sign was positive. She favored the right leg, and there was decreased right arm swing. There

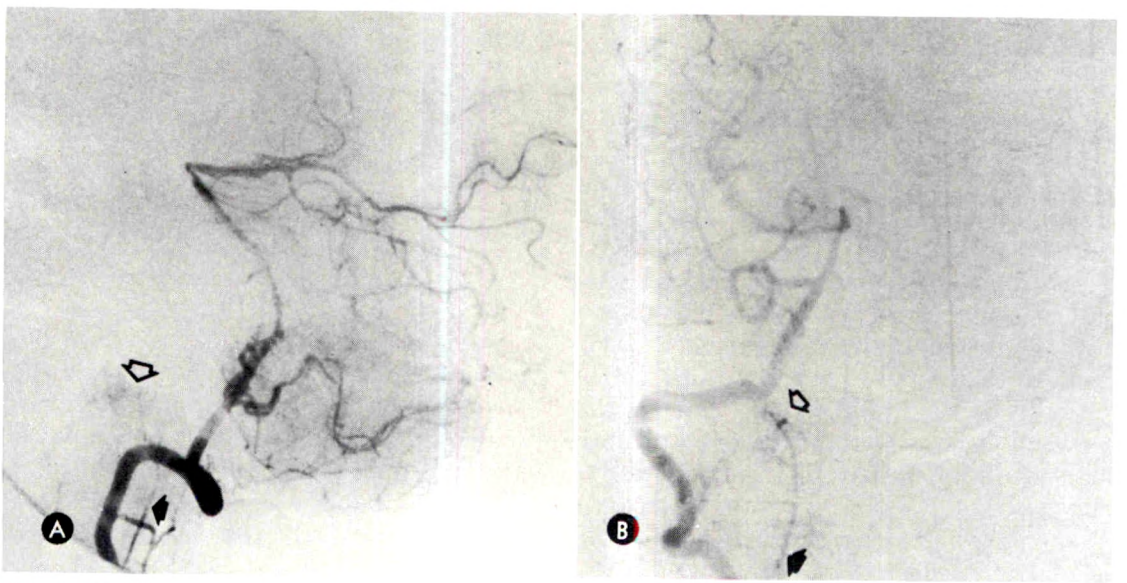


FIG. 2. *Chordoma.*
(*A*) Lateral and (*B*) Caldwell views of right vertebral arteriogram. The anterior meningeal branch (closed arrow) is enlarged in diameter, and supplies a small focus of tumor stain (open arrow). The basilar artery is displaced posteriorly by this largely avascular extra-axial tumor in the clival region.

was a slight facial lag on the right side and no hearing on the right. Decreased gag was observed bilaterally. There was atrophy of the right side of the tongue. Muscle strength was good throughout. Reflexes were equal and active.

A right internal carotid arteriogram revealed a vascular tumor involving the right petrous pyramid. Supplying branches arose from the petrous portion of the right internal carotid artery, and the medial tentorial branch of the cavernous segment. Bilateral vertebral arteriograms were obtained (Fig. 3, *A–D*).

SCHWANNOMA

A 32 year old female had abnormal testing of the cranial nerves IX, X, XI, and XII. A left vertebral arteriogram revealed a mass lesion in the left posterior fossa. During the arteriographic procedures, the patient lost consciousness and experienced respiratory difficulty. She remained acutely ill after this brief episode, and over the next several days it became apparent that she had developed tonsilar herniation. A left suboccipital craniectomy was performed with gross total removal of what was found to be a large schwannoma arising in the region of the tenth nerve. This was reported at surgery to be essentially extradural. It was invested with a stretched covering of dura, but had deeply invaginated the left cerebellar hemisphere. At surgery it was noted that the patient had developed necrosis of both cerebellar tonsils which were herniated to C-2. There was also an area of necrosis in the inferior border of the left cerebellar hemisphere. The roentgenographic findings are shown in Figure 4, *A–C*.

ARTERIOVENOUS MALFORMATION

A 32 year old female began losing vision bilaterally approximately 10 years earlier. At that time arteriography was done which showed an arteriovenous malformation fed mainly by the external carotid arteries. She was treated with ligation of several branches of the external carotid artery bilaterally. Vision did not improve following these ligations. Bilateral common carotid and left vertebral arteriograms demonstrated an extensive arteriovenous malformation (AVM) fed by the internal and external carotid arteries bilaterally, and also by the vertebral arteries posteriorly (Fig. 5, *A* and *B*).

TRAUMATIC ARTERIOVENOUS FISTULA

A 23 year male was admitted to the emergency room following a gunshot wound in which the path of the bullet entered the left neck in the suboccipital area, traveled under the left mastoid tip through the pterygoids and the maxillary sinus, and settled in the left suborbital area.

Bilateral common carotid, selective left internal and external carotid, left vertebral, and aortic arch injections were performed. A traumatic aneurysm of the left internal carotid artery was noted. In addition (Fig. 6, *A* and *B*), an arteriovenous fistula from the left vertebral artery to the occipital and posterior cervical veins, and laceration of the internal maxillary artery immediately posterior to the maxillary sinus were demonstrated.

DISCUSSION

Tumors at the base of the skull usually do not show neovascularity on angiography. Bone tumors with intracranial extension are exceptions to this, and tumor stain may be seen. Chordomas, chondromas, epidermoids, craniopharyngiomas, and pituitary adenomas are usually diagnosed by arterial displacement, except for the occasional pituitary tumor which stains by feeding *via* hypophyseal arteries.[2]

In the chordoma we have illustrated, the anterior meningeal branch of the vertebral artery is supplying the tumor. The artery is enlarged in diameter and ends in neovascularity which supplies a small focus of tumor stain.

A right common carotid arteriogram in this patient showed multiple small vessels coming from branches of the external carotid artery, primarily the ascending pharyn-

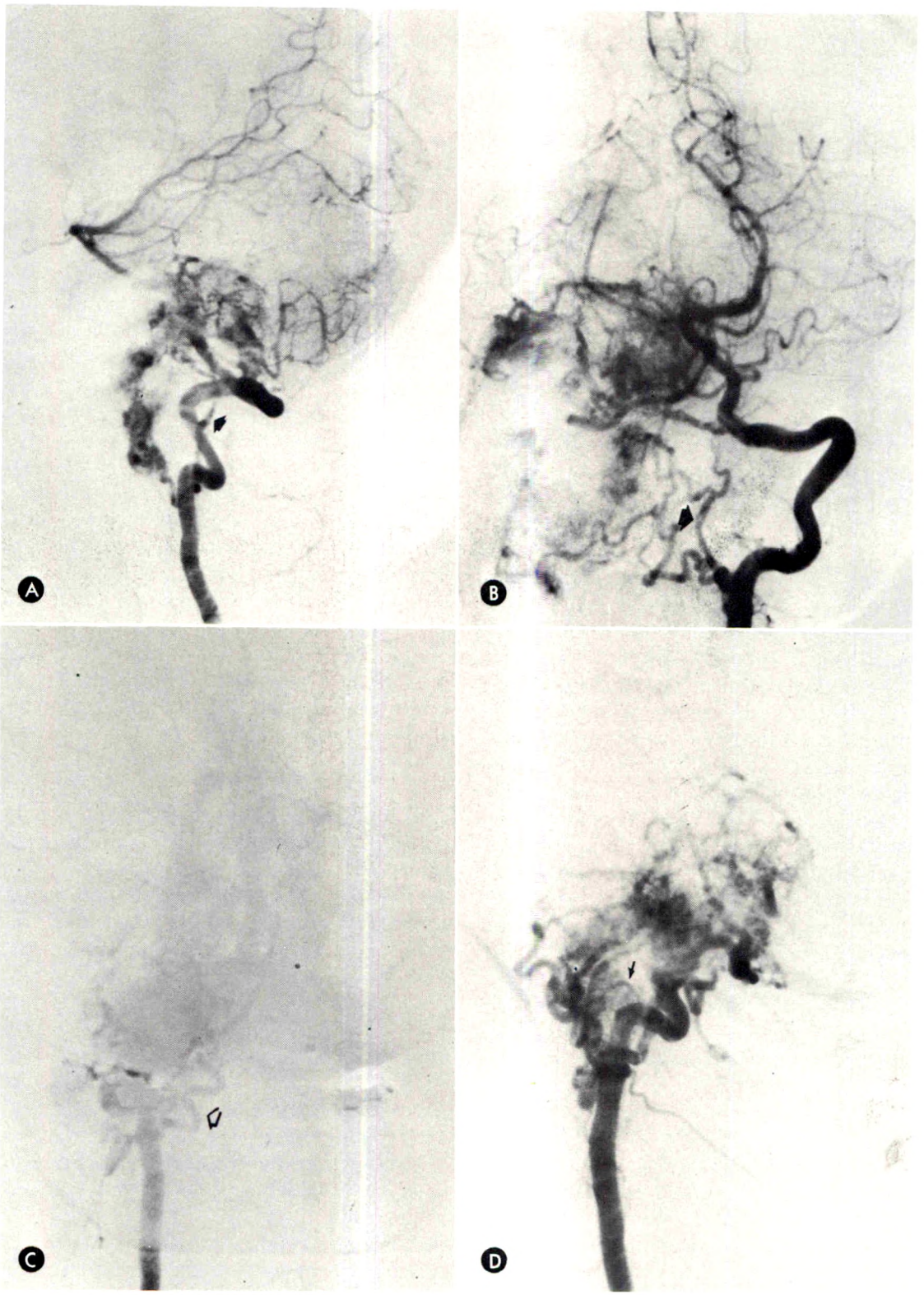
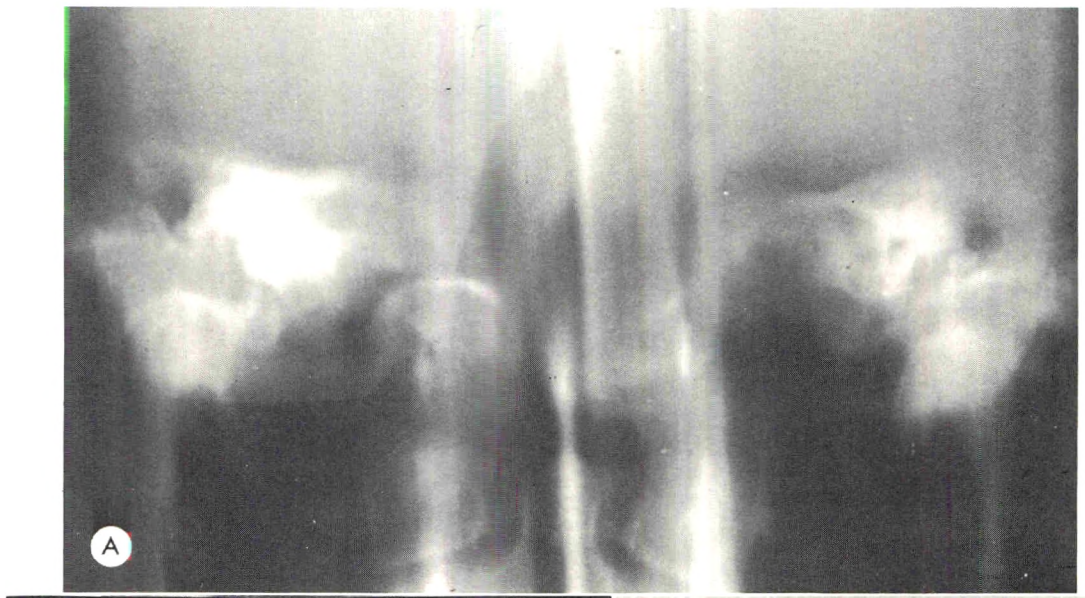
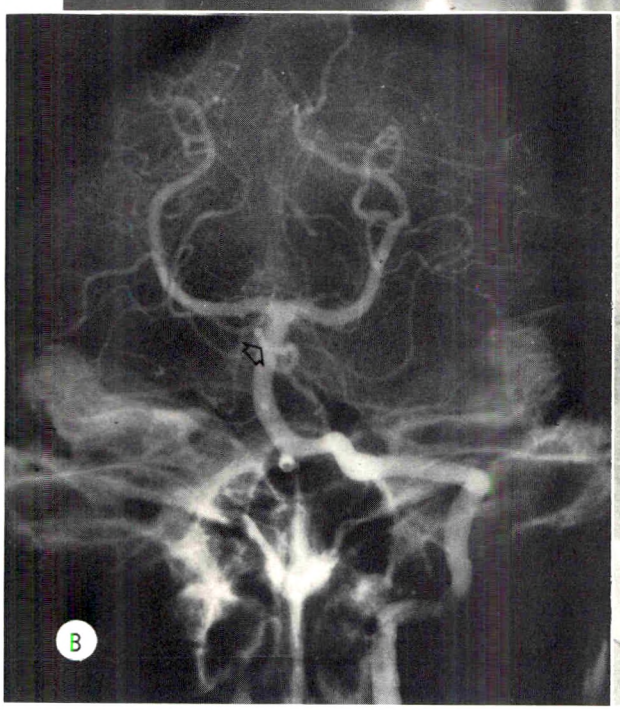

A
B
C
D

FIG. 3. *Chemodectoma.*

(*A–D*) A 43 year old female with multiple intracranial glomus tumors. A chemodectoma in the region of the foramen magnum is partially supplied by the anterior meningeal branches (arrows) of the left (*A* and *B*) and the right (*C* and *D*) vertebral arteries.





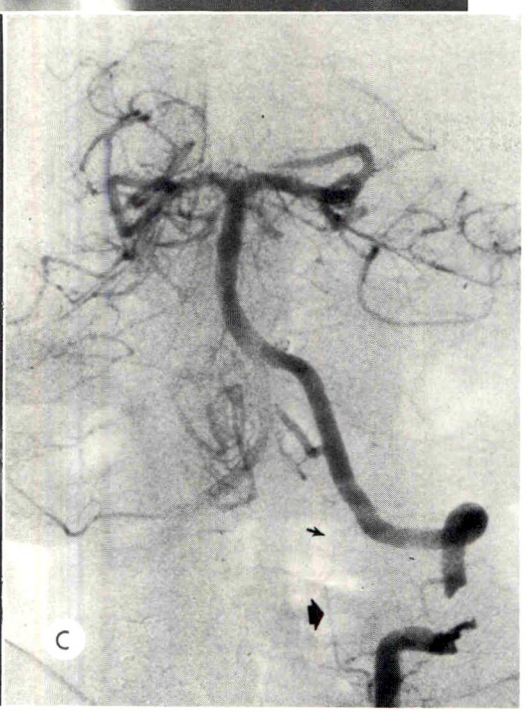


FIG. 4. *Schwannoma.*

(*A*) Anteroposterior tomogram of the petrous bones shows bone erosion of the inferior left petrosal region. (*B*) Towne's projection shows the left PICA displaced toward the right (open arrow). (*C*) The subtracted Caldwell view shows the anterior meningeal branch of the left vertebral artery (closed arrow) ending in an accumulation of tortuous branches (small arrow) at the lower extent of the tumor.

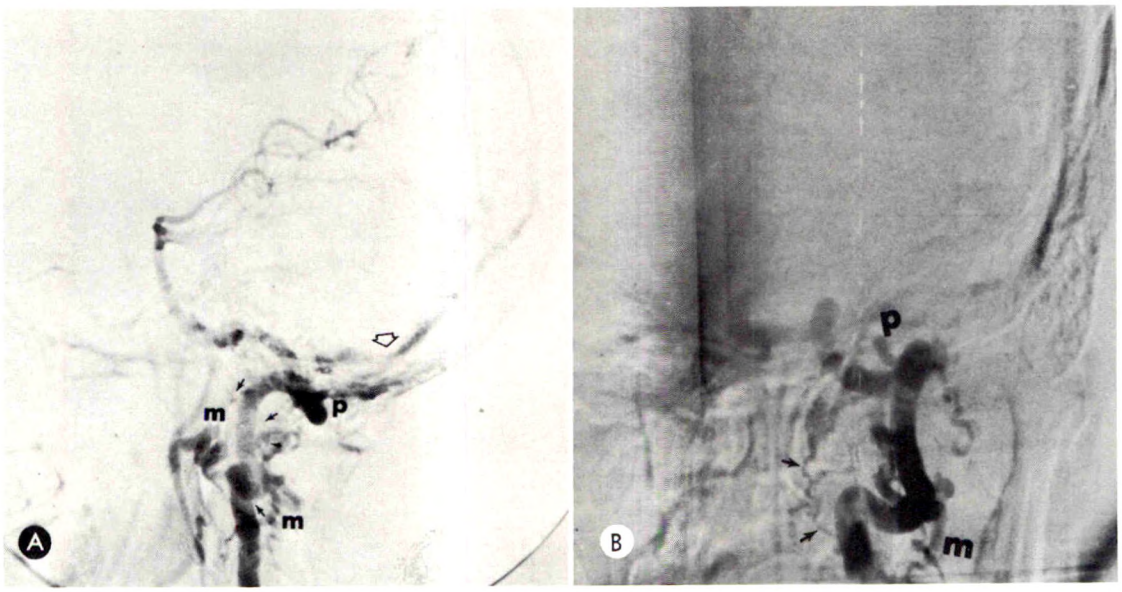


FIG. 5. *Arteriovenous malformation.*

(*A*) Lateral and (*B*) Towne projections of left vertebral arteriogram show a posterior fossa dural AVM (open arrow) fed by both the anterior (small arrow) and posterior (p) meningeal branches of the left vertebral artery. Note the prominence and tortuosity of these vessels, and also the massive enlargement of the muscular branches (m) which also enter the AVM.

geal branches. These fed small areas of tumor stain in the region of the posterior pharynx and nasopharynx. It was noted that the degree of neovascularity was not impressive in view of the size of the soft tissue mass in the posterior pharynx and nasopharynx.

It would appear, therefore, that the growing mass had parasitized the vessels which normally fed the dura at the base of the skull causing them to enlarge in response to blood need. These, then, did end in neovascularity and a tumor stain.

As described by Krayenbühl and Yašargil,[2] a glomus tumor of the cerebellopontine angle may be fed by branches of the external carotid artery, and/or by branches of the basilar artery. These tumors, which arise from paraganglionic structures in the adventitia of the jugular bulb are located in very close proximity to the dura of the floor of the posterior fossa.

Glomus tumors are very vascular and act somewhat like arteriovenous malformations, usually with enlargement of the feeding vessels.[6,7]

According to Palacios,[5] the blood supply of intratympanic and jugular chemodectomas are derived mainly from the carotid artery *via* the tympanic branch of the ascending pharyngeal artery as well as meningeal branches of the internal carotid artery.

The auditory nerve is frequently the sight of solitary schwannoma. The tenth cranial nerve (vagus) may contain a schwannoma as one of many neurofibromas in von Recklinghausen's disease. As the site of a solitary tumor, however, it is extremely rare. It is always extra-axial.[6]

These usually do not show increased vascularity or stain on angiograms. Vascular changes are present, however, which would become apparent with fine focal spot technique, magnification, and subtraction. These changes are due to vascular thrombosis which results in areas of necrosis, and often also results in hemorrhage into adjacent tissues.[6] The normal presentation angiographically is that of displacement of vessels.

In the case illustrated (Fig. 4, *A–C*), a

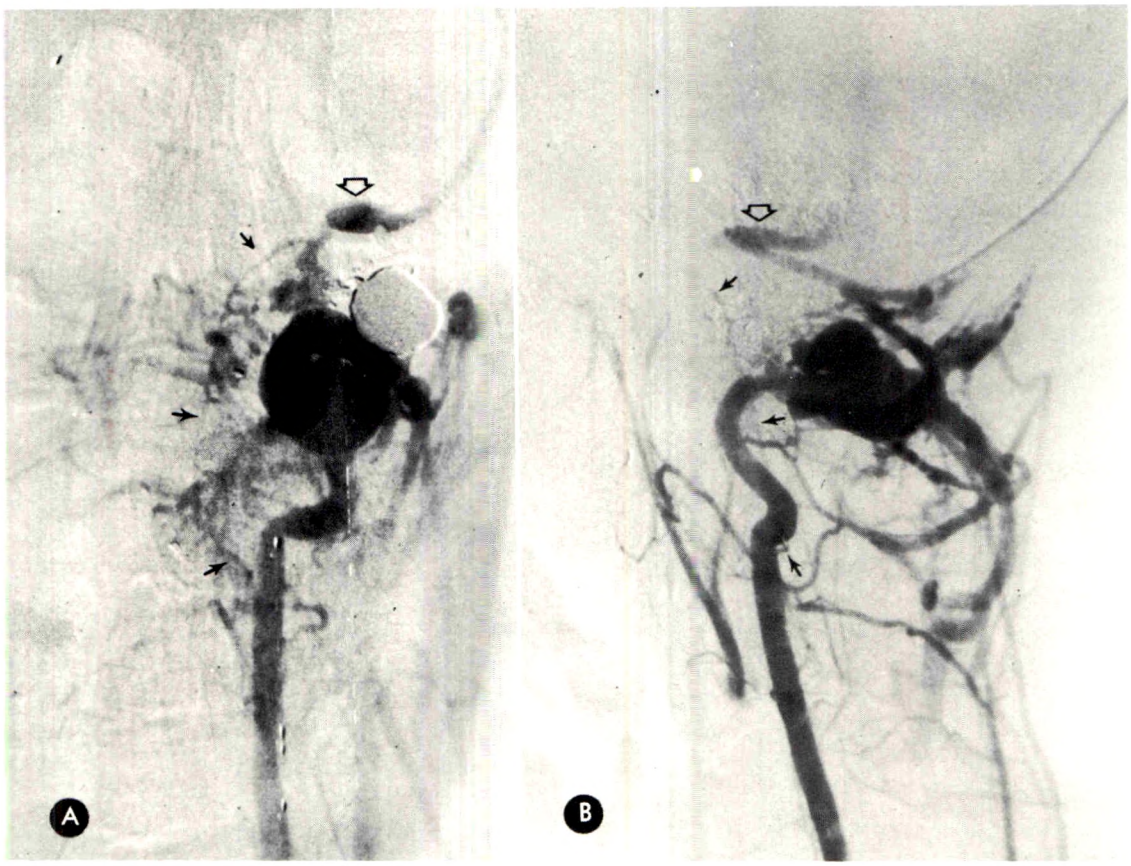


Fig. 6. *Traumatic arteriovenous fistula.*
(*A*) Caldwell and (*B*) lateral projections of left vertebral arteriogram. There was thrombosis of the internal jugular vein. The anterior meningeal branch (small arrow) participates in the shunt, with drainage *via* the neck veins and retrograde into the sigmoid sinus (open arrow).

schwannoma of the Xth cranial nerve not only shows displacement of the left PICA (posterior inferior cerebellar artery), but we also see an increase in vascularity, as evidenced by the accumulation of fine tortuous branches at the distal end of the anterior meningeal branch of the left vertebral artery, at the lower extent of the tumor. The anterior meningeal branch itself is enlarged in diameter.

Infratentorial arteriovenous malformations are sometimes small and well localized, or may extend over the entire cerebellum. Feeding is from the vertebral artery and the branches of the meningohypophyseal trunk. They may also be fed by branches of the external carotid artery.[2] A tortuous vascular mass usually occupies an area in the leptomeninges. The main arteries and veins leading to and from the mass are also enlarged as a rule. The cerebellum itself is rarely affected by major malformations.[6]

Our patient, 10 years previously, had had ligations of branches of both external carotid arteries for multiple arteriovenous malformations which were fed mainly by branches of the external carotid artery. Progressive loss of vision did not improve, and she came in for further work-up and evaluation. Bilateral carotid and left vertebral arteriograms demonstrated extensive arteriovenous malformation fed by the internal and external carotid arteries bilaterally, and also by the left vertebral artery.

The right carotid injection showed the dural AVM fed by a large medial tentorial artery, meningeal branches arising from the ophthalmic artery, and by an anterior falx artery. On the left side, the feeding was similar except for the lack of feeding by meningeal branches of the ophthalmic artery. Multiple collateral branches from the muscular branches of the left vertebral artery reconstituted the occipital branch of the left external carotid artery, which had been ligated previously. The posterior fossa dural AVM was fed by both the anterior and posterior meningeal branches of the vertebral artery, as well as the muscular branches of the vertebral artery. All these vessels were enlarged (Fig. 5, *A* and *B*).

Arteriovenous fistulae are most often due to trauma, and usually involve the intracavernous portion of the internal carotid artery resulting in direct communication between an artery and an adjacent vein. Those involving the vertebral circulation are rare.[7] The left vertebral arteriogram in our patient revealed, in addition to the traumatic aneurysm of the left internal carotid artery, an arteriovenous fistula from the left vertebral artery to the occipital and posterior cervical veins. In addition, there was thrombosis of the internal jugular vein. Venous drainage is *via* the neck veins, and retrograde into the sigmoid sinus. The enlarged meningeal branches of the left vertebral artery participates in the arteriovenous shunting (Fig. 6, *A* and *B*).

As stated by Newton[4] "Selective vertebral catheterization, as well as subtraction techniques, greatly aid in the recognition" of the anterior meningeal branch of the vertebral artery. We have found that the Caldwell view is the best anteroposterior projection for visualization of this vessel.

The anterior meningeal branch of the vertebral artery had been described previously, and its enlargement in posterior fossa extra-axial tumors has been demonstrated. These tumors include glomus jugulare, meningioma, hemangioblastoma, metastatic hypernephroma, and plasmacytoma.[1,3,4] We have shown here enlargement of the anterior meningeal branch of the vertebral artery in chordoma, chemodectoma, schwannoma, arteriovenous malformation, and arteriovenous fistula. We also had 2 patients with enlargement of this artery in meningioma, but since these have been so adequately described previously[3,4] we have not included them in this report.

It should be noted, that enlargement of the anterior meningeal branch of the vertebral artery had been described only in neoplastic lesions, whereas we now see its enlargement in arteriovenous malformation, and in a traumatic arteriovenous fistula. These neoplastic and non-neoplastic lesions therefore considerably widen the spectrum of posterior fossa extra-axial lesions in which this may be seen.

It has become obvious that this vessel may enlarge in many kinds of posterior fossa extra-axial lesions. It was seen in only 24 per cent of normal studies,[4] and when present it is extremely small. Therefore, when the anterior meningeal branch of the vertebral artery is visualized, a careful search for extra-axial lesions should be made.

CONCLUSION

We have demonstrated enlargement and involvement of the anterior meningeal branch of the vertebral artery in nontumor lesions; *i.e.*, arteriovenous malformation, and traumatic arteriovenous fistula.

We have added to its tumor spectrum by demonstrating its feeding of chordomas and Xth nerve schwannoma. We also have shown increased vascularity or stain—a finding not usually seen.

The anterior meningeal branch of the vertebral artery is seen infrequently in normal studies. When present it is extremely small. It has become obvious that this vessel may enlarge in a wide spectrum of posterior fossa extra-axial lesions. Therefore, a careful search for extra-axial lesions should be made when the anterior menin-

geal branch of the vertebral artery is visualized.

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REFERENCES

1. Greitz, T., and Lauren, T. Anterior meningeal branch of vertebral artery. *Acta radiol.* (Diag.), 1968, *7*, 219–224.
2. Krayenbühl, H. A., and Yašargil, M. G. Cerebral Angiography. J. B. Lippincott Company, Philadelphia, 1968.
3. Morris, M. B. Anterior meningeal branch of vertebral artery. *Brit. J. Radiol.*, 1969, *42*, 308–310.
4. Newton, T. H. Anterior and posterior meningeal branches of vertebral artery. *Radiology*, 1968, *91*, 271–279.
5. Palacios E. Chemodectomas of head and neck. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 129–140.
6. Russell, D. S., and Rubenstein, L. J. Pathology of Tumors of Nervous System. Williams & Wilkins, Baltimore, 1972
7. Taveras, J. M., and Wood, E. H. Diagnostic Neuroradiology. Williams & Wilkins Company, Baltimore, 1964.

A PRACTICAL APPROACH TO THE DIAGNOSIS OF CEREBELLOPONTINE ANGLE TUMORS*

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ATLANTA, GEORGIA

PETROUS ridge tomography followed by cerebellopontine angle myelography has been used at Emory University Hospital to diagnose and localize cerebellopontine angle mass lesions. The authors have reviewed 50 consecutive cases to determine the value of each procedure, separately and in conjunction, in the diagnosis of cerebellopontine angle mass lesions.

The purpose of this communication is to report the results of the study and to describe a practical approach to the evaluation of cerebellopontine angle masses.

METHOD AND MATERIAL

In all patients tomograms of each petrous bone were obtained. Cuts of 1 mm. in depth were obtained, using a Philip's polytome with a hypocycloidal motion.

Our technique in cerebellopontine angle myelography is first to obtain scout roentgenograms, using the standard submentovertex and posteroanterior half axial projections with the patient in the lateral decubitus position. The patient is placed in a prone position with the ankles secured to the foot of the roentgenographic table. Using fluoroscopic control a lumbar subarachnoid puncture is performed and 12 cc. of Pantopaque are instilled and manipulated into the cervical subarachnoid space. The patient is then turned into a lateral decubitus position with the side to be examined dependent. The head is lowered until contrast medium fills the dependent cerebellopontine angle. The table is then returned to the horizontal position with the patient's head extended. A cross table submentovertex exposure is made using a grid cassette. The head is then flexed and a cross table posteroanterior half axial exposure is made with the tube angled 30° to the canthomeatal line. Oblique spot roentgenograms are obtained as needed. The opposite cerebellopontine angle may be studied by repeating the procedure just described.

REPORT OF CASES

Case I. L.B., a 67 year old female, presented with dizziness and unsteady gait. Physical examination was essentially noncontributory. Audiometry revealed good vestibular function and auditory discrimination. Petrous ridge tomography demonstrated the right internal auditory canal to be 4 mm. larger than the left. Right cerebellopontine angle myelography showed no evidence of a mass lesion, with good opacification of the internal auditory canal (Fig. 1, *A* and *B*; and 2). The explanation for the enlarged right internal auditory canal was not found, and was thought to be on a congenital basis. The patient is being followed with no progression of symptoms.

Case II. H.C., a 74 year old male, presented with tinnitus, occasional unsteadiness, and a slight gradual hearing loss over a 2 year period. Physical examination revealed left-sided neurosensory hearing loss and an augmented Romberg sign to the right. Petrous ridge tomography was interpreted as normal. Left cerebellopontine angle myelography demonstrated nonfilling of the left internal auditory canal, with a concave filling defect present in the same region, suggesting an acoustic neuroma (Fig. 3, *A* and *B*; and 4). The patient was explored by a translabyrinthine approach with the removal of a 1 cm. acoustic neuroma.

Case III. N.B., a 26 year old male, presented with a noticeable hearing deficit in the right ear for approximately 2 years and difficulty with his equilibrium for the past month. Physical examination showed decreased air and bone conduction on the right. Petrous ridge tomography revealed an unusual bony flaring

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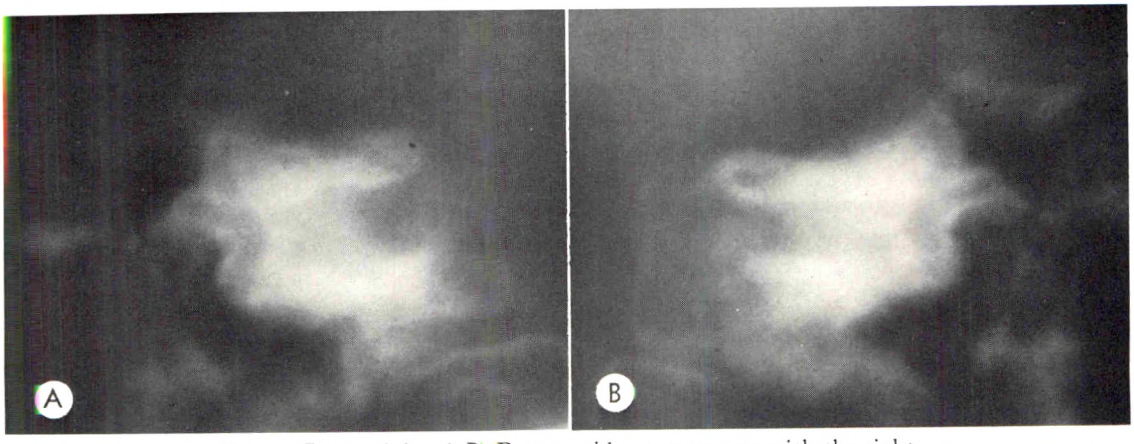


Fig. 1. Case 1. (*A* and *B*) Petrous ridge tomograms with the right internal auditory canal 4 mm. larger than the left.

of the right porus acusticus. Cerebellopontine angle myelography demonstrated nonfilling of the right internal auditory canal. Vertebral angiography revealed a large anterior inferior cerebellar artery with no evidence of neovascularity or displacement (Fig. 5, *A–C*). The patient was explored and a large arterial loop was noted obstructing the internal auditory meatus. No tumor was present.

RESULTS

All of the patients studied presented with one or a combination of the following symptoms: dizziness; vertigo; unilateral retrocochlear hearing loss; and unsteady gait. In 62 per cent of the total cases cerebellopontine angle myelography and petrous ridge tomography were both negative (Table 1). In 16 per cent of the cases, cerebellopontine angle myelography and petrous ridge tomography were both positive. Ten per cent of the patients had negative cerebellopontine angle myelograms but positive petrous ridge tomograms. In 12 per cent of the patients studied the cerebellopontine angle myelograms were positive in spite of negative petrous ridge tomograms.

All 14 patients who had a positive finding on cerebellopontine angle myelography included retrocochlear hearing loss as one of their symptoms. Of these patients 8 had acoustic neuromas (Table II). The cerebellopontine angle myelograms of each of these patients demonstrated a mass in the Pantopaque-filled angle as well as nonfilling of the internal auditory canal. No acoustic neuromas were confined solely to the internal auditory canal. Baker's[1] experience has been similar. Of the other 6 patients,

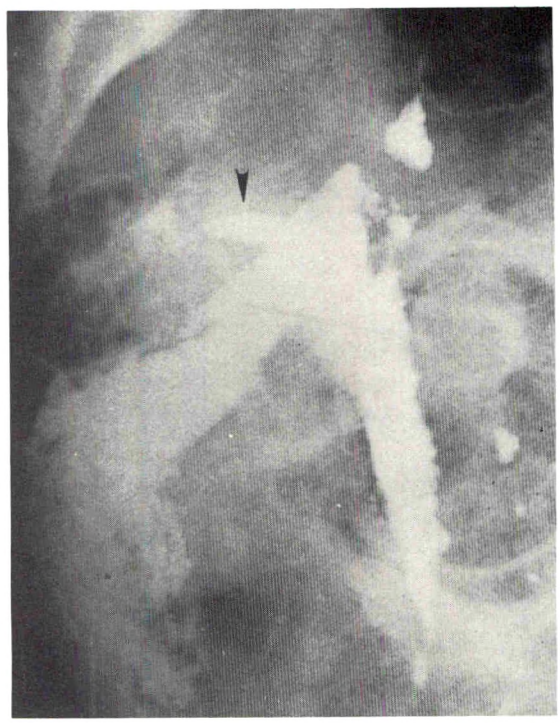


Fig. 2. Case 1. Submentovertex view of a right cerebellopontine angle myelogram demonstrating filling of the right internal auditory canal (arrow) with no evidence of a mass.

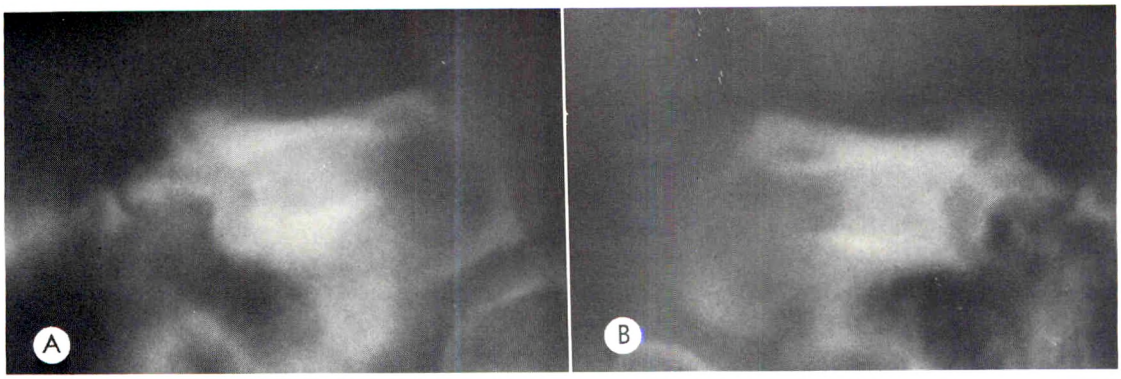


FIG. 3. Case II. (*A* and *B*) Normal internal auditory canals.

2 were found at exploration to have congenitally small internal auditory canals, 1 patient (Case III) had an enlarged anterior inferior cerebellar artery obstructing the canal, 1 patient had a meningioma, 1 patient had an exophytic pontine glioma, and 1 patient had an adenocarcinoma of the nasopharynx with extension of the mass into the petrous ridge. These last 3 patients had evidence of a mass as well as nonfilling of the porus acusticus. Five patients who had positive findings on petrous ridge tomography but negative cerebellopontine angle myelography are currently being followed with no evidence of tumor.

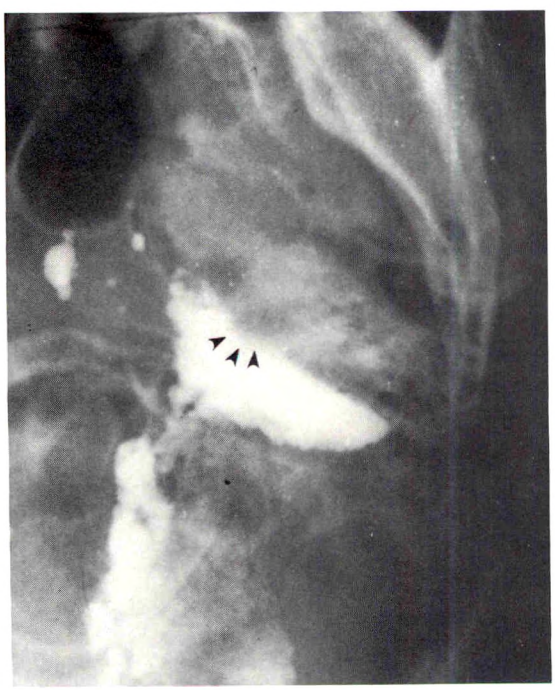


FIG. 4. Case II. There is nonfilling of the internal auditory canal on the left with a mass defect clearly present (arrows).

DISCUSSION

The anatomy of the posterior fossa as demonstrated by positive contrast myelography has been discussed by several authors.[3,4,7] Unlike Pribram[5] and Siew *et al.*,[6] who prefer air encephalography, we believe that Pantopaque cisternography is the procedure of choice for examining the cerebellopontine angle, especially in patients presenting with unilateral retrocochlear hearing loss. Routine plain skull roentgenograms alone are not adequate to evaluate the petrous ridges because of the superimposition of other structures. Petrous ridge tomography alone is a useful screening procedure in patients presenting without unilateral retrocochlear hearing loss. If this study proves negative, we believe these patients should be followed clinically and a positive contrast medium study of the posterior fossa delayed until hearing loss develops.

In patients presenting with unilateral retrocochlear hearing loss, we feel that the first roentgenographic study should be Pantopaque cerebellopontine angle myelography. If the cerebellopontine angle myelogram is normal with good filling of the internal auditory canals demonstrated, no further neuroroentgenologic work-up is needed. In these patients petrous ridge

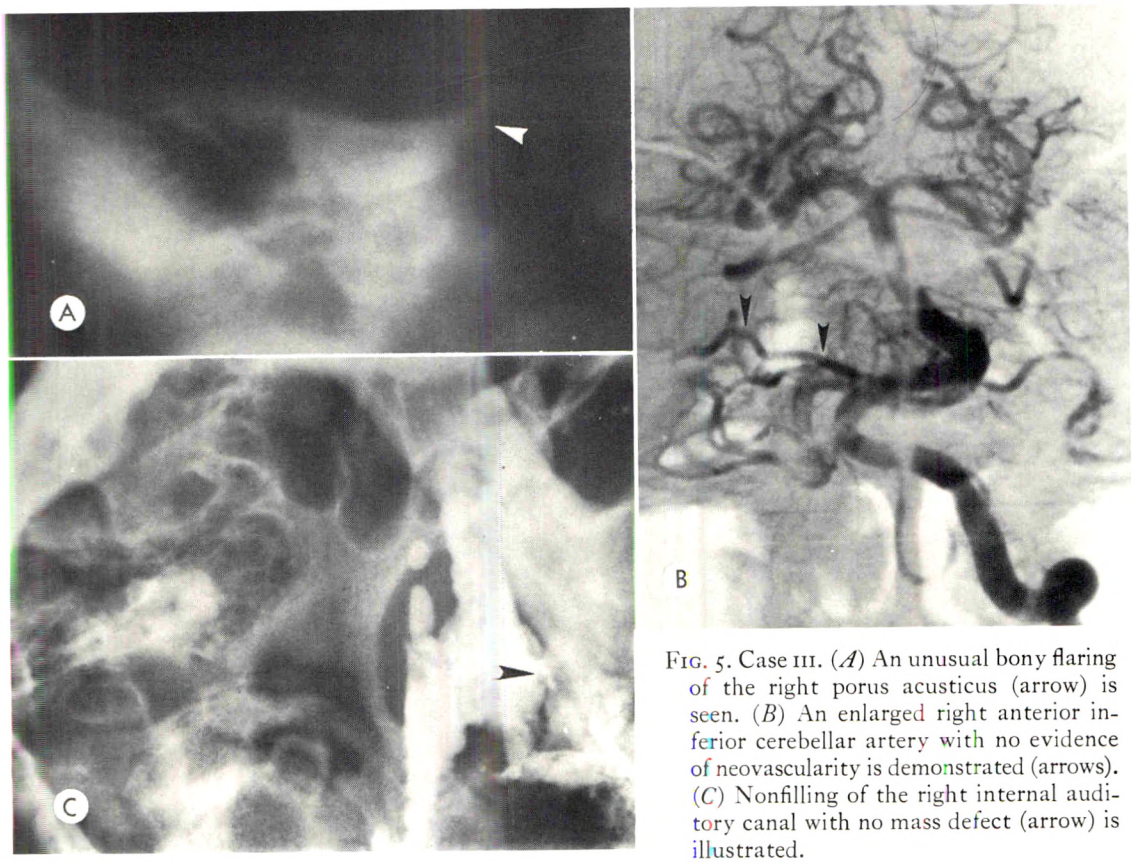


Fig. 5. Case III. (*A*) An unusual bony flaring of the right porus acusticus (arrow) is seen. (*B*) An enlarged right anterior inferior cerebellar artery with no evidence of neovascularity is demonstrated (arrows). (*C*) Nonfilling of the right internal auditory canal with no mass defect (arrow) is illustrated.

tomography with its attendant irradiation of as great as 10 rads in the unshielded patient[2] may be avoided. If the cerebellopontine angle myelogram shows nonfilling of the internal auditory canal, then by better defining the bone changes, petrous ridge tomography is helpful in delineating the lesion. The definitive procedure, in our experience, is cerebellopontine angle myelography, as 2 of our patients with acoustic neuromas had negative petrous ridge tomograms, and none of the 5 patients with a positive petrous ridge tomogram and normal cerebellopontine angle myelogram have had a progression of symptoms, as might be expected if a tumor were present.

Nonfilling of the internal auditory canal on Pantopaque myelography is not sufficient as an isolated finding to indicate the presence of a cerebellopontine angle mass. A filling defect in the cerebellopontine angle must be demonstrated, as there are other causes for nonfilling of the internal auditory canal beside tumor. Nonfilling may result from congenitally small canals, occlusion by vascular structures, or arachnoid adhesions. In our series nonfilling of the internal auditory canal occurred in the absence of tumor in 3 cases. Two of these

Table I

COMPARATIVE RESULTS OF CEREBELLOPONTINE ANGLE MYELOGRAPHY AND PETROUS RIDGE TOMOGRAPHY

Results of Studies		No. of Cases	Percentage of Cases
CPAM −	PRT −	31	62
+	+	8	16
−	+	5	10
+	−	6	12

CPAM = cerebellopontine angle myelography.
PRT = petrous ridge tomography.

TABLE II

CLINICAL FINDINGS AND PATHOLOGY IN PATIENTS WITH POSITIVE CEREBELLOPONTINE ANGLE MYELOGRAPHY

Patient	Symptoms	Results of Studies		Abnormality/Lesion
		CPAM	PRT	
A.G.	Headaches, right hearing loss	+	+	Acoustic neuroma
H.C.	Tinnitus, left hearing loss	+	−	Acoustic neuroma
K.P.	Right hearing loss	+	+	Acoustic neuroma
J.W.	Tinnitus, left hearing loss	+	+	Acoustic neuroma
R.H.	Left hearing loss	+	+	Acoustic neuroma
W.S.	Tinnitus, right hearing loss	+	+	Acoustic neuroma
L.T.	Ataxia, right hearing loss	+	−	Acoustic neuroma
V.B.	Left hearing loss	+	+	Acoustic neuroma
M.O.	Headaches, right hearing loss	+	+	Congenitally small right internal auditory canal
R.G.	Sudden right hearing loss	+	−	Meningioma of right cerebellopontine angle
G.L.	Ataxia, left hearing loss	+	−	Exophytic pontine glioma
H.Wh.	Dizziness, left hearing loss	+	−	Congenital stenosis of left internal auditory canal
N.B.	Dizziness, right hearing loss	+	+	Large loop AICA filling right internal auditory canal
H.We.	Pain left ear and face	+	−	Metastatic adenocarcinoma from nasopharynx

CPAM = cerebellopontine angle myelography.
PRT = petrous ridge tomography.
AICA = anterior inferior cerebellar artery.

had congenitally small canals and the other had a vascular compromise. We believe that unless a definitive concave defect is seen in the Pantopaque column, exploratory surgery is not indicated, and the cerebellopontine angle myelogram should be repeated in 3 to 6 months.

In conclusion, petrous ridge tomography is a useful screening procedure in patients presenting with vertigo and without evidence of retrocochlear hearing loss. If the tomograms show no abnormality, these patients may be followed. In patients presenting with unilateral hearing loss, Pantopaque cerebellopontine angle myelography is the procedure of choice and should be the initial examination. If the internal auditory canal fills, no further roentgenographic study is needed.

SUMMARY

The authors have reviewed petrous ridge tomography and cerebellopontine angle myelography in 50 patients presenting with various symptoms including dizziness, vertigo, unsteady gait, and unilateral hearing loss, in order to ascertain the value of each procedure and to develop a practical approach in diagnosing cerebellopontine angle mass lesions.

Petrous ridge tomography is a useful screening procedure which should be used judiciously, since it involves significant irradiation to the eye.

In patients with unilateral retrocochlear hearing loss, Pantopaque cerebellopontine angle myelography, which is the definitive procedure, should be performed first.

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REFERENCES

1. Baker, H. L., Jr. Cerebellopontine angle myelography. *J. Neurosurg.*, 1972, *36*, 614–624.

2. Chin, F. K., Anderson, W. B., and Gilbertson, J. D. Radiation dose to critical organs during petrous ridge tomography. *Radiology*, 1970, *94*, 623–627.
3. Gold, L., and Kieffer, S. Positive contrast evaluation of posterior cranial fossa. *Radiology*, 1972, *102*, 63–70.
4. Hastings, J. R. Anatomy of posterior fossa in relation to positive contrast cisternography. *Radiology*, 1969, *92*, 1065–1072.
5. Pribram, H. F. W. Differentiation of extrinsic from intrinsic intracranial tumors with particular references to posterior fossa tumors. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 542–549.
6. Siew, F. P., Kricheff, I. I., and Chase, N. E. Demonstration of small acoustic neuromas using negative contrast medium with tomography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *91*, 764–769.
7. Valvassori, G. E. Myelography of internal auditory canal. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 578–586.

A COMBINED TECHNIQUE FOR INVESTIGATION OF THE CEREBELLO-PONTINE ANGLE CISTERN AND INTERNAL AUDITORY CANAL*

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ADVANCES in neurosurgical and otologic techniques, especially the development of the translabyrinthine approach with the use of the dissecting microscope,[7] have made possible the uncomplicated removal of even the smallest lesions in the internal auditory canal or cerebello-pontine angle cistern. Included in this category are those lesions confined to the internal auditory canal which may only be adequately demonstrated or excluded by positive contrast cisternography. This technique has been used for many years and is described by many workers.[1,2,4–6,10,13,14,17] Shafron and Wiener,[14] and Burrows[4] had used tomography during positive contrast cisternography and Dorland *et al.*[5] also mentioned the use of tomography. However, it was Valvassori,[16] who first fully described the findings with a technique using positive contrast cisternography and tomography.

Small lesions can be demonstrated by gas cisternography,[8,15] but these lesions must be projecting, at least in part, into the cerebello-pontine angle cistern in order to show a defect in the gas-filled cistern.

These basic approaches to the evaluation of these lesions each have their protagonists. Other workers have attempted to tailor the investigation to the suspected size of the lesion. Wende and Lüdecke[18] discussed both gas and positive contrast cisternography and concluded that the former was more useful if the clinical features suggested a large lesion, and the latter should be used for small lesions. Similarly, Bull[3] recommended pneumoencephalography for large tumors and positive contrast cisternography for small tumors.

With the possible exception of very large tumors, it is often extremely difficult, on clinical grounds, to predict accurately the size of the lesion. We are also of the opinion that lesions projecting into the cerebello-pontine angle cistern can be demonstrated most adequately by gas cisternography with tomography. Positive contrast cisternography will demonstrate these lesions but may require the use of large amounts of contrast material[2,9] and this in itself, because of the radiodensity of the contrast material, may obscure part of the lesion or make evaluation of the internal auditory canal more difficult. In addition, it may not be possible to assess changes in the brain stem, and it is certainly not possible to demonstrate the position, size and shape of the ventricular system using positive contrast medium only.

METHOD

Gas cisternography is carried out as previously described by Morris and Wylie[11] and includes a complete examination of the ventricular system and basal cisterns.

POSITIVE CONTRAST CISTERNOGRAPHY

The examination is usually performed under basal sedation (Valium and Fortral) unless it follows immediately on the pneumoencephalography, in which case the patient is under general anesthesia.

The patient is placed in the Isocentric chair in a sitting position (Fig. 1) and the chair is adjusted so that the external auditory meatus is at the Isocentric point.

An amount of 1.5–2 ml. of positive contrast agent is injected after lumbar puncture. The chair is rotated 90° around the Isocentric point so that the spine of the

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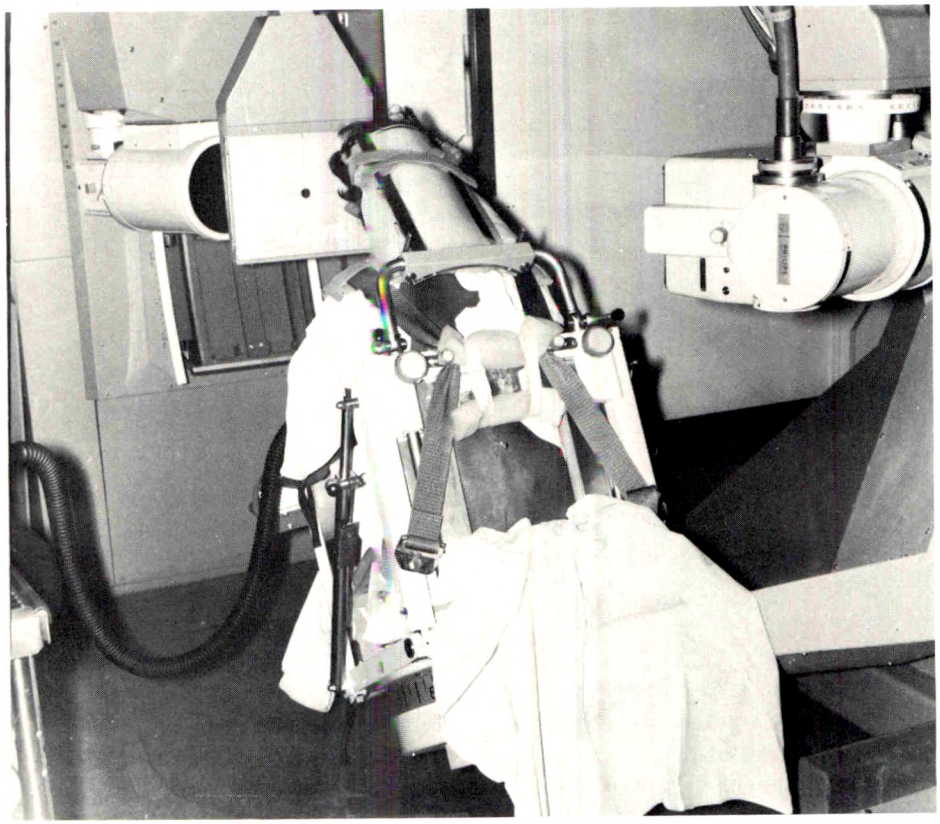


Fig. 1. Erect position for coronal tomography of the cerebello-pontine angle cistern and brain stem.

patient is horizontal and the chair is then rotated around its base about 50° so that the side under investigation is dependent (Fig. 2). If the examination follows a pneumoencephalography, the patient can be placed in this position prior to contrast medium injection, the needle being *in situ*. The main arm of the Diagnost N (Philips) is then adjusted so that the beam is horizontal and under screen control is centered to the relevant area. The base of the spine is elevated so that the head becomes dependent and the transfer of contrast medium is monitored under screen control so that it can be seen to enter the basal cisterns (Fig. 3). Minor adjustments in chair and tube movement can be made at this stage to visualize the upper cervical spine, skull base and petrous bone regions.

Once the contrast medium is seen to come to lie over the back face of the petrous bone in the region of the porus acusticus and to outline the arcuate eminence of the petrous bone, the chair can be rotated so that the patient is in a more horizontal position. This prevents any supratentorial spill of contrast material, while retaining contrast material in the region of the porus acusticus. A normal internal auditory canal will be seen to fill at this stage. The position is similar to that shown in Figure 2. Further adjustments under screen control can then be made by rotating the chair around its base so that the internal auditory canal is seen to best advantage. We have found that the best visualization is with the sagittal plane at about an angle of 20° to the horizontal. The full decubitus position (*i.e.* with the sagittal plane horizontal) allows contrast material to run laterally away from the region of the porus acusticus. The level of tomographic sec-

FIG. 3. The base of the spine is elevated to allow the contrast medium to run into the right cerebello-pontine angle cistern.

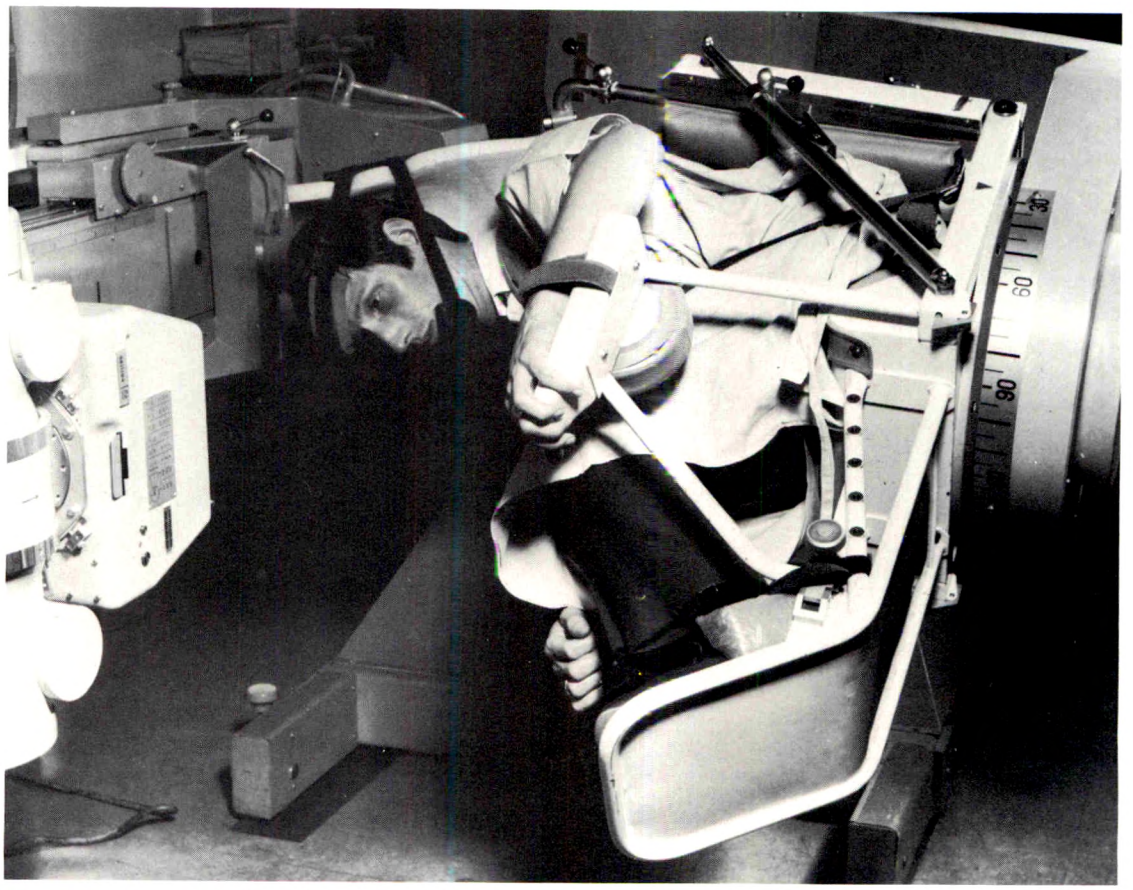


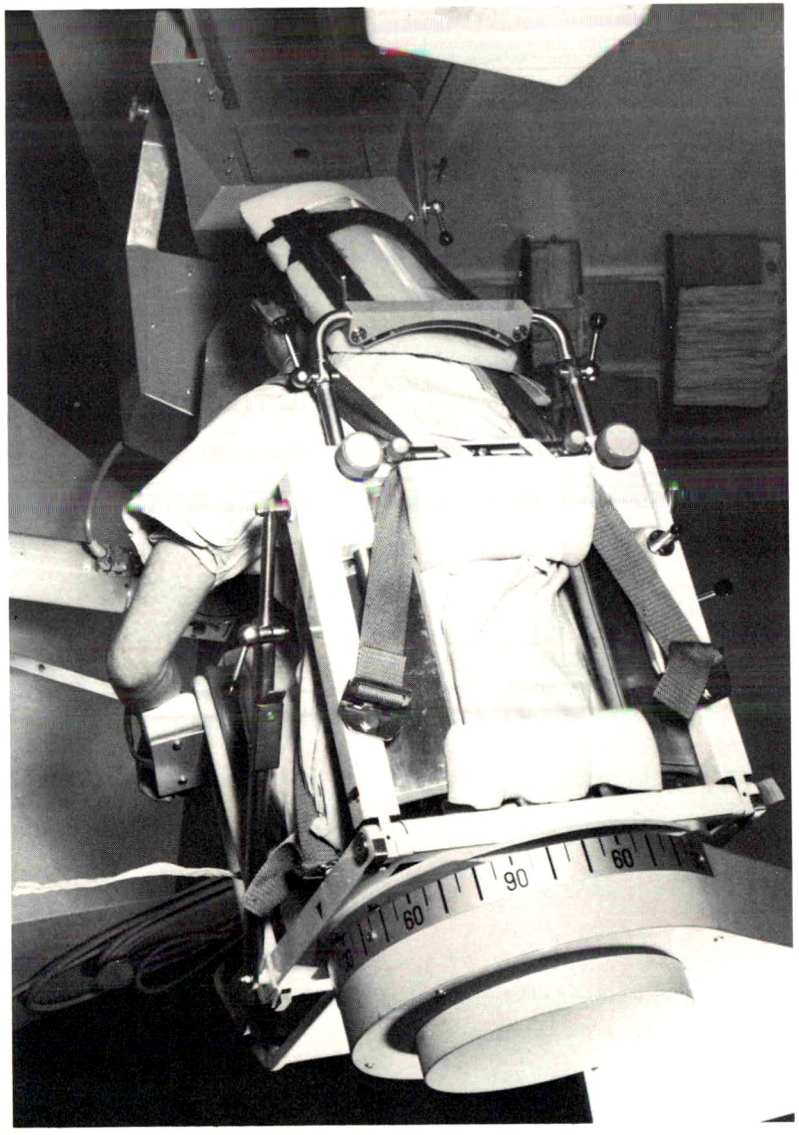
FIG. 2. Horizontal position after the injection of positive contrast medium; the patient is rotated so that the side to be examined (right) is dependent. X-ray beam is horizontal.

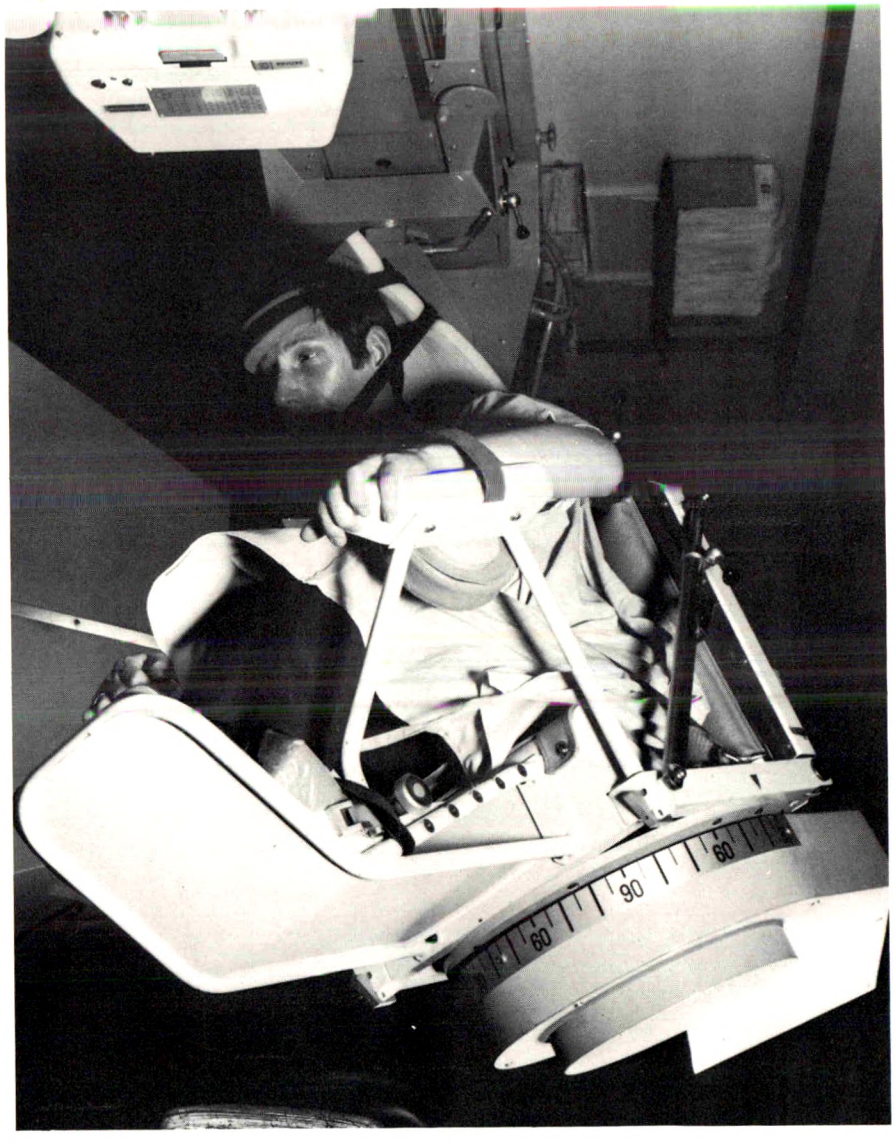
tion is then selected (about midway from the outer canthus to the external auditory meatus on the side being examined) and tomography is commenced. This position is, in fact, a "cross-table frontal oblique projection." Once the area of interest has been localized, tomographic sections at 0.25 mm. spacings are obtained.

If it is desirable to examine the opposite side, the chair can be returned to the vertical position to empty the cerebello-pontine angle cistern and the procedure can be repeated with the opposite side dependent (Fig. 4).

Results have shown that it has been possible in practically all cases to obtain an adequate examination of the angle cistern. Occasionally, difficulties have arisen due to

FIG. 4. The patient is rotated so that the left side is dependent. X-ray tube in position for tomography.


60
90
60


60
90
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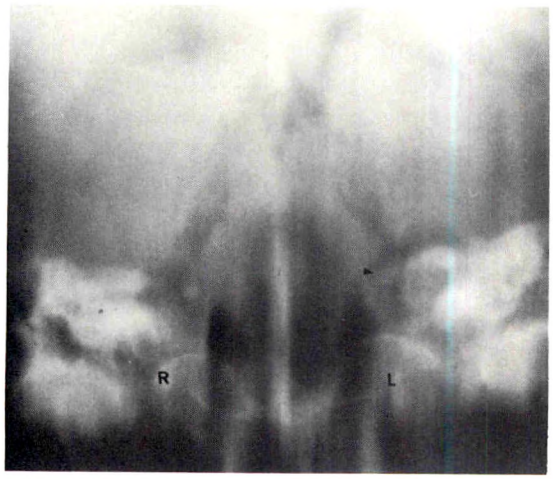


FIG. 5. Small left acoustic nerve tumor projecting just beyond the porus. The internal auditory canal is widened. The tumor displaces the trigeminal nerve (arrowhead).

marked spinal deformity, *i.e.*, kyphosis or scoliosis, in which case it may be difficult to transfer the contrast material from the sacral to the upper cervical regions. In these instances, the patient is placed in the true decubitus position initially, and this aids the transfer of contrast material in a cranial direction.

DISCUSSION

The method described has the basic advantage of flexibility. As stated, we consider that gas cisternography is the best method to demonstrate those lesions projecting into the cerebello-pontine angle cistern. This is so even with small lesions (Fig. 5; and 6), and even in these there is the added advantage that the relationship of the lesion to the trigeminal nerve can be demonstrated. Larger lesions impinging on the brain stem, with or without clinical evidence of this (Fig. 7; and 8), are clearly outlined. Where the lesion is very large, *i.e.*, deforming the brain stem and fourth ventricle, the effects of the lesion on these structures can be seen (Fig. 9; and 10, *A*

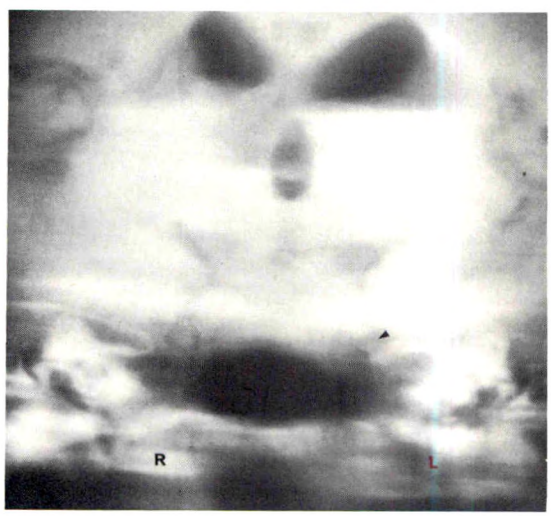


FIG. 6. Small left acoustic nerve tumor projecting just beyond the porus. The internal auditory canal is widened. The tumor slightly displaces the trigeminal nerve (arrowhead).

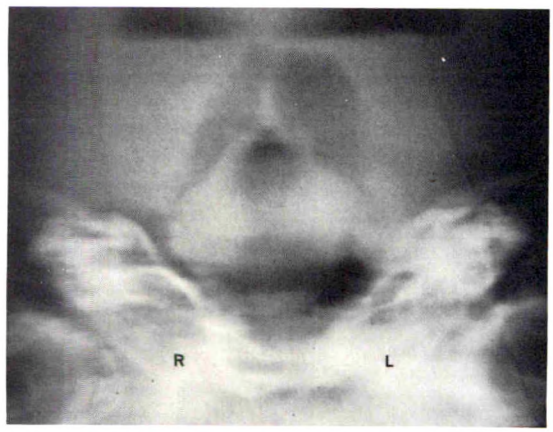


FIG. 7. Medium-sized left acoustic nerve tumor impinging on cerebello-pontine angle cistern and flattening the left side of the brain stem.

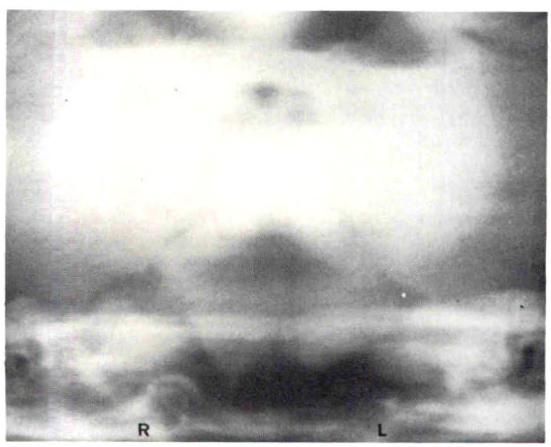


FIG. 8. Medium-sized right acoustic nerve tumor impinging on cerebello-pontine angle cistern and flattening the right side of the brain stem.

and *B*). With earlier clinical suspicion of these lesions, we have seen relatively few tumors of this large size. In these cases, therefore, gas cisternography completely fulfills the basic requirements after demonstrating the size and shape of the lesion and its effect on related structures. The true size of that part of the lesion extending beyond the internal auditory canal can be measured as the apparatus used has a constant magnification factor.

In those cases where the pneumoencephalography fails to show a lesion, or where positive contrast cisternography is the first examination, this latter form of investigation will allow us to determine whether or not a lesion is present. A normal meatogram (Fig. 11) will positively exclude a lesion. A positive contrast meatogram, either after pneumoencephalography (Fig. 12, *A* and *B*; and 13, *A* and *B*) or conducted as a primary procedure (Fig. 14,

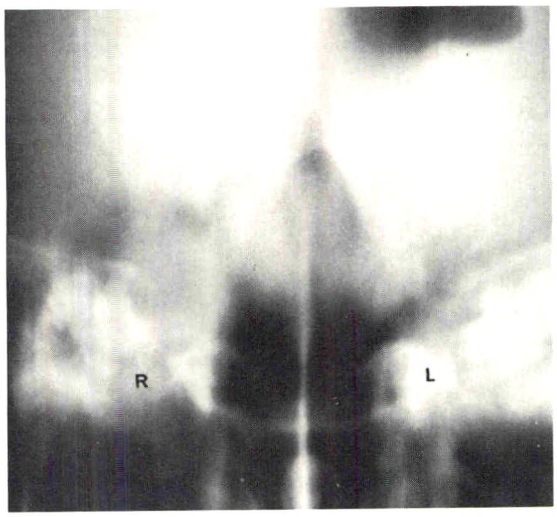
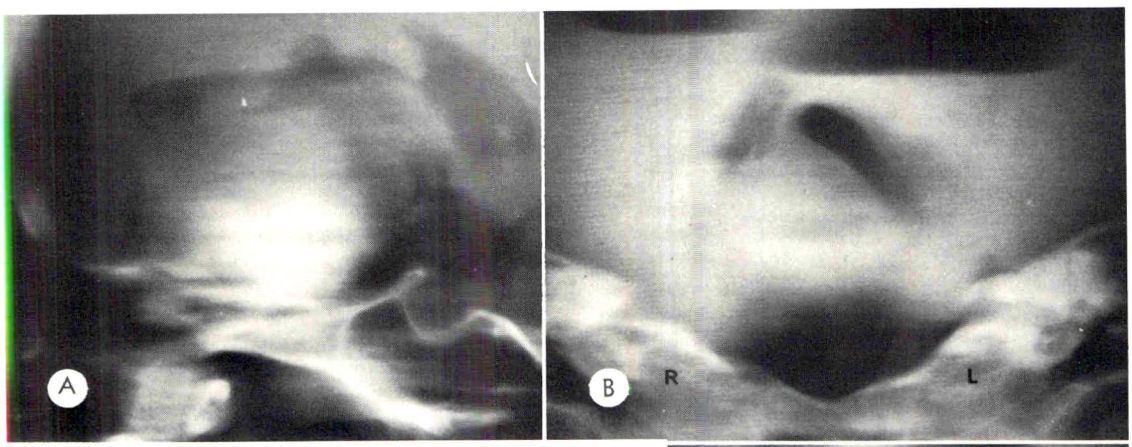


Fig. 9. Large right acoustic nerve tumor. The brain stem is flattened and rotated and the fourth ventricle is flattened on its right side.



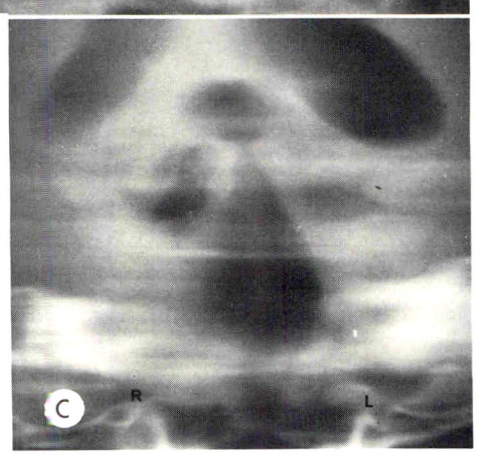


Fig. 10. Massive right acoustic nerve tumor. (*A*) Lateral tomogram showing deformity and rotation of fourth ventricle. (*B*) Straight posteroanterior tomogram showing elevation of floor of the fourth ventricle on the right side. (*C*) Angled posteroanterior tomogram showing displacement of the body of the fourth ventricle to the left.



FIG. 11. Tomogram through the contrast-filled internal auditory canal. Normal appearances.

A and *B*) will prove the presence of a lesion, and this type of case illustrates the fact that no procedure other than positive contrast cisternography could possibly have demonstrated the lesion. Difficulty in diagnosis may arise where some contrast material enters but does not completely fill the canal. Valvassori[17] has dealt with this subject, but we have found that relatively minor abnormalities (Fig. 15, *A* and *B*; 16, *A* and *B*; and 17, *A* and *B*) may be impossible to assess, and in these cases we think that it is probably preferable to wait for a short time (3–6 months) and repeat the examination before attempting to reach a definitive conclusion. This policy is borne out by the fact that the smallest lesion which we have found and which has been surgically confirmed, 0.5 cm. (Fig. 12, *A* and *B*) caused complete obstruction to the canal, and we feel that it may be unwise to attempt to diagnose with certainty lesions smaller than this. The findings of Baker[2] of 10 false positive "non-tumors" due to a small canal filled by normal structures, adhesions, or tufts of arachnoid, support this view.

A practical problem is the choice of the first examination. Gas cisternography followed by positive contrast cisternography is a feasible procedure, but pneumoencephalography requires a general anesthetic and is more disturbing to the patient. One may, therefore, wish to avoid pneumoencephalography if this is possible. If positive contrast cisternography is employed as a primary procedure and the lesion is found to be too large to be adequately demonstrated by a small amount of contrast material, a pneumoencephalogram would become necessary. In this situation an anesthetic would have to be administered in the middle of the procedure, or the pneumoencephalography would have to be deferred. Also, it may not always be possible

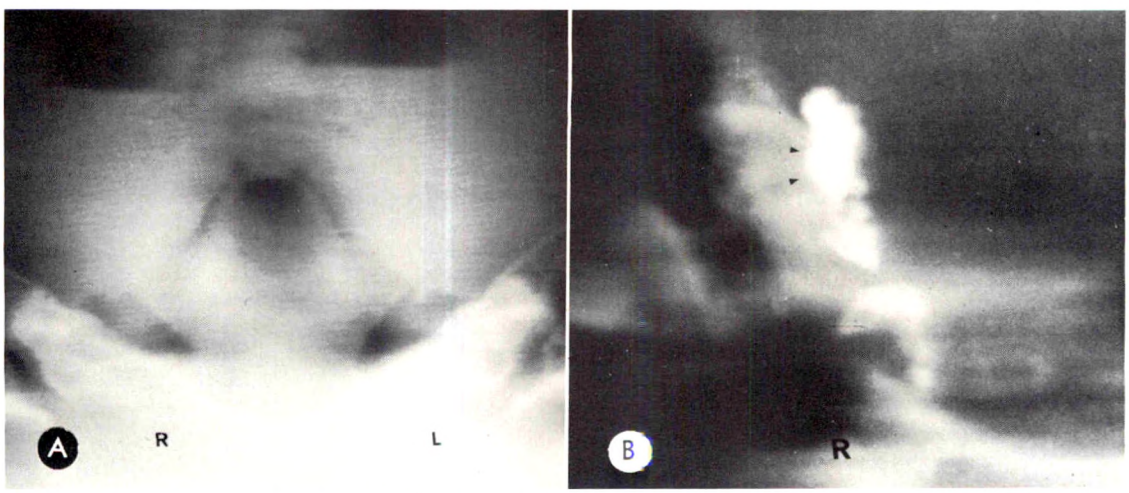


FIG. 12. Intracanalicular tumor. (*A*) Pneumoencephalogram. No abnormality is seen in either cerebellopontine angle cistern. (*B*) Positive contrast cisternogram. The internal auditory canal is blocked at the porus. A 0.5 cm. tumor was present. Histologically, a small vascular malformation was demonstrated.

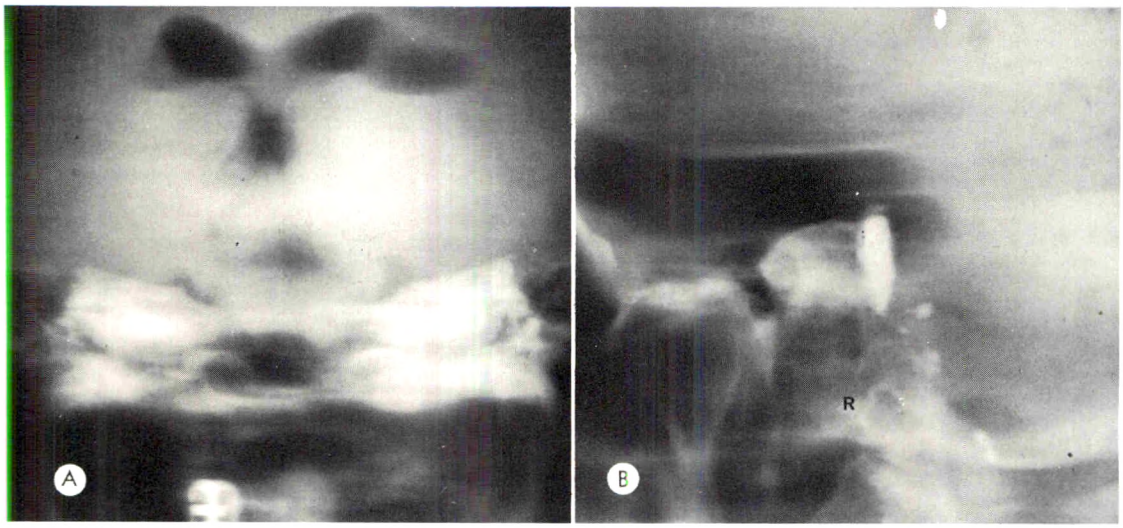


FIG. 13. Intracanalicular acoustic nerve tumor. (*A*) Normal cerebello-pontine angle cistern at pneumoencephalography. (*B*) Positive contrast cisternogram. The internal auditory canal is blocked at the porus.

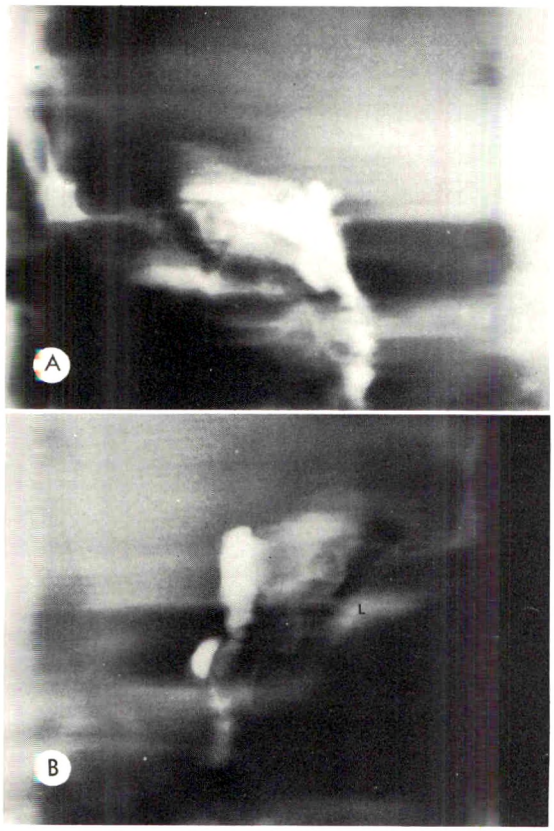


FIG. 14. Positive contrast cisternograms. (*A*) The right internal auditory canal is normal. (*B*) The left canal is blocked at the porus by acoustic nerve tumor.

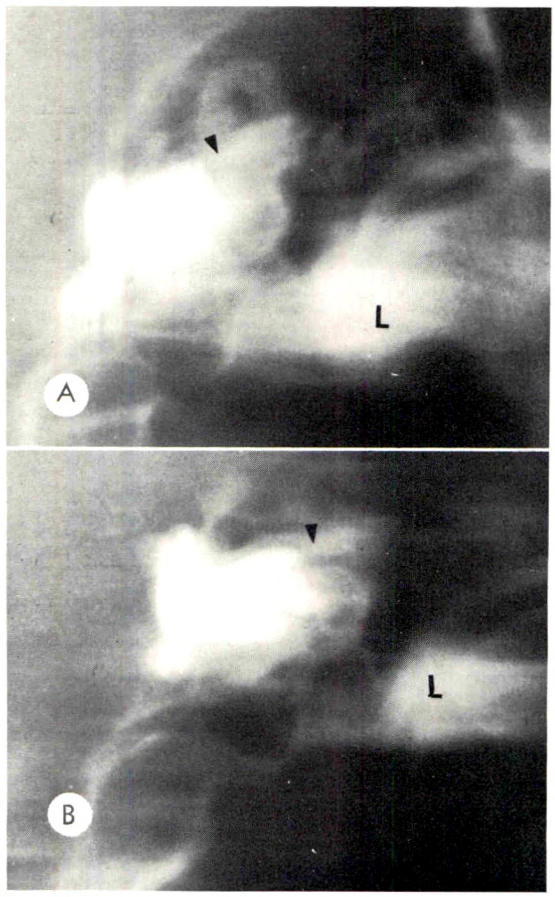


FIG. 15. (*A*) Minor deformity in the internal auditory canal in its lateral aspect (arrowhead). Doubtful appearances. (*B*) Second examination after 6 months. No change.

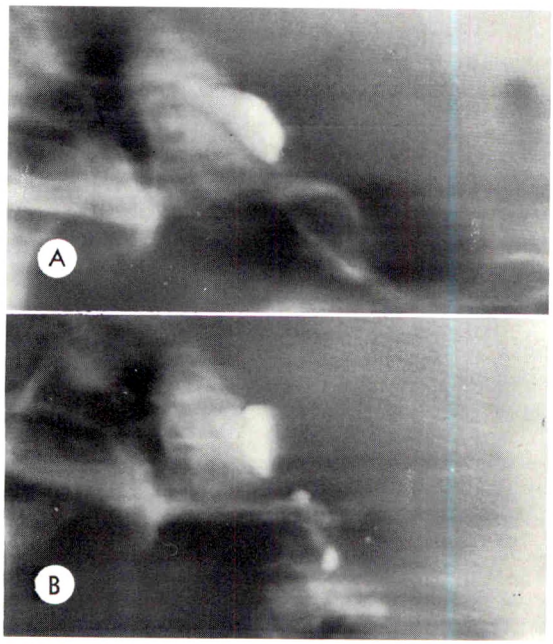


FIG. 16. (*A*) The right internal auditory canal is only partly filled with contrast medium. Considered strongly suggestive of a mass lesion. (*B*) Repeat examination 6 months later. The canal is less well filled, indicating growth in size of the lesion. Operation confirmed the presence of a right acoustic neuroma.

to clear the positive contrast material from the cerebello-pontine angle cistern prior to pneumoencephalography, and this contrast material may obscure detail. As stated before, we do not favor the use of large amounts of positive contrast material.

As a general rule, it has been found practicable to start with gas cisternography in those cases where there is clinical evidence of extension of the lesion beyond the porus (trigeminal involvement, brain stem signs, etc.) or if there are obvious bone changes in the petrous apex, porus acusticus or internal auditory canal. If this is negative, one may proceed to positive contrast cisternography (Fig. 12, *A* and *B*; and 13, *A* and *B*).

If the clinical evidence is less definite (sensineural or partial deafness, abnormal caloric responses) with no or minimal bone change, positive contrast material is used initially. This is still not always foolproof. One case (Fig. 18, *A* and *B*) showed canal dilatation only just outside the accepted limits of normal,[17] yet there was a moderately sized lesion present in the cerebello-pontine angle cistern.

It should be stressed that with the use of the modified "cross-table frontal projection," the whole length of the internal auditory canal is almost vertical and therefore parallel to the plane of tomographic section. Dorland *et al.*[5] described a similar projection, but in most other techniques, with the patient prone oblique and the beam directed posteroanterior,[16] the position is a variant of the Stenver's position and this in itself will result in foreshortening of the internal auditory canal. The technique can also be used for assessment of the jugular canal (Fig. 19).

Three definitive conclusions may be drawn:

1. If the lesion is projecting into the cerebello-pontine angle cistern, gas

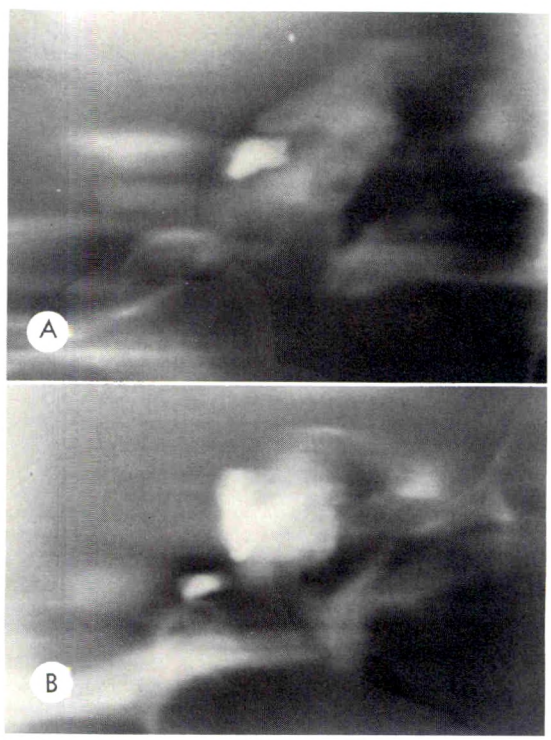


FIG. 17. Same case as in Figure 16. (*A*) Left meatogram. The lateral part of the canal is unfilled. (*B*) Repeat examination 6 months later. Normal filling of the whole canal, indicating that the earlier partial filling was artifactual.

cisternography will show it to best advantage and demonstrate its effects on adjacent structures.

2. If the lesion is intracanalicular, only positive contrast cisternography can demonstrate it.
3. The roentgenographic position used in this study for positive contrast cisternography is almost ideal in that the plane of tomographic section nearly corresponds to the full length of the internal auditory canal.

SUMMARY

The problems of investigating lesions in the internal auditory canal and cerebello-pontine angle cistern are outlined.

A method to investigate these lesions with negative or positive contrast material and using tomography is described.

Criteria for the choice of method are discussed, and cases showing the findings in lesions of varying size are illustrated.

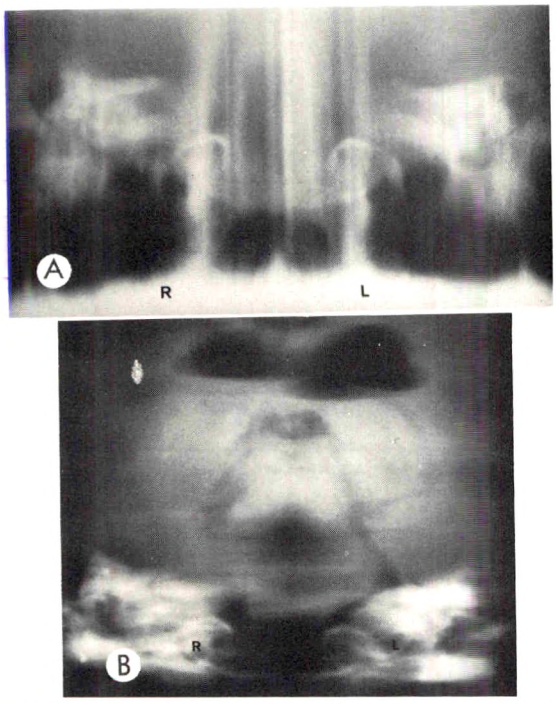


FIG. 18. Medium-sized acoustic nerve tumor. (*A*) Tomogram shows the right canal 2 mm. wider than the left with slight widening of the porus. (*B*) Pneumoencephalogram shows 1.5 cm. tumor impinging on the cerebello-pontine angle cistern and deforming the brain stem.



FIG. 19. Positive contrast cisternogram. The internal auditory canal is filled normally (arrow). Contrast medium is seen outlining the neurovascular structures in the jugular canal. The canal is wide, but no mass lesion is present (arrowheads).

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REFERENCES

1. BAKER, H. L., JR. Myelographic examination of posterior fossa with positive contrast medium. *Radiology*, 1963, *81*, 791–801.
2. BAKER, H. L. Cerebello-pontine angle myelography. *J. Neurosurg.*, 1972, *36*, 614–624.
3. BULL, J. In: discussion on acoustic tumours. *Proc. Roy. Soc. Med.*, 1965, *58*, 1077–1078.
4. BURROWS, E. H. Positive contrast examination (cerebello-pontine cisternography) in extra-meatal acoustic neurofibromas. *Brit. J. Radiol.*, 1969, *42*, 902–913.
5. DORLAND, P., KOBAYASCHI, N., MELZGER, J., STEEKERS, J. M., and VIAMO, J. Indications et technique de l'opacification du conduit auditif interne. *J. de radiol., d'électrol. et de méd. nucléaire*, 1972, *53*, 39–46.

6. Gass, H. Pantopaque anterior basal cisternography of posterior fossa. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 1197–1204.
7. House, W. F. Otosurgical approach to tumours of VIIIth nerve. *Proc. Roy. Soc. Med.*, 1965, *58*, 1071.
8. Isherwood, I. Air meatography. *Clin. Radiol.*, 1972, *23*, 65–77.
9. Long, J. M., Kier, E. L., and Hilding, D. A. Pitfall of posterior fossa cisternography using 2 ml. of Iophendylate (Pantopaque). *Radiology*, 1972, *102*, 71–75.
10. Mones, R., and Werman, R. Pantopaque myeloencephalography. *Radiology*, 1959, *72*, 803–809.
11. Morris, L., and Wylie, I. G. Tomography in cerebral pneumoencephalography. *Clin. Radiol.*, 1973, *24*, 221–230.
12. Reese, D. F., and Bull, J. W. D. Positive contrast demonstration of normal internal acoustic meatus, Meckle's cave and jugular foramen. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 650–655.
13. Scanlan, R. L. Transtemporal bone microsurgical removal of acoustic neuroma: positive contrast medium (Iophendylate) in diagnosis of acoustic neuroma. *Arch. Otolaryngol.*, 1964, *80*, 698–706.
14. Shahon, M., and Wiener, S. N. Pantopaque examination of cerebello-pontine angle. *Radiology*, 1965, *85*, 921–926.
15. Siew, F. P., Kricheff, I. I., and Chase, N. E. Demonstration of small acoustic neuromas, using negative contrast medium with tomography. *Radiology*, 1968, *91*, 764–769.
16. Valvassori, G. E. Myelography of internal auditory canal. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 578–586.
17. Valvassori, G. E. Abnormal internal auditory canal: diagnosis of acoustic neuroma. *Radiology*, 1969, *92*, 449–459.
18. Wende, S., and Lüdecke, B. Technique and value of gas and Pantopaque cisternography in diagnosis of cerebello-pontine angle tumors. *Neuroradiol.*, 1971, *2*, 24–29.

Vol. 122, No. 3

PEG "WAVES"*

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AIR-FLUID levels produced within the ventricles and subarachnoid spaces during pneumoencephalography (PEG) are often wave-like in contour[2] (Fig. 1). To investigate the frequency with which these PEG "waves" occur and to determine whether their presence correlates with clinical or cerebrospinal fluid (CSF) abnormalities, 135 consecutive cerebral air studies were evaluated.

The majority of the 135 patients in this series were studied for atrophic and degenerative conditions, although some tumors and infectious conditions were included. There was a wide age distribution, from months to 89 years, with the number of females slightly predominant over the number of males, 73 to 62. Cerebrospinal fluid protein levels ranged from low normal to markedly elevated.

The pneumoencephalograms were done by the usual fractional technique,[13] air being introduced in several fairly slow 10 or 15 cc. injections up to a total amount of 40 to 60 cc. No more than a few cc. of fluid were removed until later stages of the study.

RESULTS

PEG "waves" occurred commonly in the lateral ventricles (Fig. 1; and 2, *A* and *B*), but much less frequently elsewhere. In fact, in this series they were found to some degree on early filling films of the lateral ventricle in approximately 3 cases out of every 4 (97 of 135, or 72 per cent). There was no apparent correlation with CSF protein levels. Thus the occurrence of PEG "waves" in the lateral ventricles on early filling films of pneumoencephalography *can be considered normal or typical* and certainly does not imply an increase in CSF protein.

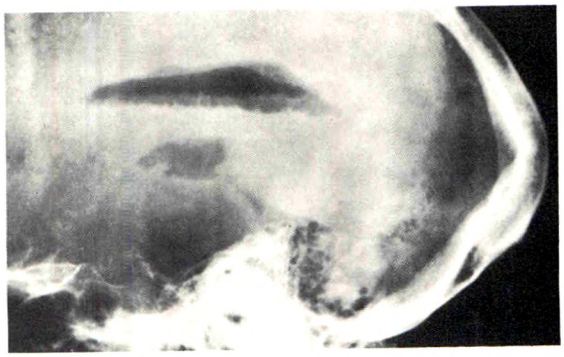


FIG. 1. Typical PEG "waves" (actually, a foam of air bubbles and CSF) shown in the lateral view during early filling of the lateral ventricles. The patient was an elderly male with normal CSF protein. Subsequent roentgenograms showed dilated cortical sulci but the "waves" disappeared.

As the air study proceeds, and more air fills the lateral ventricles, the PEG "waves" disappear, often first becoming irregular and of longer "wavelength," (Fig. 2, *A* and *B*). Where there is unequal filling of the lateral ventricles early in the study, almost invariably the wave-like appearance is more pronounced in the ventricle which is less well filled. In most cases PEG "waves" disappear by the time brow-up roentgenograms are obtained, although at times they persist and can be observed along the inferior aspect of the frontal horns or even passing through the foramen of Monro.

In a few patients (14 of 135) "waves" were present on early PEG films in "atypical" locations, other than (or in addition to) the lateral ventricles. These sites included the basal cisterns, fourth ventricle, third ventricle and superior cerebellar cisterns. In 1 infant with hydrocephalus, "waves" were present in continuity through a dilated third ventricle, aqueduct and fourth ventricle. In all cases where "waves" occurred at atypical sites, there was considerable dilatation of the ventricles

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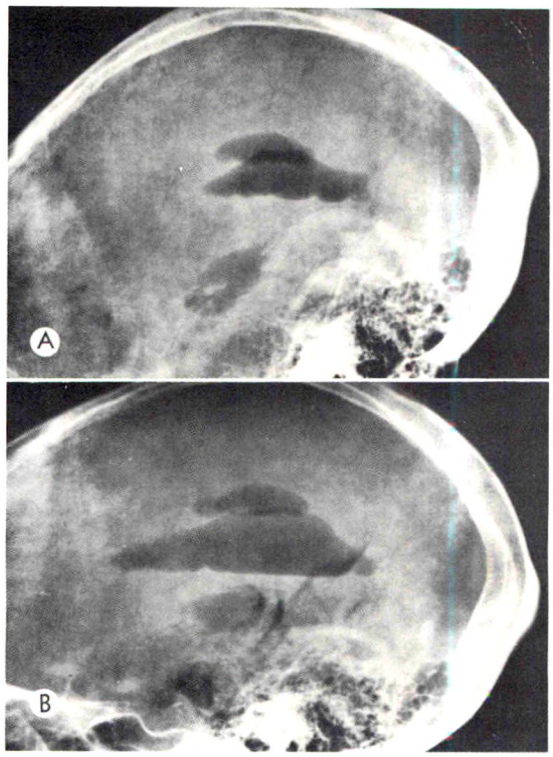


FIG. 2. (*A*) Typical lateral ventricle PEG "waves" in a 65 year old female examined for dementia. (*B*) As more air was introduced, the "waves" were of longer "wavelength" and thus became less prominent. Although it is not too striking in this example, PEG "waves" are generally more pronounced in the less well-filled ventricle, when filling is asymmetric.

or cisterns concerned.

In 30 cases, or 22 per cent of the series, PEG "waves" were entirely absent. In this group of patients, females outnumbered males 22 to 8.

THE NATURE OF PEG "WAVES"

Although cerebrospinal fluid pulsations exist and have been correlated with the arterial and venous pulses[3,5,10,14] as well as with slower fluctuations due to respiration,[5,12] PEG "waves" are not true waves at all. The wave-like air fluid levels seen at pneumoencephalography are made up of bubbles of air and CSF which have combined to form an unstable foam.[2,4,7,11] A wave-like appearance is simulated because the upper surfaces of the air bubbles are often quite thin and usually do not visualize in lateral views. The approximately spherical outlines of the bubbles may be more apparent in frontal views, especially in hydrocephalic patients.

One can reproduce PEG "waves" *in vitro* by bubbling air through a glass flask containing CSF and air.[2] Even more simply, a foam lasting 2 to 4 minutes can be produced in a partially filled bottle or tube of CSF* by shaking it. After shaking is stopped the foam decays irregularly. The last bubbles to burst are those at the ends of the air-fluid level in contact with the glass bottle or tube.

FOAM FORMATION AND CSF CONSTITUTION

A foam is a coarse dispersion of gas in a liquid achieved by agitating or bubbling the gas and liquid together. Most of the volume of a foam is gas with liquid present only in thin layers (called lamellae) between the gas bubbles.[7] A foam begins to decay immediately after agitation or bubbling of the gas ceases, because liquid drains from the lamellae under the influence of gravity and surface tension forces.[7] Although the decay of a foam is often roughly exponential, it should be noted that "foams may collapse irregularly, perhaps leaving blocks of foam stranded on the sides of a vessel while the last few bubbles often prove exceptionally persistent."[11] In general, as a foam decays, larger bubbles tend to grow even larger at the expense of smaller because of physical factors.[7]

Extremely small amounts of some surface active materials, such as saponins, promote significant foaming, since they become concentrated at the surface of the fluid out of all proportion to their over-all bulk concentration. Thus, in one experiment,[1] the life of a foam produced in a saponin solution measured 170 seconds for a 1 per cent solution. Foam life decreased to 160 seconds for a $\frac{1}{4}$ per cent solution, yet still remained 120 seconds for a solution of saponin as dilute as .008 per cent.

* Q Pak spinal fluid control available from Travenol Laboratories, Inc., Costa Mesa, California 92626.

Protein solutions, including albumins, promote foaming if not in quite as dilute concentrations as saponins.[4] Their behavior with decreasing bulk concentration is also qualitatively fairly similar although more complex because the foaming of protein solutions is also affected by pH and the concentration of polyvalent cations, especially calcium ions.[4] Other surface active components in CSF, especially phospholipids,[9] may contribute to foaming.

High viscosity in a bulk fluid tends to interfere with drainage of liquid from the thin lamellae of a foam.[11] This is familiar from commercial applications in aerosols. For the patients in this series with increased CSF protein, however, this viscosity effect appears to have been relatively minor since their PEG "waves" did not appear to differ noticeably from those of patients with normal CSF protein levels.

Mechanical Factors in Foam Production in Relation to the Formation of PEG "Waves"

Gas introduced through a capillary tube within a fluid produces bubbles. Their size depends in a fairly complicated way upon the size of the tube, its orientation and the rate at which the gas is introduced.[4,6] At low gas velocity, the radius R of the bubble is proportional to the cube root of the radius of the capillary tube, r. Under these conditions, the buoyancy force approximately equals the surface tension force.

The orientation of the capillary tube is important, since it is found experimentally that a vertically oriented capillary tube gives rise to bubbles which are twice as large as those emanating from a horizontal tube.[4,6] Finally, increasing the rate at which gas is bubbled through the capillary tube may yield bubbles whose size is in excess of those produced by slow bubbling.[4]

These considerations can be applied to arrive at an understanding of the mechanical factors tending to promote the formation of PEG "waves" in the lateral ventricles during pneumoencephalography. They are not due to pulsation of the ventricles, for although the ventricles dilate at pneumoencephalography,[13,15,16] there is little or no pulsatile movement of the lateral ventricles.[8] Moreover, Robertson[15,16] has shown that essentially the same filling mechanisms occur during pneumoencephalography in cadavers as in patients. According to Robertson,[17] after 12 ml. of gas was injected into the lumbar region with the head flexed to 45° and lateral films were exposed at a rate of 2 films per second: "Gas appeared in the cisterna magna during the third second and in the fourth ventricle at the fifth second. At five and one half seconds the fourth ventricle was fairly well filled and the third ventricle was seen to be filling. During the sixth second, the gas level had fallen to the level of the foramen of Monro and *suddenly* a fair volume of gas appeared in the lateral ventricles. At seven seconds, more gas was seen in the lateral ventricles and the amount increased until, *after nine seconds, no further change occurred.*" (Italics those of the present author.) Amler,[1] performing pneumoencephalography under direct vision using an image amplifier also was impressed by the rapidity of filling of the lateral ventricles. He noted that it occurred in an unexpectedly rapid blow-like fashion after air first had "stagnated" for a few seconds within the third ventricle. Amler[1] also reported seeing wave-like air fluid levels in PEGs done in children and hydrocephalic adults, although he erroneously attributed them to to-and-fro head movements.

Correlating these observed events at pneumoencephalography with the physical factors involved in foam formation discussed earlier, one can conclude that PEG "waves" tend to form within the lateral ventricles during the early stages of ventricular filling because of the sudden high rate of air influx as air first reaches the foramen of Monro. This is when buoyancy forces are maximal; *i.e.*, the height of the fluid level within the lateral ventricle is greatest and it is also when the lateral ventricles are most distensible.[13,15,16] The influx of air into the lateral ventricles is

aided by the relatively large size of the foramina of Monro and their more or less vertical position, along with the anterior wall of the third ventricle, during flexion of the patient's head. There is a tendency for PEG "waves" to be more striking and persistent in hydrocephalics[2] probably reflecting enlargement of the foramina of Monro in these patients and a relative increase in pressure head because of the large ventricles. The presence of PEG "waves" at atypical sites other than the lateral ventricles, also seems to be promoted by the presence of unusually dilated channels.

SUMMARY

PEG "waves" were found at air-fluid interfaces in the lateral ventricles on early filling roentgenograms in about 75 per cent of patients in this series of 135 consecutive air studies. PEG "waves" were found at atypical sites (other than the lateral ventricles) in 14 cases. PEG "waves" were entirely absent in approximately 22 per cent of patients, the majority of whom were females.

Analysis of the roentgenographic appearance of these PEG "waves" leads to the conclusion that they represent a foam of air bubbles and CSF. The fact that this foam tends to form most often in the lateral ventricles correlates with the very rapid rate at which air has been observed to enter the lateral ventricles through the foramina of Monro during the first stages of ventricular filling at pneumoencephalography. This rapid inrush of air under buoyancy and surface tension forces during early filling is promoted by certain physical factors:

1. The foramina of Monro are relatively large and are approximately vertical in position, along with the anterior wall of the third ventricle, when the patient's head is slightly flexed.
2. Maximal pressure head as well as maximal ventricular distensibility occur at the beginning of lateral ventricular filling.

It is observed that, when there is asymmetric filling of the lateral ventricles, PEG "waves" are generally more prominent in the lateral ventricle which is less well filled. Furthermore, the "wavelength" of PEG "waves" becomes longer as more air is introduced. Both of these observations can be explained by the tendency of an air-CSF foam to decay within a few minutes with larger bubbles persisting and growing at the expense of smaller ones.

Although it might have been anticipated that an increased spinal fluid protein content should tend to make PEG "waves" more persistent, this effect was not observed. Two plausible explanations for this appears to be that:

1. Proteins are not the only surface active material in CSF.
2. Surface active materials, since they are concentrated at air-fluid interfaces, affect foam formation to a much greater and more uniform degree than one might otherwise expect from their simple bulk concentration.

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REFERENCES

1. Ammer, G. Pneumonencephalographie unter Kontrolle des Bildverstärkers. *Nervenarzt*, 1964, *35*, 509–512.
2. Bäcker, F. Wodurch wird der Flüssigkeitspiegel beim Pneumonencephalogramm Wellenartig Verformt? *Nervenarzt*, 1968, *39*, 556–557.
3. Bering, E. A., Jr. Choroid plexus and arterial pulsations of CSF: demonstration of choroid plexus as CSF pump. *A.M.A. Arch. Neurol. & Psychiat.*, 1955, *73*, 165–172.
4. Bikerman, J. J. Foams: Theory and Industrial Applications. Reinhold, New York, 1953.
5. Dardenne, G., Dereymaeker, A., and Lacheron, J. M. Cerebrospinal fluid pressure and pulsatility. *Europ. Neurol.*, 1969, *2*, 193–216.
6. Datta, R. L., Napier, D. H., and Newitt, D. M. Properties and behaviour of gas bubbles

formed at circular orifice. *Tr. Inst. Chem. Engnrs.*, 1950, *28*, 14–26.

7. DAVIES, J. T., and RIDEAL, E. K. Interfacial Phenomena. Academic Press, Inc., New York, 1961.
8. DUBOULAY, G. H. Pulsatile movements in CSF pathways. *Brit. J. Radiol.*, 1966, *39*, 255–262.
9. GILLAND, O. Cerebrospinal fluid. *Prog. Neurol. & Psychiat.*, 1970, *25*, 214–237.
10. HAMIT, H. F., BEALL, A. C. R., and DEBAKEY, M. E. Hemodynamic influences upon brain and CSF pulsations and pressures. *J. Trauma*, 1965, *5*, 174–184.
11. KITCHENER, J. A. Foams and free liquid films. In: Recent Progress in Surface Science. Volume I. Edited by J. F. Danielli, K. G. A. Pankhurst, and A. G. Riddiford. Academic Press, Inc., New York, 1964.
12. LEVINGER, I. M., and KEDEM, J. Further mechanisms determining respiratory waves in cerebrospinal fluid pressure. *Europ. Neurol.*, 1972, *8*, 325–332.
13. LIM, S. T., POTTS, D. G., and DECK, M. D. F. Changes in ventricular size during fractional pneumoencephalography. *Radiology*, 1972, *104*, 585–592.
14. MOSKALENKO, Y. E., KISLYAKOV, Y. Y., VAINSHTEIN, G. B., and ZELIKSON, B. B. Biophysical aspects of intracranial circulation. *Am. Heart J.*, 1972, *83*, 401–414.
15. ROBERTSON, E. G. Further Studies in Encephalography. The Macmillan Company, Melbourne, 1946.
16. ROBERTSON, E. G. Some physical aspects of encephalography. *Brain*, 1947, *70*, 59–74.
17. ROBERTSON, E. G. Pneumoencephalography. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1967.

A "PIGGY-BACK" BULLET WOUND OF THE HEAD*

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THE skull represents a unique area in which a gunshot injury is rather easy to evaluate roentgenologically. Since the skull is a closed area, the radiologist can demonstrate entrance and exit sites in a penetrating wound. Skull bone fragments will often be propelled into the brain at the site of entrance of a bullet. Similarily, bone fragments may be displaced into subcutaneous location at the exit wound. Metal fragments along the bullet track may give evidence of the direction taken by the bullet. If the bullet loses enough kinetic energy during brain excursion, it may remain within the skull and can easily be located on roentgenograms. The clinical advantages of a skull roentgen examination are evident and may aid in assessing what vital brain or vascular structures have been injured.

The skull roentgenologic examination may also be of value to the forensic pathologist in his prospective evaluation before autopsy. Clues to the type of injury may be defined on roentgenographic study of the bullet fragments present.

We recently encountered a case in which the skull roentgenogram alerted the pathologist to the presence of an unusual form of injury, the so-called "piggy-back" bullet wound.

REPORT OF A CASE

A 52 year old white man was admitted to the emergency room in a comatose condition with a diagnosis of a gunshot wound of the head. He was completely unresponsive, and no movement resulted from deep, noxious stimulation.

Skull roentgenograms were obtained and revealed a single, oval, lucent skull entrance defect in the right temporal area. Figure 1, *A*

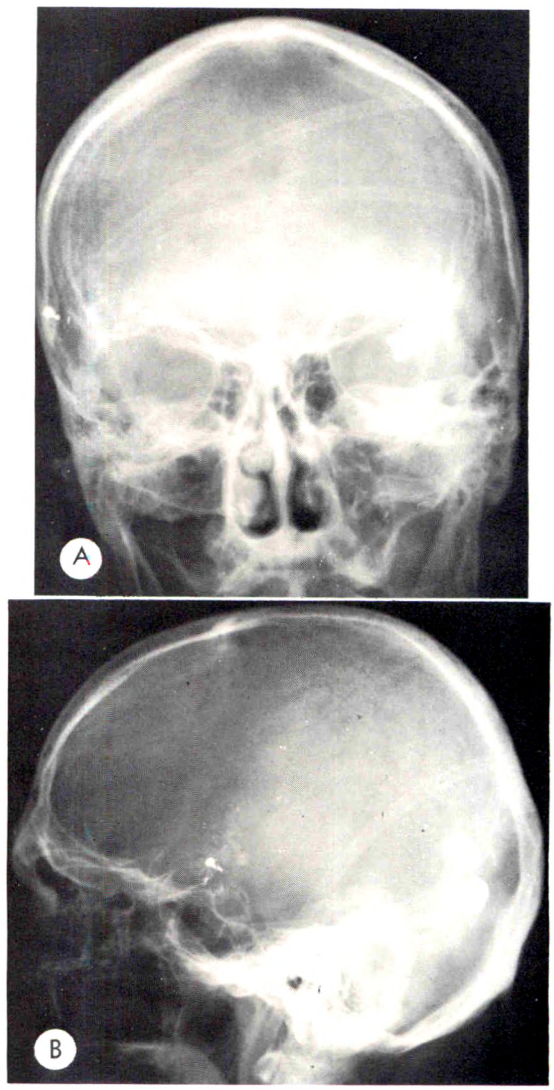


FIG. 1. (*A*) A frontal skull view with a temporal bone defect on the right, medial to which are bone and metallic fragments indicating an entrance gunshot wound. Two bullets are present on the left. The lower bullet is the leading portion, and the upper bullet is the propulsive portion of a "piggy-back" missile. (*B*) A lateral view of the same skull. Bullet positions are the same as in *A*.

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and *B*, illustrates the frontal and lateral views which were obtained. No other skull wound was present. The left parietal-occipital area contained 2 rather complete bullet fragments. Tiny metal fragments demonstrated the left posterolateral direction which the bullets took.

The patient remained unresponsive despite supportive care and died 48 hours after admission.

Past history was well documented, because the patient had been evaluated for an embryonal carcinoma of both testes 12 years earlier and had been followed regularly. He had been treated with bilateral orchiectomy and retroperitoneal lymph node dissection at that time. One periaortic lymph node contained metastatic embryonal carcinoma. He also had been hospitalized for chronic alcoholism 2 years before this admission. There was no previous history of gunshot or head injury of any sort.

Autopsy examination of the body revealed a single wound in the right temporal area that was consistent with an entrance type of gunshot wound.[2] Associated with the cutaneous wound was a 9 mm. circular defect of the right temporal bone outer table and a 1.5 cm. "dished out" defect of the inner table. The dura was torn and "blackened" under the bone defect. Two bullets were found in the subdural area of the left occipital region. Examination of the brain revealed a single bullet track extending from the right sylvian fissure and temporal lobe in a left posterior direction to the tip of the left occipital lobe.

Other incidental findings were: evidence of a recent myocardial infarct (approximately 5 days old); and no evidence of recurrent embryonal carcinoma.

The general investigation of this case revealed that the patient was a childless, divorced man who worked as a painter. He was a rather difficult individual, particularly when drinking. He had been living in a single room of a rooming house for several years. He was found by another boarder who, after knocking and receiving no response, entered the room and found the victim lying on his bed with a .32 caliber revolver beside him. A suicide note was found. An acquaintance identified the gun as one belonging to the victim.

DISCUSSION

Cushing, as quoted by Hooper *et al.*[4] called attention to the temporal area as the favorite site for self-inflicted gunshot wounds. A single entrance wound is incongruous with the presence of 2 bullets unless it is assumed that both entered the skull through the same defect. The roentgenologic examination was the first clue to the presence of the "piggy-back" arrangement of the bullets which entered the skull. If one studies the roentgenographic appearance of the bullets shown in Figure 1, *A* and *B*, it is apparent that the lower bullet has a flattened distal tip in the frontal view. The flattening implies that this was the bullet which first passed through the skull. The lower bullet also has a concave base in the frontal view which conforms to the shape of the nose of the upper bullet as seen in the lateral projection (Fig. 1*B*). The base of the upper bullet is flat.

Spitz[7] has called attention to the "piggy-back" or tandem bullet and describes a case in which a bullet mass removed from the head of a patient with a .38 caliber gunshot wound weighed nearly twice as much as an intact bullet recovered from another wound in the same patient. He postulates that a bullet which had remained in the barrel of a gun was propelled by and fused to a second bullet that was fired later. He lllustrates a rather amorphous metallic mass which does simulate the shape of 2 bullets fused during firing. Roentgenograms were not presented with this case.

Ballistics study in our case revealed that the 2 bullets could be assembled into a single missile (Fig. 2, *A* and *B*). Figure 2*C* illustrates the nose appearance of the 2 bullets. That on the right is the leading portion of the "piggy-back" missile which was flattened as it penetrated the skull. The bullet on the left represents the second propelling portion of the missile which relatively retains its original shape. In Figure 2*D*, flaring of the proximal end of the first bullet resulting from intrusion by the propelling bullet is seen on the right. The proximal end of the propelling bullet is flat. The shape of the leading bullet corresponds to the lower metallic image in Figure 1, *A* and *B*. Similarly, the shape of the propel-

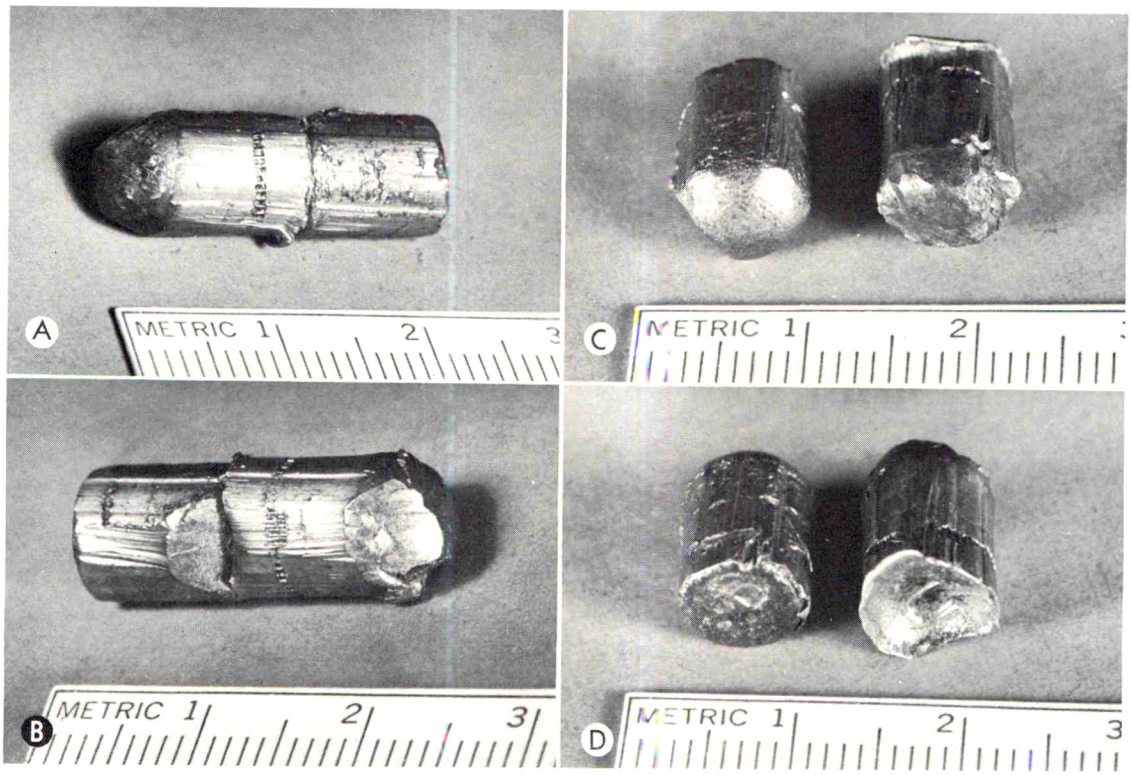


FIG. 2. (*A*) A side view of the assembled "piggy-back" missile. (*B*) A view of the opposite side of the "piggy-back" missile. The leading edge of each bullet has a similar chip fragment missing. (*C*) View of the nose ends of the disassembled missile. The leading bullet is on the right and has a flattened surface. The left object is the propelling bullet and retains nearly normal shape. (*D*) View of the following ends of the disassembled missile. The leading bullet has an indented, flared end on the right. The propelling bullet has a flat, unindented end (left).

lant bullet corresponds to the upper metallic image in this illustration.

One other important ballistics feature is revealed in Figure 2*B*, *i.e.*, each bullet has a "chipped-off" portion adjacent to the leading end. This defect is due to imperfect alignment between the cylinder of the gun and the barrel. As a bullet is fired, a portion of the nose is sheared off by the edge of the barrel which overlaps the cylinder. This defect further confirms the origin of both bullets as being from the same gun.

The best explanation for the production of a "piggy-back" or tandem bullet lies in defective ammunition. If a cartridge is loaded with too little powder, firing will fail to propel a bullet completely through the barrel. Similarly, wet or aged powder may be inadequate to move a bullet out of the barrel. A second shot is then fired and has adequate propulsive force to fuse the 2 bullets into 1 mass, as in the case described by Spitz, or gives flight to both bullets as in our case.

Tandem loading of 2 or more bullets in 1 cartridge has been reported. Petty[6] refers to a military tandem or duplex load in which a solid base bullet is inserted into the hollow base of another bullet and both are propelled by a single powder charge. These bullets are only available for rifles. An F.B.I. bulletin[3] describes a reloaded .38 caliber cartridge in which 2 extra projectiles were seated on the original bullet in a cartridge case. Theoretically, the military tandem load or that described in the F.B.I. bulletin might produce an injury similar to that seen in our case, if fired with the barrel of the weapon in contact with the head. However, none of the other cartridges

in the gun found in the present case had multiple bullet loads. Also, the nose defect of the missiles, as previously described, would indicate individual firing.

It is not known when the first bullet was fired in this case. No witness heard a shot fired. The leading missile may have been propelled into the barrel some time previously or immediately before the second cartridge was discharged. The "piggy-back" effect would result in either circumstance.

Separation of the 2 parts of the "piggy-back" missile must have occurred either as a result of the "tumbling" effect as described by Mittleholzer and Shields[5] or as a result of a gravitational change in position as reported by Adeloy and Odeku.[1]

This case represents the first report of the roentgenologic aspects of a "piggy-back" bullet wound which has appeared in the English language literature.

SUMMARY

A suicide fatality in which 2 bullets entered the head in tandem through a single skull defect is described. This has been termed a "piggy-back" bullet wound.

Such an injury is produced by a defective cartridge allowing a bullet to remain in the gun barrel after firing, followed by a second shot which then propels both bullets as a single missile.

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REFERENCES

1. Adeloy, A., and Odeku, E. L. Radiology of missile head wounds. *Clin. Radiol.*, 1971, *22*, 312–320.
2. Adelson, L. Microscopic study of dermal gunshot wounds. *Am. J. Clin. Path.*, 1961, *35*, 393–402
3. Anonymous. "Piggy back" cartridge has surprising results. F.B.I. Law Enforcement Bulletin, 1965, *34*, 19.
4. Hooper, R. E., Ruben, R. J., and Khalid, M. Gunshot injuries of temporal bone. *A.M.A. Arch. Otolaryng.*, 1972, *96*, 433–440.
5. Mittleholzer, E., and Shields, J. B. Tumbling bullet. *Radiology*, 1970, *97*, 625–627.
6. Petty, C. S. Firearms injury research. *Am. J. Clin. Path.*, 1969, *52*, 277–288.
7. Spitz, W. U. Medicolegal Investigation of Death. Charles C Thomas, Publisher, Springfield, Ill., 1973, pp. 225–226.

SELECTIVE TEMPORAL ARTERIOGRAPHY AND BIOPSY IN GIANT CELL ARTERITIS: POLYMYALGIA RHEUMATICA*

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SINCE Horton's original description of temporal arteritis in 1932,[10] many investigators have studied this entity. As early as 1941, Gilmour[5] set forth the pathologic findings under the title "Giant Cell Arteritis." Temporal arteritis thus became known as a syndrome in the elderly, consisting of band-like headaches with painful and tender superficial temporal arteries often followed by blindness and/or stroke.

Then in 1957, Barber[1] coined the eponym polymyalgia rheumatica, thus ultimately diagnosing and defining a new clinical syndrome. The clinical features of polymyalgia rheumatica include pain and stiffness of the muscles of the neck, back and proximal girdle areas, but without weakness or atrophy. Anorexia, weight loss, malaise and low grade fever are often associated complaints. Physical examination may reveal mild arthritis of larger joints and, rarely, bruits over large arteries secondary to stenosis. Laboratory studies reveal an elevated sedimentation rate of 50 mm./hr. or greater, Westergren method; a mild anemia of normocytic hypochromic type; elevated fibrinogen; and α 2 globulin. The electromyogram, muscle biopsy, muscle enzymes, rheumatoid factor and antinuclear antibody studies are usually negative. The disease classically responds dramatically to glucocorticoids, usually within a few days.

Both polymyalgia rheumatica and temporal arteritis are diseases with chronic inflammatory involvement of the medium sized arteries. Nevertheless, the difficulty in establishing the diagnosis of vascular involvement in polymyalgia rheumatica through biopsy has long been recognized.[2] This prompted Palm in 1958[13] to undertake a retrospective study of carotid angiograms in patients with temporal arteritis and polymyalgia rheumatica. Four of these exhibited abnormal studies suggesting arteritis. Later, in 1964, Hamrin *et al.*[9] reported the first major series of biopsy cases in patients with clinical giant cell arteritis, with 60 per cent exhibiting positive histology. Still, the persistent small yield of positive biopsy findings in many series of cases of polymyalgia rheumatica can be as low as 5–10 per cent.[8,11]

This discrepancy suggested that arteriography combined with biopsy might be complementary methods in establishing the diagnosis.[6] We would therefore like to present our own experience with selective temporal arteriography and biopsy.

MATERIAL

Out of a large Rheumatology Clinic, 20 patients suspected of having polymyalgia rheumatica were referred to the Radiology Department for temporal arteriography and biopsy. Their positive clinical and laboratory findings are depicted in Figure 1. It must be stressed that this series has been limited to the search for arteritis in those patients with polymyalgia rheumatica. We have carefully excluded patients with temporal arteritis, specifically those with headaches, visual disturbances and palpable temporal arteries.

METHOD

The procedure is carried out entirely under the auspices of the Radiology De-

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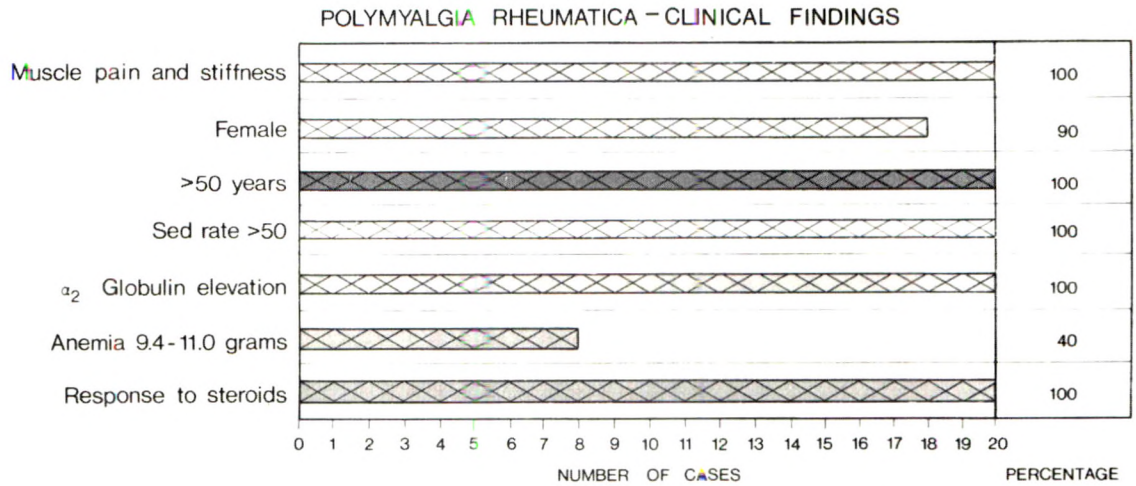


FIG. 1. Positive clinical and laboratory data in 20 patients with polymyalgia rheumatica.

partment. The patient is brought to the Special Procedures Room. Surgical preparation of the skin just anterior to the ear is obtained. Following infiltration with procaine 1 per cent a 2 cm. incision is made horizontally just anterior to the tragus of the ear above the malar bone. Utilizing blunt dissection, the artery is freed up and tied at its most proximal end. A polyethylene longdwell catheter is introduced 0.5 to 1 cm. within the artery and tied in place. Care must be taken not to introduce the catheter too far as the superficial temporal artery frequently bifurcates just above this level. Subsequently, 2 to 4 cc. of meglumine diatrizoate* is rapidly introduced and magnification films are obtained at the rate of 1 film per second for 4 seconds utilizing a 0.3 mm. focal spot, object film distance of 20 inches and focal film distance of 40 inches. The patient's discomfort is small. The catheter is then removed, the artery is tied distally and a biopsy specimen as long as possible is obtained. Closure is completed with 5/0 nylon.

PATHOLOGY

Early or mild disease affects the intima with elastic fiber degeneration and chronic inflammatory cell infiltration. Giant cells containing phagocytized elastic fibers are present. The endothelium and subendothelial fibroblasts proliferate, resulting in nodular or diffuse thickening of the intima and narrowing of the arterial lumen. The artery may be involved circumferentially or in only a portion of the wall. The result is either a concentric or eccentric deformity and frequent skip areas. Extension of this

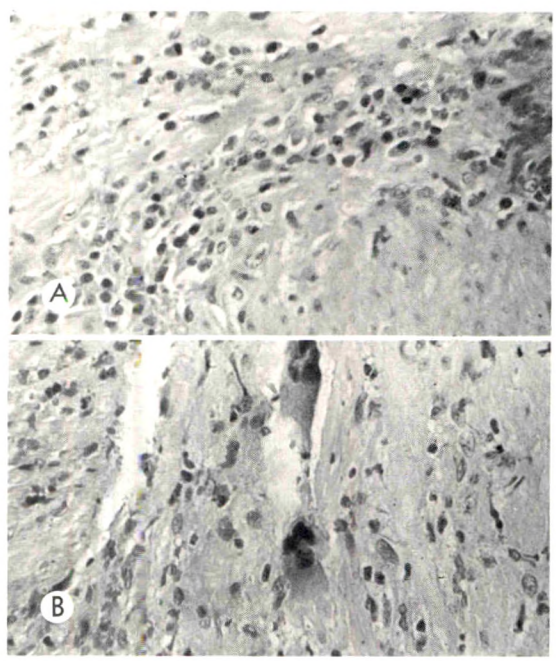


FIG. 2. (*A*) High power cross section view in temporal arteritis. Extensive infiltration of media with inflammatory mononuclear cells. (*B*) Same as *A*. Multiple inflammatory giant cells are present.

* Renografin 76.

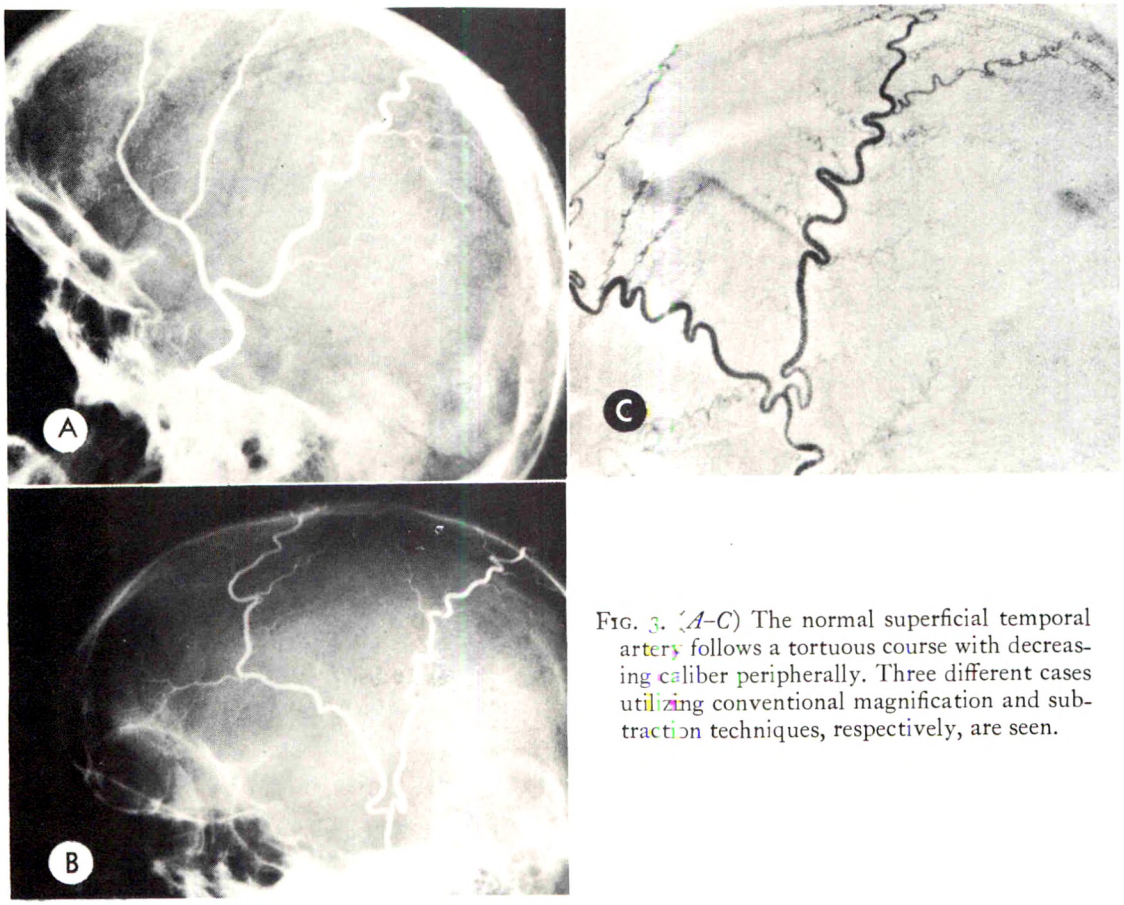


FIG. 3. (*A–C*) The normal superficial temporal artery follows a tortuous course with decreasing caliber peripherally. Three different cases utilizing conventional magnification and subtraction techniques, respectively, are seen.

process leads to medial degeneration and, rarely, to total fibrosis of the vessel with thrombosis and obliteration of the artery lumen (Fig. 2, *A* and *B*).

RESULTS

The normal superficial temporal artery follows a rather serpiginous course with decreasing caliber peripherally (Fig. 3, *A–C*). The pathologic vessel usually runs a straighter course with areas of narrowing, beading and dilatation (Fig. 4, *A–C*). In occasional cases, areas of disease involvement will alternate with areas of normal vessel. The operator then has the option of obtaining an additional biopsy at the distal site of angiographically demonstrated disease, if he considers that microscopically the main temporal artery is normal. It must be stressed that these subtle findings are best demonstrated with magnification.

Occasionally, the catheter may be advanced beyond the bifurcation resulting in filling of only the anterior or posterior branch of the temporal artery (Fig. 5, *A* and *B*).

Likewise, manipulation of the tissues in dissection of the artery will frequently result in the spasm of that vessel leading to inadvertent injection of a corresponding vein (Fig. 6). The vein, however, is identified by its size, general configuration and presence of valves.

It should be appreciated that an area of tapered narrowing commonly seen at the catheter tip simply represents the "jet collapse" described by Doumanian and Amplatz.[3] This should not be interpreted as an area of diseased vessel (Fig. 7).

Temporal arteriography can also be utilized to visualize the vascular supply of metastatic lesions (Fig. 8) or primary

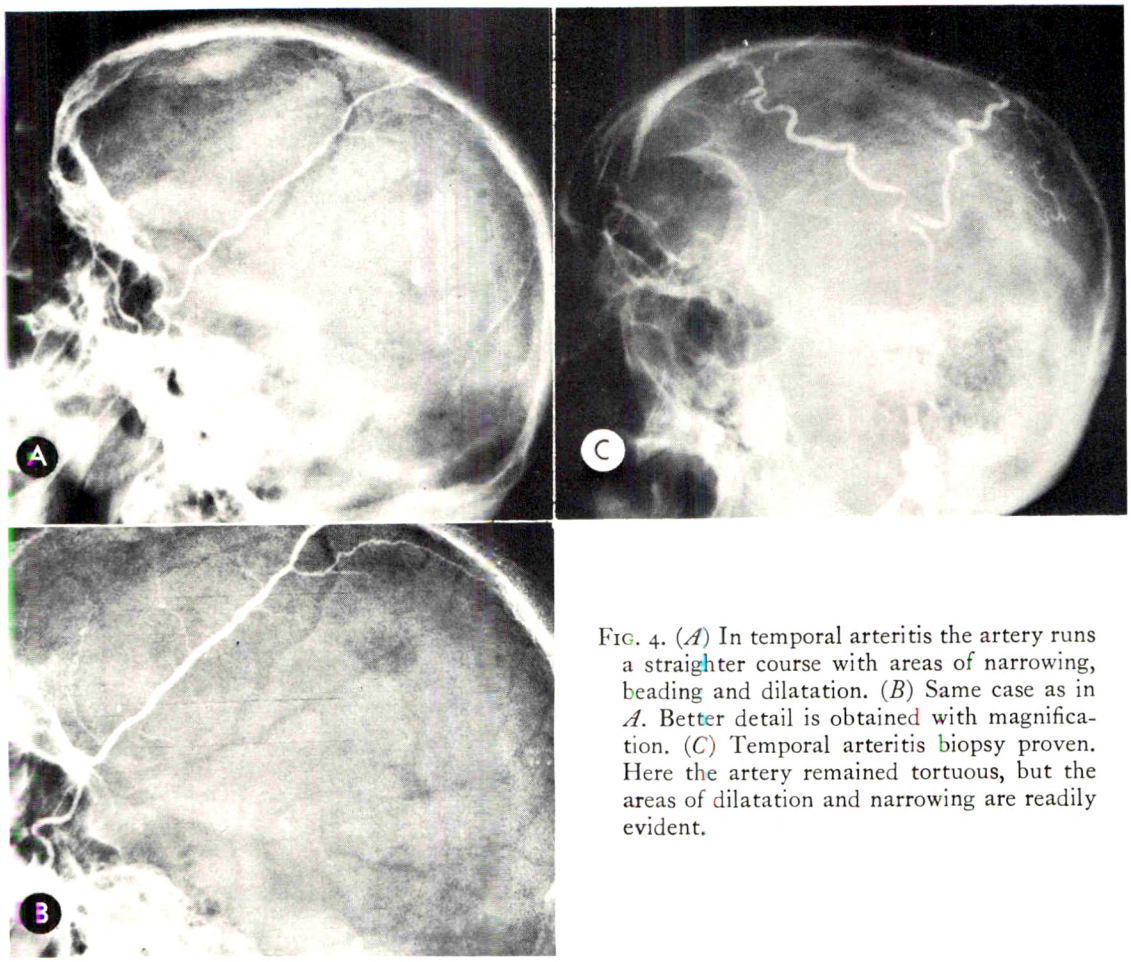


Fig. 4. (*A*) In temporal arteritis the artery runs a straighter course with areas of narrowing, beading and dilatation. (*B*) Same case as in *A*. Better detail is obtained with magnification. (*C*) Temporal arteritis biopsy proven. Here the artery remained tortuous, but the areas of dilatation and narrowing are readily evident.

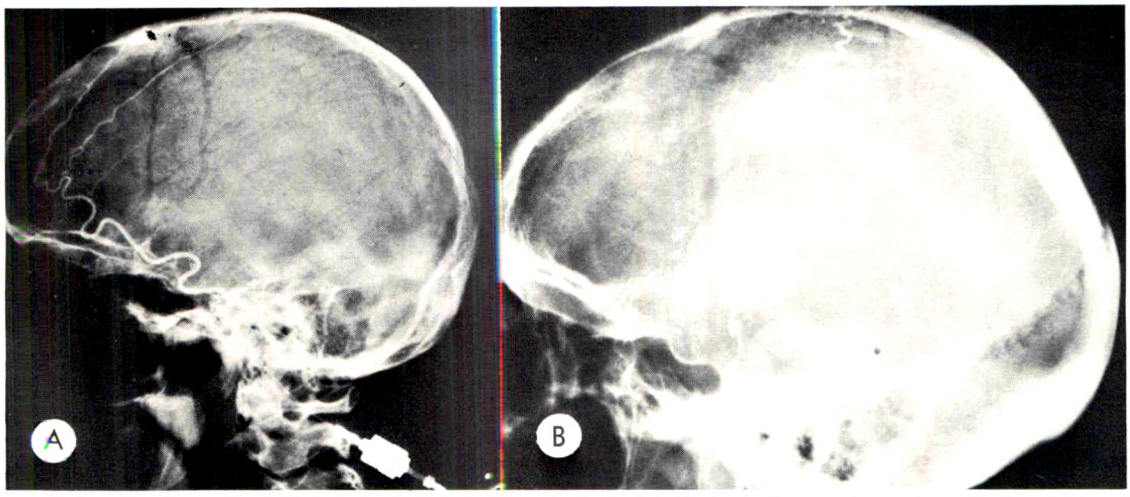


Fig. 5. (*A* and *B*) Inadvertent advancement of the catheter beyond the bifurcation resulting in only opacification of the anterior or posterior branch, respectively.

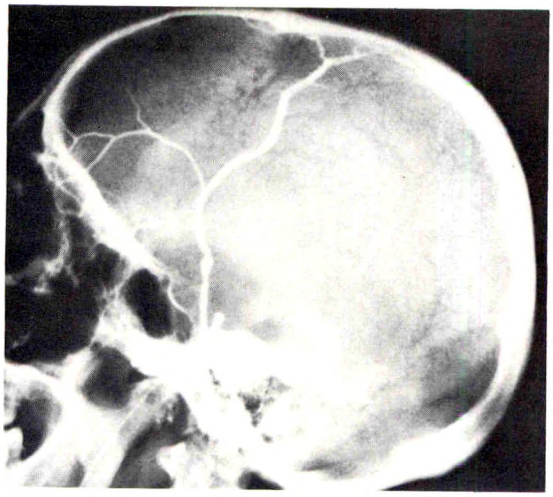


FIG. 6. Inadvertent injection of a temporal vein. It is identified by its general configuration and the presence of valves.

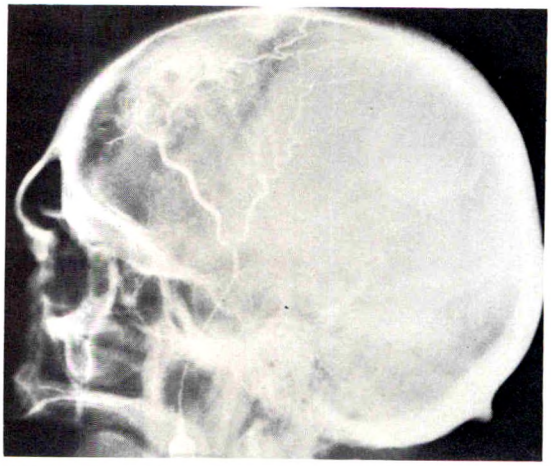


FIG. 8. Superficial temporal arteriography demonstrating vascular supply of metastatic bronchogenic carcinoma.

peripheral lesions such as meningioma or vascular malformation.

Of the 20 cases studied, 5 were diagnosed as positive angiographically with 1 of the 5 also having a positive biopsy. These findings are quite similar to those of Elliott *et al.*[4]: of their 34 cases of polymyalgia rheumatica and/or cranial arteritis, 7 had abnormal angiograms, but of these 5 had positive biopsies. In this same series, as reported by Hunder *et al.*[12] 17 of the 34 cases were patients with polymyalgia rheumatica: 2 had abnormal arteriograms and 1 was biopsy positive.

The only other reported series of temporal arteriography is that of Gillanders:[7] 15 patients were suspected of having cranial arteritis, however, 4 had abnormal arteriograms and 5 had abnormal biopsies.

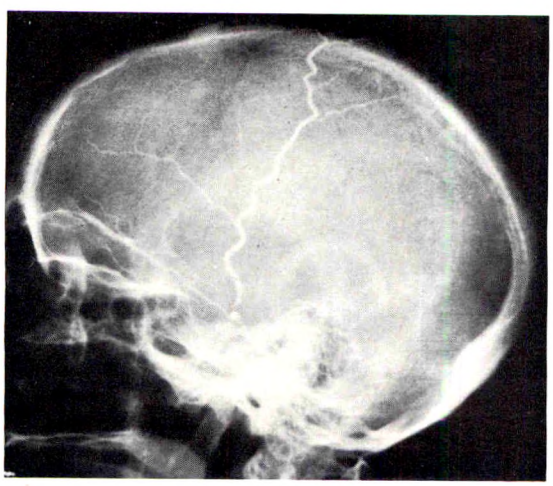


FIG. 7. Injection of contrast material results in a "jet collapse" distal to catheter tip. This is not an area of diseased artery.

CONCLUSIONS

1. Temporal arteriography is abnormal in a substantial per cent of patients with polymyalgia rheumatica.

2. In patients with polymyalgia rheumatica, the incidence of positive temporal arteriograms is greater than the incidence of abnormal temporal artery biopsies.

3. It appears that temporal arteriography may be a more sensitive criterion of cranial arteritis than a random temporal artery biopsy. When an arteriographic abnormality in the distal artery is seen, an external marker can be placed at this site as a guide for an optional biopsy and tissue confirmation.

4. It is therefore recommended that both biopsy and temporal arteriography be done in cases where cranial arteritis is suspected.

5. No complication of temporal arteriography has been encountered.

SUMMARY

Twenty patients suspected of having vascular involvement in polymyalgia rheumatica were studied by temporal arteriography and temporal artery biopsy. This series reveals a substantial number of angiographically positive cases of arteritis, while biopsy provided only 1 abnormal case.

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REFERENCES

1. Barber, H. Myalgia syndrome with constitutional effects: polymyalgia rheumatica. *Ann. Rheumat. Dis.*, 1957, *16*, 230–237.
2. Birdhead, N. C., Wagener, H. P., and Shick, R. M. Treatment of temporal arteritis with steroids: results in 55 cases in which lesion is proved at biopsy. *J.A.M.A.*, 1957, *163*, 821–827.
3. Doumanian, H. O., and Amplatz, K. Vascular jet collapse in selective angiocardiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 344–352.
4. Elliott, P. D., Baker, H. L., and Brown, A. L. Superficial temporal artery arteriogram. *Radiology*, 1972, *102*, 635–638.
5. Gilmour, J. E. Pathology of temporal arteritis. *J. Path. & Bact.*, 1941, *53*, 263–277.
6. Gillanders, L. S., Straghan, R. W., and Blair, D. W. Temporal arteriography. *Ann. Rheumat. Dis.*, 1969, *28*, 267–269.
7. Gillanders, L. S. Temporal arteriography. *Clin. Radiol.*, 1969, *20*, 149–156.
8. Hamilton, C. R., Shelley, W. M., and Tumultym, P. A. Giant cell arteritis: including temporal arteritis and polymyalgia rheumatica. *Medicine*, 1971, *50*, 1–27.
9. Hamrin, B., Jonsson, N., and Landberg, T. Arteritis in polymyalgia rheumatica. *Lancet*, 1964, *1*, 397–402.
10. Horton, B. T., Magath, T. B., and Brown, G. E. Undescribed form of arteritis of temporal vessels. *Proc. Staff Meet. Mayo Clin.*, 1932, *7*, 700–701.
11. Hunder, G. G., Disney, T. F., and Ward, L. E. Polymyalgia rheumatica. *Mayo Clin. Proc.*, 1969, *44*, 849–875.
12. Hunder, G. G., Baker, H. L., Rhoton, A. L., Sheps, S. G., and Ward, L. E. Superficial temporal arteriography in patients suspected of having temporal arteritis. *Arth. Rheumat.*, 1972, *15*, 561–570.
13. Palm, E. Ocular crisis of temporal arteritis syndrome. *Acta. ophthal.*, 1958, *36*, 208–243.

ARTIFICIAL SLOW-FLOW CAROTID ANGIOGRAPHY WITH TUMORS OF THE GLOMUS JUGULARE AND OF THE CAROTID BODY*

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IN 2 previous papers the author has reported on the application of a new technique, artificial slow-flow carotid angiography.[1,2]

It consists of percutaneous carotid injection of the contrast medium with digital compression of the common carotid artery below the site of puncture.

This method was called proximal compression angiography (P.C. angiography) or artificial slow-flow angiography, one of its properties being the slow transport of the contrast medium injected.

The technical details, properties and contraindications have been extensively reported before,[1] the present JOURNAL[2] included.

Briefly summarized, the procedure is performed as follows: After digital occlusion of the common carotid artery, 8 ml. of 60 per cent urografin is injected. When 3 ml. is left in the syringe, the first film is exposed, followed by 2 other exposures at intervals of 1.5 sec. After the third film has been exposed, the digital compression is released. The most reliable criterion of effective proximal occlusion of the common carotid artery is the visualization of the internal and/or the external carotid arteries on all 3 films.

With adequate experience the total duration of digital compression does not exceed 8 sec. The procedure is carried out under general anesthesia. Contraindications are untoward baroreceptor responses or electroencephalographic abnormalities following compression of the carotid artery, and abnormalities in the routine angiograms always made before, such as an already existing diminished flow rate, arterial spasm, signs of intracranial vascular disease or injection of contrast medium in the wall of the carotid artery (a considerably reduced risk when using a plastic intra-arterial cannula).

MAIN FEATURES OF THE P.C. CAROTID ANGIOGRAMS

Distal to the digital occlusion of the common carotid artery, blood pressure and blood-flow are reduced. Apart from the resulting slow transport of the contrast medium through the internal and external carotid arteries, the angiograms show denser vascular opacification than with routine angiography.

Various but unpredictable flow types have been observed with P.C. angiography. In most instances the transport of contrast medium is reversed in either the internal or external carotid arteries. If transported through the internal carotid artery, the intracranial arteries are rarely stained in all 3 films (3 seconds): frozen appearance. In most cases, however, the contrast medium within the carotid siphon is washed out by collateral circulation through the circle of Willis resulting in opacification of some of the intracranial branches. The transport through these branches depends on the flow rate of collateral circulation. It usually is slower than in the routine angiograms, so that the P.C. angiograms remain entirely arterial during the total exposure time of 3 seconds.

In rare instances, collateral circulation is rapid enough for producing contrast medium filling of some intracranial veins in the third P.C. angiogram.

Such effects of collateral circulation are never observed in the flow patterns of the contrast medium through the external carotid arteries and its branches in the P.C. angiograms. *The opacification of veins in the supply area of the external carotid artery with P.C. angiography is never seen unless in the presence of arteriovenous anomalies.* This

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fact is of great importance with regard to the subject of the present paper.

ANGIOGRAPHIC SUBTRACTION WITH P.C. ANGIOGRAPHY

Often the anterior and posterior cerebral arteries, when visualized in the routine carotid angiograms, do not fill with contrast medium in the P.C. angiograms. This suppressed filling was termed "angiographic subtraction."[2] Angiographic subtraction may result in better visualization of vessels which do transport the contrast medium.[1,2] Angiographic subtraction is also seen in the territory of the external carotid artery. The angiographic subtraction phenomena may reveal special properties in the presence of pathologic processes, such as arteriovenous aneurysms, carotico-cavernous fistulae and meningiomas, whose vascular territories have a lower impedance than that of the other intra- or extracranial areas. In such instances the differences in impedance are accentuated in the P.C. angiograms by preferential filling of those pathologic areas, whereas the other vascular territories are poorly opacified or not at all.

The angiographic subtraction phenomena also help to evaluate whether arteriovenous aneurysms are dependent upon one or more vascular territories.[2]

"ANGIOGRAPHIC ADDITION" WITH P.C. ANGIOGRAPHY

A common property of all P.C. angiograms is the denser opacification of the internal and/or external carotid arteries and of some or all of their branches.

Other angiographic additional phenomena are visualization of vessels not demonstrated in the routine angiograms and opacification of more extensive tumor patterns than seen in the routine angiograms. Particular additional phenomena may be observed with arteriovenous shunts where features of venous drainage visualized in the successive routine angiograms together with the arterial supply may be observed in 1 of the 3 P.C. angiograms.

So far we have not reported on P.C. angiography with tumors of the glomus jugulare and of the carotid body. Our findings with 5 tumors of the glomus jugulare and 6 tumors of the carotid body, 3 of them associated with glomus jugulare tumors, are described in the present paper.

TUMORS OF THE GLOMUS JUGULARE (5 cases)

In 1 case of tumor of the glomus jugulare the contrast medium had been injected into the internal carotid artery for routine angiography, whereby no tumor pattern was visualized. P.C. angiography with the needle point in the same place as with the preceding routine angiography resulted in contrast medium filling of both the external and the internal carotid territories and visualization of tumor patterns of the glomus jugulare and of the carotid body (Fig. 1, *A* and *B*). The presence of both tumors was verified at operation. Differences in impedance between the various flow areas, tumoral and nontumoral becoming more apparent with P.C. angiography, may be the explanation of these findings. A similar overflow from contrast medium injected into the internal carotid artery towards the external carotid artery has been observed in P.C. angiograms of meningiomas supplied by the external carotid artery.[1]

In 4 cases the tumor patterns were visualized in the routine angiograms. The contrast medium had been injected into the common carotid artery in 3, and in the internal carotid artery in 1 case. The position of the needle was unchanged for P.C. angiography.

ANGIOGRAPHIC ADDITIONAL PHENOMENA IN THE P.C. ANGIOGRAMS

The presence of a tumor pattern in the routine and P.C. angiograms in 4 tumor cases allowed demonstration of the following additional phenomena in the P.C. angiograms:

a. *More detailed visualization of arterial feeders* due to denser opacification of the ves-

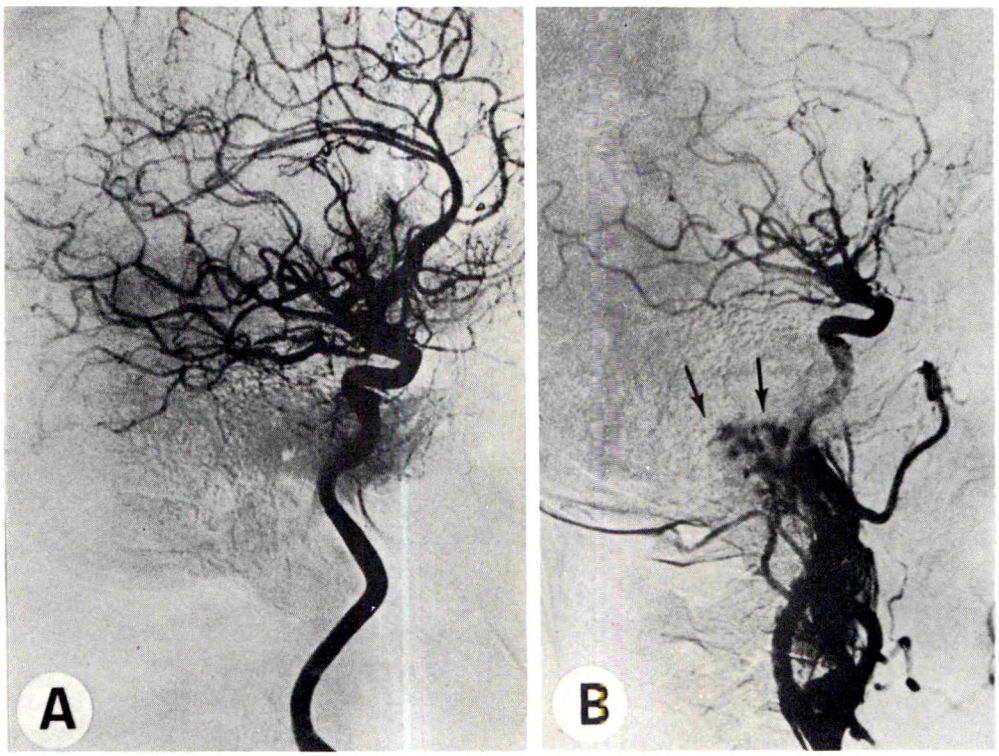


FIG. 1. Tumor of the glomus jugulare. (*A*) Subtraction of routine internal carotid angiogram does not show any abnormality. (*B*) Subtraction of P.C. (proximal compression) angiogram with needle point at same site as in *A* shows filling of both the internal and external carotid arteries and demonstrates large tumor pattern in the cervical and mastoid areas (arrows). Note area of great density at the level of the carotid bifurcation.

sels and slow transport of the contrast medium. This increase in detail information was not the same in all 4 cases but much pronounced in 2 of them (Fig. 2*B*; and 3*B*).

b. *More extensive visualization of the tumor pattern* was observed in three patients (Fig. 3; and 5). The only other tumor type in which such a phenomenon was observed in P.C. angiograms was, in our experience, the meningioma. With the fourth tumor of the glomus jugulare the extent of the tumor patterns in the routine and P.C. angiograms was equal. In all 4 cases the P.C. angiograms showed that the contrast medium was arrested in the internal carotid arteries, no intracranial circulation being visualized (angiographic subtraction).

c. *Demonstration of venous outflow from tumors of the glomus jugulare in P.C. angiograms.* As mentioned before, transport of the contrast medium through the external carotid artery during P.C. angiography is very slow and *never* results in staining of veins unless in the presence of arteriovenous anomalies. This particular property results in interesting findings with P.C. carotid angiography, not only with arteriovenous aneurysms in the territory of the external carotid artery but with tumors of the glomus jugulare.

In 2 of the 5 cases of tumors of the glomus jugulare the P.C. angiograms showed a much more extensive venous drainage pattern than seen in the routine angiograms. Also, these veins showed denser staining and displayed properties of slow flow. In 1 of these cases venous drainage was already demonstrated in the first of the 3 P.C. angiograms; *i.e.*, in the intracranial dural sinuses (base view of the cranium, Fig. 4*B*), while venous drainage in the cervical

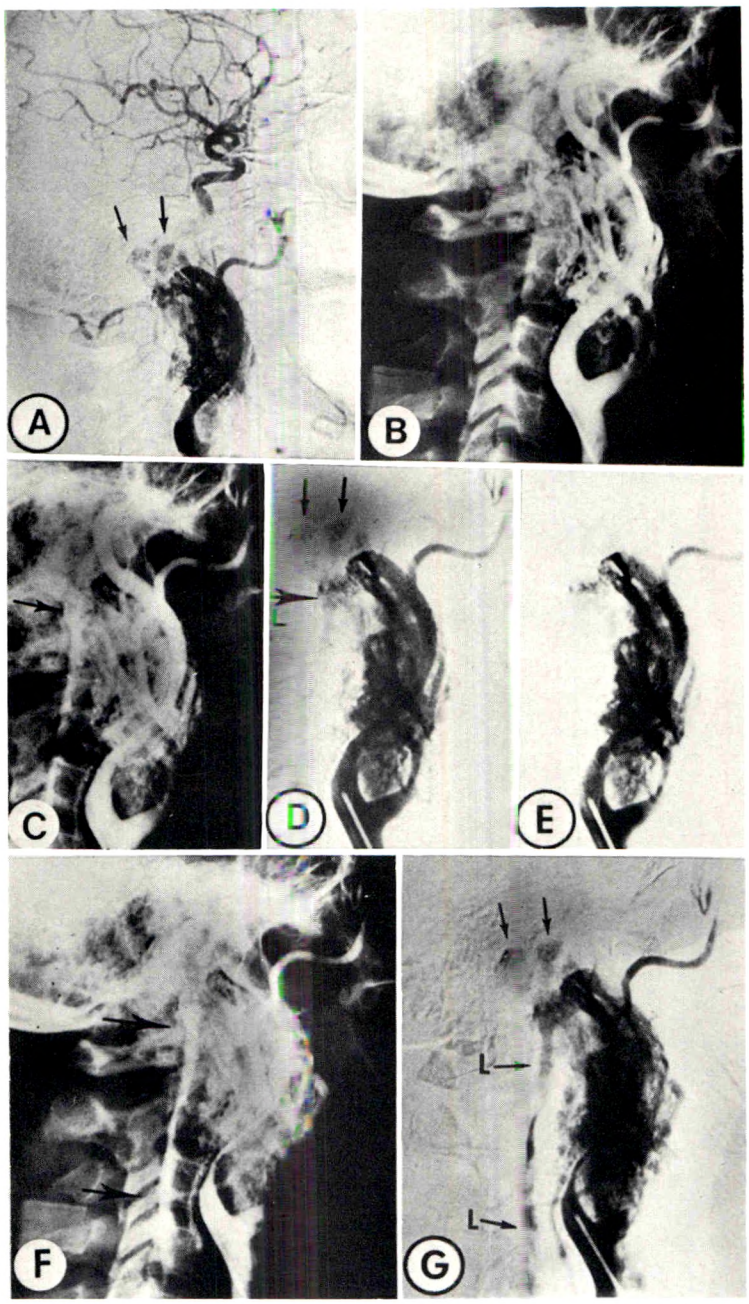


FIG. 2. (*A*) Subtraction of routine angiogram with needle point in internal carotid artery shows opacification of large tumor pattern extending into the mastoid (arrows). Tumor at the bifurcation faintly stained. (*B*) First P.C. angiogram presents more detailed visualization and denser staining of arteries (angiographic addition), supplying the tumor of the glomus jugulare. The contrast medium is arrested in the internal carotid artery. Faint staining of tumor of the carotid body. (*C* and *F*) Second and third P.C. angiograms with 1.5 second intervals show dense staining of patterns of tumors of the glomus jugulare and of the carotid body. Venous drainage of the tumor visualized in the longitudinal epidural sinuses (arrows).

Apart from better angiographic detail information the preservation of the image of the skeletal structures allows information about spatial relationships of the abnormalities, and easy identification of draining veins.

The consequence of the smaller useful density range of paper than that of photographic film is shown in *D* and *E* (photographic subtractions from *C*), where different processing was needed for demonstrating venous drainage and intramastoid tumor pattern (*D*) and detail of cervical tumor pattern (*E*). (*G*) Subtraction of *F*. Arrow L points to longitudinal epidural sinus. The subtractions *D* and *G* show tumor pattern in the mastoid area (arrows) not seen in the P. C. angiograms.

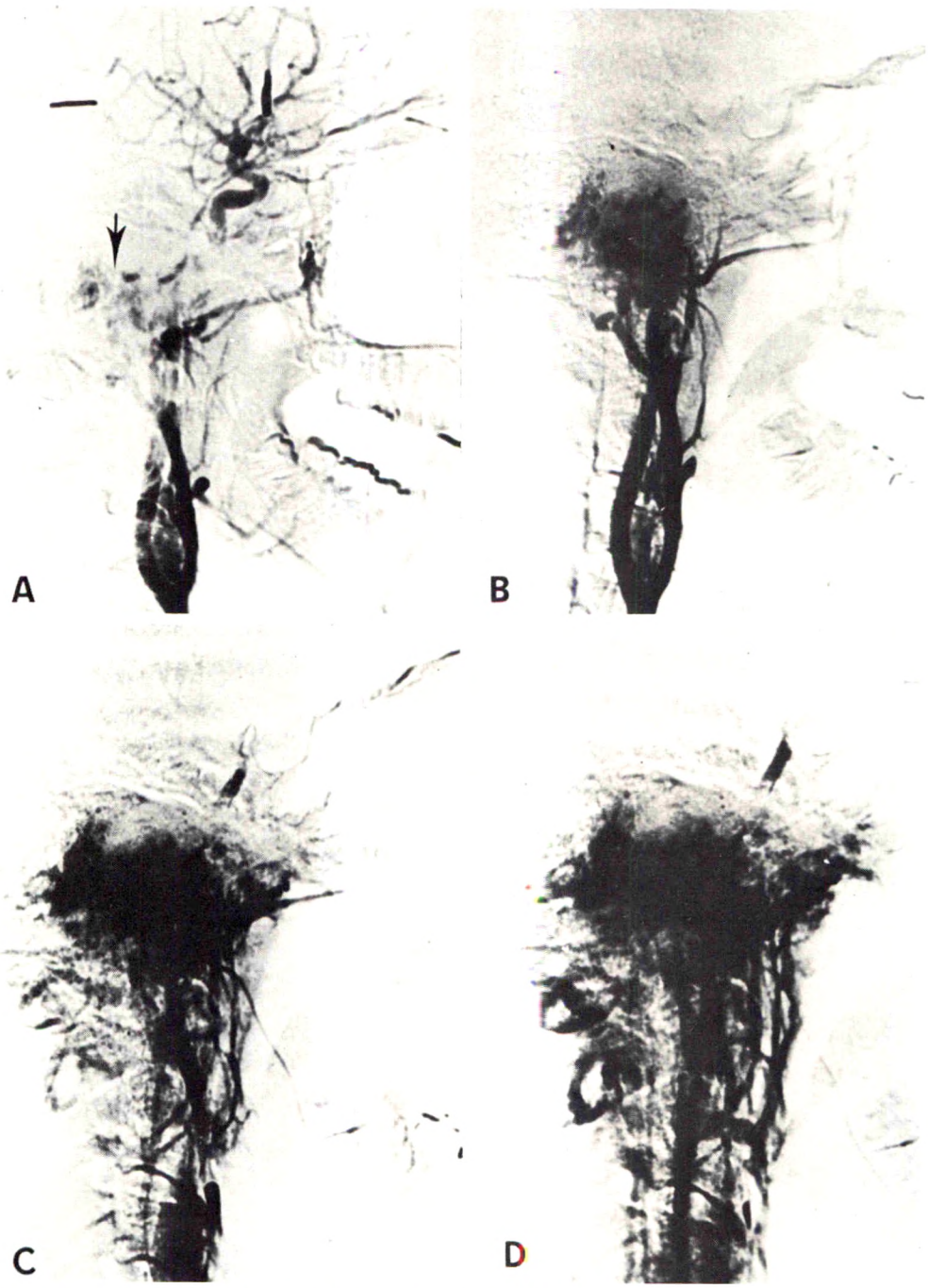


FIG. 3. (*A*) Subtraction of routine common carotid angiogram shows pattern of tumor of carotid body and of glomus jugulare (arrow). (*B–D*) Subtractions of P. C. angiograms with 1.5 second intervals show denser and more extensive staining of tumor patterns, more detailed staining of supplying vessels in *B* and extensive venous drainage in *C* and *D*.

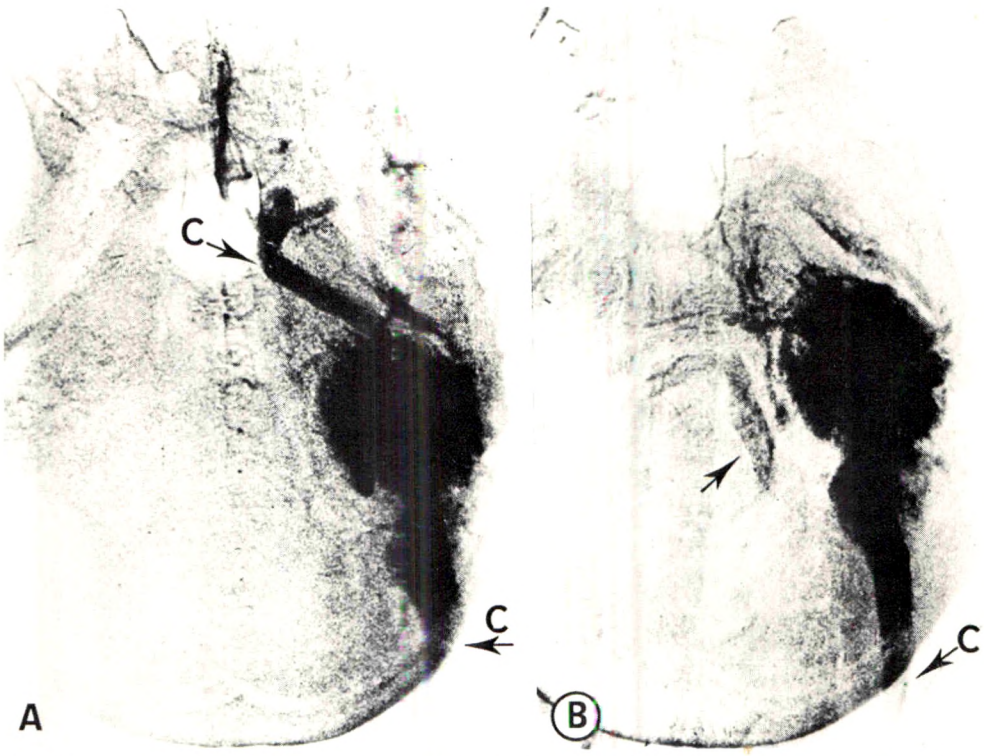


Fig. 4. Same case as in Figure 3. Base views of the cranium. (*A*) Subtraction of common carotid angiogram shows tumor pattern in mastoid area and staining of intracranial arteries. C = carotid artery. (*B*) Subtraction of first P.C. angiogram. Angiographic subtraction of the intracranial carotid artery results in a good view of venous drainage, which is more extensive than in the routine arteriogram. Arrow points to venous plexus of foramen magnum.

areas appeared in the second and third P.C. angiograms (Fig. 3, *C* and *D*).

In the other patient the examination was limited to anteroposterior and lateral views showing extensive drainage through the cervical veins (Fig. 2, *C–G*), in the second and third P.C. angiograms. In 2 cases venous drainage was observed in the third P.C. angiogram and in 1 case the P.C. angiograms did not visualize any venous drainage at all.

Since the transport of contrast medium through the internal carotid artery was blocked in 4 cases, while in the fifth it did not enter intracranial veins during the procedure, the venous drainage demonstrated in the P.C. angiograms originated from the tumor.

The differences in amounts of draining veins visualized in the individual cases probably depended upon the share of arteriovenous shunts in the tumor circulation, which is not equal in all cases.

Differentiation between accessory venous drainage due to occlusion of the jugular bulb and venous drainage from the tumor proper. Unusual venous drainage observed in routine common carotid angiograms in cases of tumors of the glomus jugulare may be due to arteriovenous shunts inside the tumor and/or to accessory drainage of the intracranial circulation as a consequence of occlusion of the jugular bulb.

While P.C. angiography may demonstrate and accentuate the venous drainage of the tumor proper (Fig. 2; 3; and 4), the accessory venous drainage due to blocking of the jugular bulb is well visualized in the subtraction pictures of the phlebographic phases of routine angiograms with injection of the contrast medium into the internal carotid artery

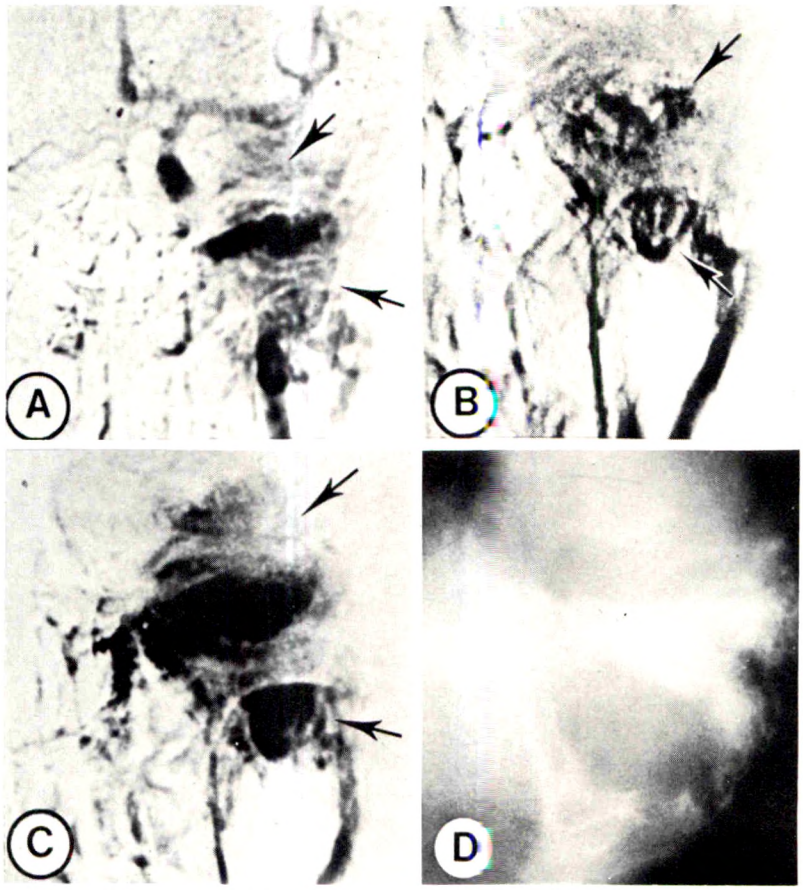


Fig. 5. (*A*) Subtraction of anteroposterior view of routine carotid angiogram shows tumor pattern of glomus jugulare tumor, only part of it densely stained. Arrows point to less opacified areas of tumor, more densely stained in P.C. angiograms *B* and *C* (3 second interval). (*D*) Frontal polytomogram showing cavity produced by the tumor in the mastoid area.

(Fig. 6, *A–C*) provided the internal carotid artery does not participate in the tumor supply.

ANGIOGRAPHIC SUBTRACTION PHENOMENA IN THE P.C. ANGIOGRAMS

In 4 cases the intracranial vessels were not stained. The contrast medium was injected in the common artery in 3 and in the internal carotid artery in 1 case (Fig. 2; 3; 4; and 5).

In 1 case the contrast medium was not arrested in the internal carotid artery (Fig. 1). Since the intracranial circulation and that of the glomus tumor usually do not overlap in the profile and anteroposterior views, the importance of angiographic subtraction with tumors of the glomus jugulare is limited to views where both circulations may project upon each other, such as the base view of the cranium (Fig. 2).

TUMORS OF THE CAROTID BODY (6 cases)

In 3 patients these tumors were associated with tumors of the glomus jugulare. In 1 of them it formed a part of a large continuous glomus tumor. At operation it was found to involve the jugular bulb, and to be intimately connected to and surrounding the ganglion nodosum, its inferior pole reaching downwards unto the bifurcation between the internal and external carotid

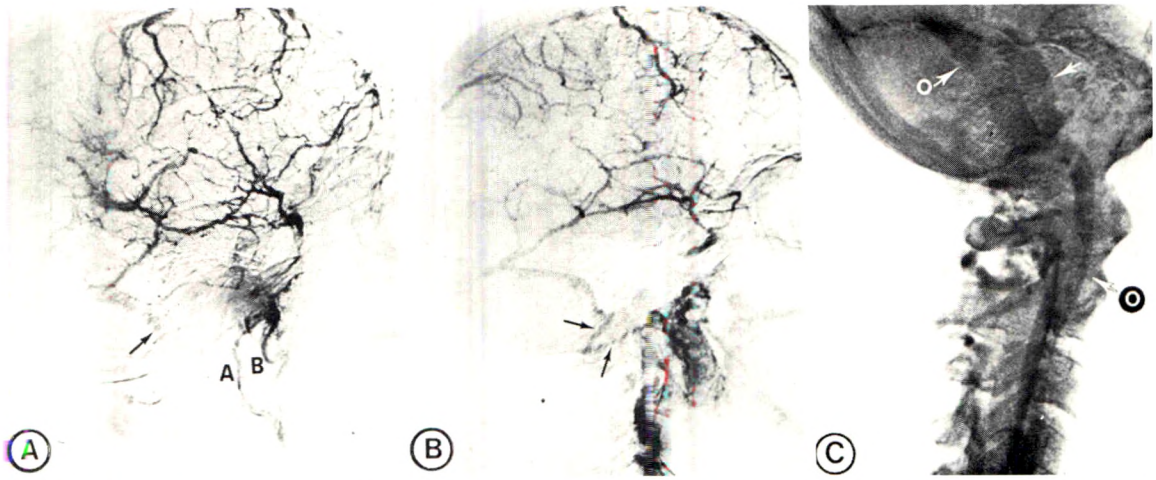


Fig. 6. Subtraction from venous phases of internal carotid routine angiography. Accessory venous drainage as a result of occlusion of jugular bulb or sigmoid sinus by tumor of glomus jugulare in 3 cases: *A*, *B* and *C*.

(*A*) Arrow points to arrest in sigmoid sinus. Posterior venous drainage occurred in the suboccipital triangle (not in picture) and retromandibular (or posterior facial) vein A and palatinel vein B, both ending in anterior facial vein. (*B*) Arrows point to accessory drainage *via* venous plexus of foramen magnum, besides venous drainage through pterygoid plexus. (*C*) ♂ points to intact outflow through right sigmoid sinus and internal jugular vein. Arrow points to left sigmoid sinus. Left jugular bulb occluded. Note extensive accessory venous drainage through postvertebral, basivertebral and vertebral veins.

arteries. The latter portion was supplied by vascular branches from the bifurcation and the external carotid artery.

The whole tumor pattern was visualized in the routine common carotid angiogram. The first P.C. angiogram showed detailed vascular supply to the tumor of the glomus jugulare and the portion surrounding the ganglion nodosum, the tumor of the carotid body being faintly stained. The whole tumor pattern, including that of the carotid body, was densely opacified in the second and third P.C. angiograms. It was impossible to identify the share of the carotid body in the venous drainage (Fig. 2).

The second patient had a small tumor of the carotid body associated with a large tumor of the glomus jugulare, which occupied the mastoid and middle ear. The tumor of the carotid body was diffusely stained in the common carotid angiogram. It was also demonstrated in the first and second P.C. angiograms, the latter showing a denser pattern than the routine angiograms (Fig. 3). As with the first patient, it was impossible to identify the venous drainage of the tumor of the carotid body among the extensive venous pattern originating from the tumor of the glomus jugulare (Fig. 3, *C* and *D*).

In the third patient the routine internal carotid angiogram did not show any supply to the tumor of the carotid body. The latter was visualized together with the tumor of the glomus jugulare in the P.C. angiogram, as a result of return flow of contrast medium from the internal to the external carotid artery (Fig. 1). In this case the supplying branches to the tumor of the carotid body were well visualized due to slow flow and dense opacification of the vessels (Fig. 7). No venous drainage from the tumor of the carotid body was noted in the P.C. angiograms. Since the routine and P.C. angiograms were made with the needle in the internal carotid artery, the usefulness and simplicity of these angiographic procedures for evaluating the share of each of the internal and external carotid arteries in the supply of the tumor is obvious.

Three tumors of the carotid bodies unassociated with glomus jugulare tumors were observed in 2 patients, 1 patient presenting with bilateral tumors. One of the

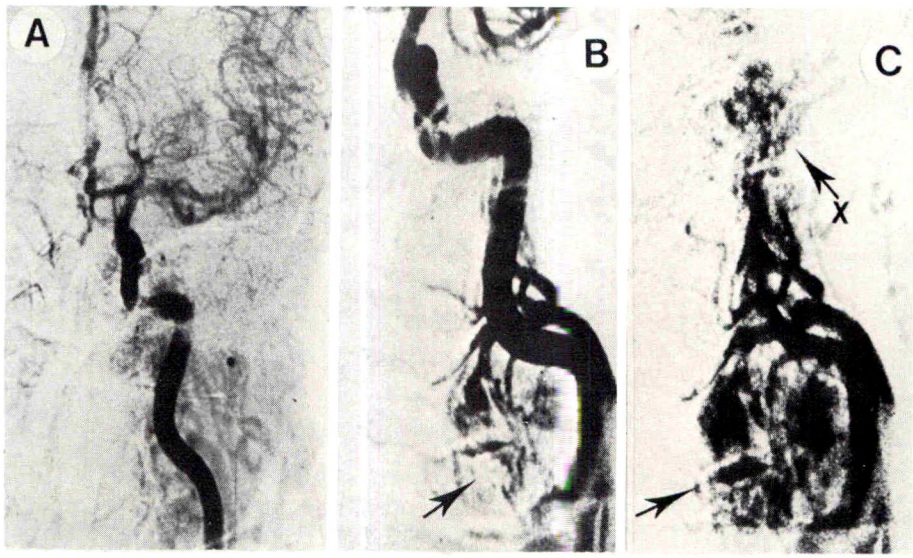


FIG. 7. Same case as in Figure 1. (*A*) Subtraction of anteroposterior view of routine internal carotid angiogram shows no abnormalities. (*B*) Subtraction of early anteroposterior cervical view of P.C. angiogram shows filling of both internal and external carotid arteries with detailed and dense staining of arteries supplying tumor of carotid body (arrow). (*C*) Subtraction of P.C. angiogram 3 seconds later shows reversal of contrast medium in internal carotid artery. Arrow points to dense pattern of tumor of carotid body. Cross and arrow point to pattern of tumor of glomus jugulare.

tumors of the latter patient was small and associated with a glomus tumor extending from the level of C_3 as far as the base of the skull (Fig. 8).

With all 3 tumors the P.C. angiograms showed more detailed visualization of arterial feeders and a denser tumor pattern than visualized in the routine angiograms. In none of the cases was any venous drainage observed (Fig. 8; and 9).

ANGIOGRAPHIC ADDITION IN P.C. ANGIOGRAMS VERSUS PHOTOGRAPHIC OR ELECTRONIC SUBTRACTION OF ANGIOGRAMS

For demonstrating refinements or shortcomings of both angiographic addition and photographic or electronic subtraction, the reproductions of the images in journals are not the ideal means. Problems with reproduction because the useful density range of photographic (and printed) paper, viewed by reflected light, is smaller than that of photographic film, are exemplified in Figure 2, *D* and *E*, both subtractions of *C*, where different processing of the photographic paper was needed for the demonstration of the draining veins and the intramastoid tumor pattern (*D*), and details of the cervical tumor pattern and supplying arteries (*E*).

Also, detail information may be lost with the subtraction of roentgenographic films, presenting great differences in contrast.[3]

In spite of such inevitable imperfections in reproduction, the illustrations of this paper may be clear enough to give support to the following conclusions:

With glomus tumors in the cervical area the advantages of angiographic addition by means of P.C. angiography over routine angiography and its subtractions are: increased density of the tumor pattern and supplying arteries; demonstration of a greater number of supplying arteries and a greater portion of the tumor pattern; and, in some instances, demonstration of a greater number of draining veins in the arterial phase.

Also, the gain in detail information regarding angiographic findings is obtained,

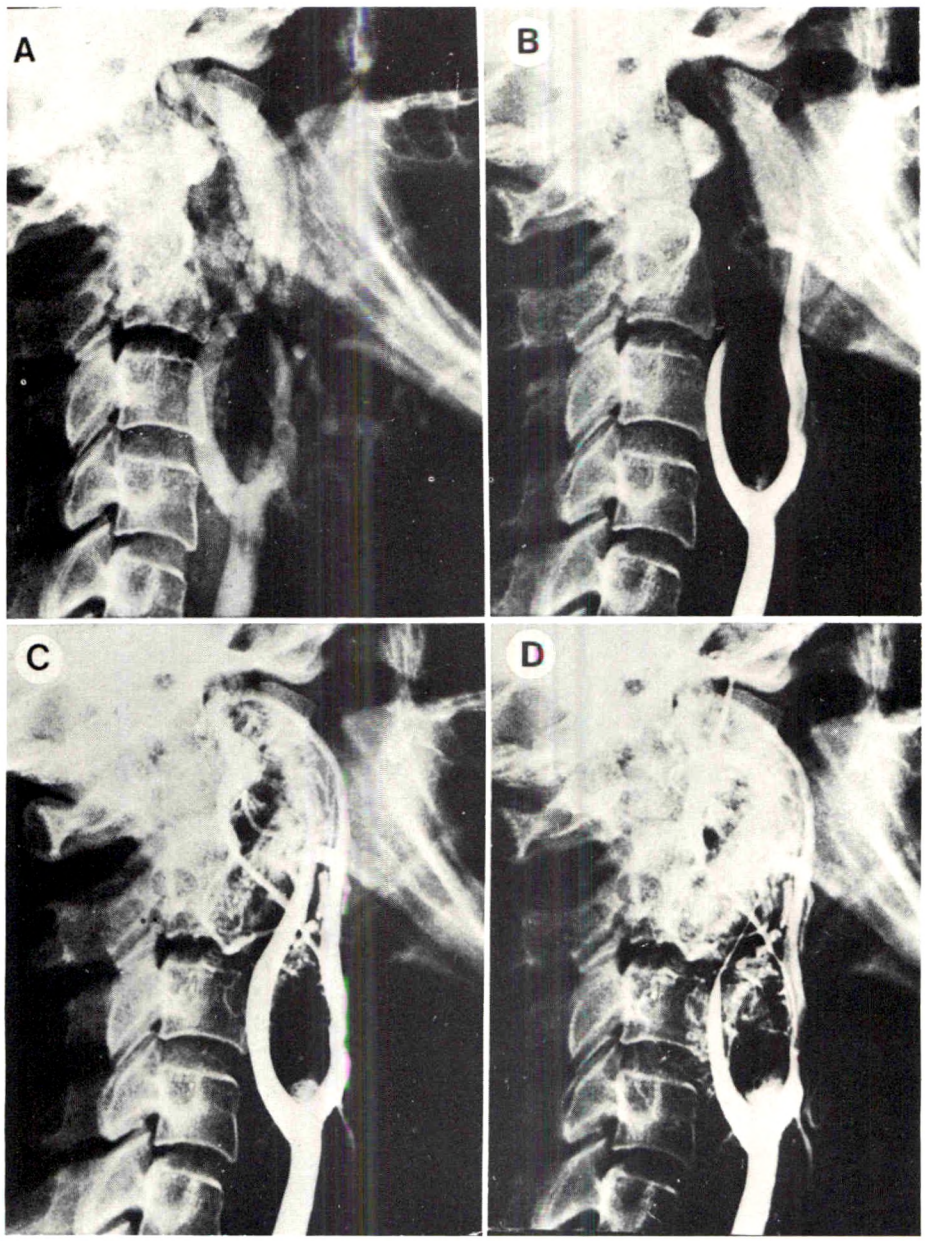


Fig. 8. (*A*) Routine common carotid angiogram shows pattern of right-sided small tumor of the carotid body associated with large glomus tumor in the upper cervical area. (*B–D*) P.C. angiograms with 1.5 second intervals. Arrest of contrast medium in internal carotid artery. Angiographic addition: detailed and denser visualization of supplying arteries and denser tumor patterns. The advantage over photographic subtraction is the preservation of skeletal structures allowing information of spatial relationships. The densest tumor mass is in the upper cervical area. Small separate tumor patterns at bifurcation.

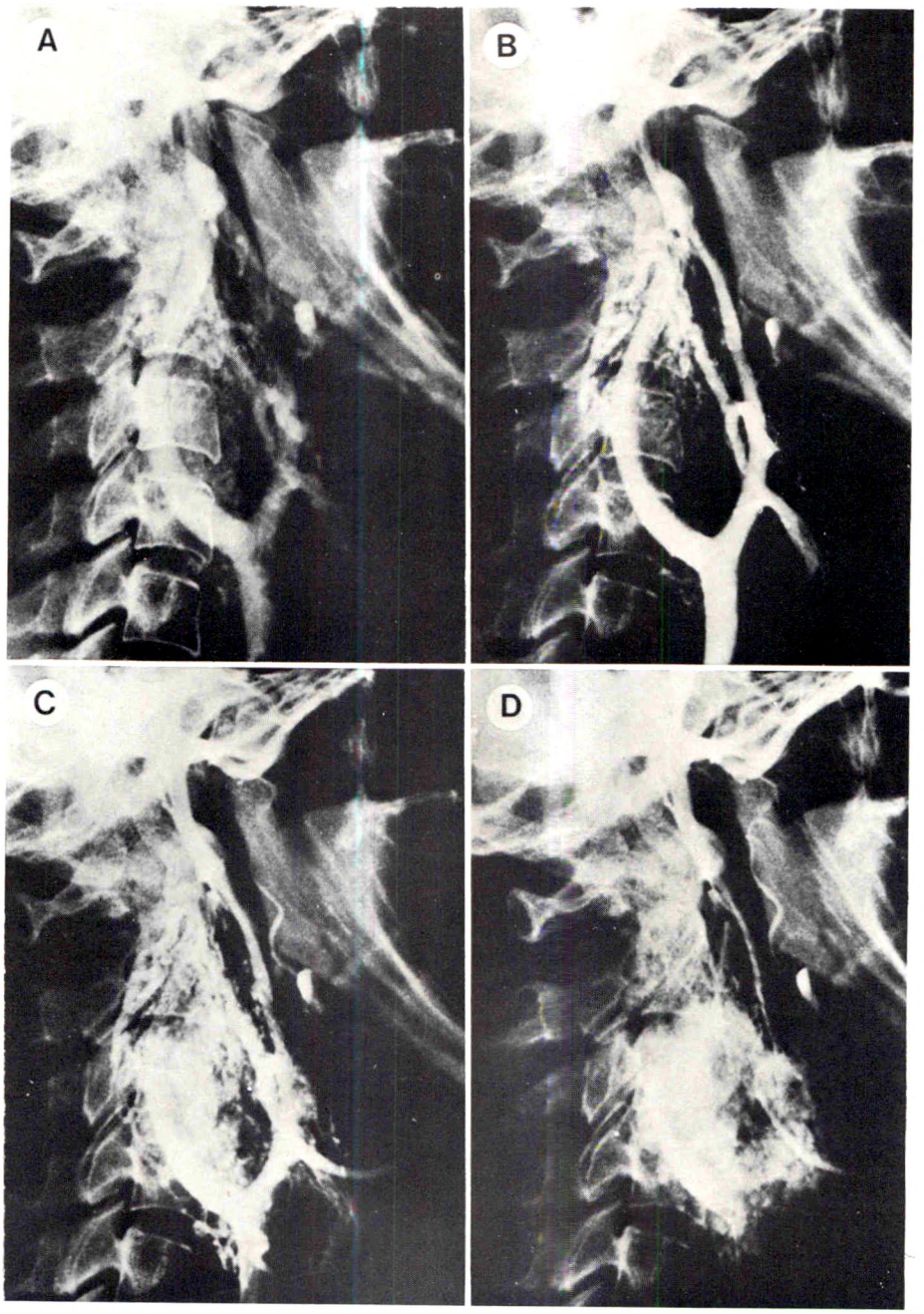


FIG. 9. Same patient, left-sided tumor of carotid body, as in Figure 8. (*A*) Routine common carotid angiogram. (*B–D*) P.C. angiograms with 1.5 second intervals. (*B* and *C*) Densely stained supplying arteries. (*C* and *D*) Dense tumor pattern.

while the image of the cervical structures is preserved—important for estimating spatial relationships of the abnormalities.

In the subtraction pictures the skeletal structures are greatly or completely obliterated. Yet, subtractions of P.C. angiograms play a part since, with their limitations mentioned above, they give an excellent impression of the vascular abnormalities themselves, with the advantages of angiographic addition, while in areas of great density, such as that of the mastoid bone, the subtraction picture may demonstrate tumor patterns not seen in the P.C. angiograms.

Immobilization of the head, essential for subtraction, is more difficult to maintain during P.C. angiography, since the proximal compression of the common carotid artery may produce some head movement.

SUMMARY

P.C. angiograms with tumors of the glomus jugulare supplied by the external carotid artery may show denser and more extensive tumor patterns and denser and more detailed opacification of supplying arteries than seen in routine angiograms.

Gain in detail information with preservation of the image of skeletal structures allows better information about spatial relationships.

Subtractions of P.C. angiograms share the advantage of angiographic addition, but may reveal tumor patterns not seen in areas of great densities in the P.C. angiograms.

Unusual venous drainage in routine angiograms with tumors of the glomus jugulare may be the result of occlusion of the jugular bulb or originate from the tumor proper.

Venous outflow in the territory of the external carotid when visualized in P.C. angiograms, originates from arteriovenous shunts inside the tumor proper. P.C. angiography accentuates this outflow pattern.

Accessory venous drainage due to blocking of the jugular bulb, can be separately visualized in subtraction pictures of venous phases of selective routine internal carotid angiograms, provided the internal carotid body does not participate in the tumor supply.

P.C. angiograms with tumors of the carotid body display similar angiographic additional phenomena as seen with tumors of the glomus jugulare, with the exception of demonstration of venous outflow. Comparative routine and P.C. angiography with the needle in the internal carotid artery may be a simple method to estimate the share of the internal and external carotid arteries in the vascular supply of tumors of the carotid bodies.

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REFERENCES

1. Verbiest, H., and Dokkum, Th. A. Artificial slow flow carotid angiography with compression proximal to site of injection. *J. Neurosurg.*, 1970, *32*, 180–196.
2. Verbiest, H. Results of artificial slow flow angiography with arteriovenous aneurysms in supply area of external or internal carotid arteries. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 1–15.
3. Ziedses des Plantes, B. G. Subtraction. Georg Thieme Verlag, Stuttgart, 1961.

PITFALLS OF SOFT TISSUE DIAGNOSIS IN THE TYMPANIC CAVITY*

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THE tomographic diagnosis of advanced cholesteatomas of the tympanic cavity usually presents little difficulty.[3,4] They show characteristic erosions of the superior and posterior medial margins of the bony external canal (the scutum), together with varying degrees of ossicular destruction and possibly erosive changes in the aditus and antrum area. On the other hand, serous otitis media and some of the very early cholesteatomas constitute a challenge to early tomographic diagnosis.

Since these early lesions are primarily soft tissue abnormalities, we wish to evaluate some of the factors that influence soft tissue visualization in the tympanic cavity and epitympanic recesses. This study was performed using moist, preserved human temporal bone specimens roentgenographed with hypocycloidal and trispiral tomography.

ANATOMY

The tympanic cavity is slit-like, with a central narrowing. It angulates medially at an angle of approximately 40–50 degrees as it courses inferiorly. It also angulates medially at approximately a 15–20 degree angle as it courses from posterior to anterior.[1] The tympanic cavity is widest superiorly (approximately 6 mm.) and narrowest in its mid-region (approximately 2 mm.), whereas inferiorly it is approximately 4 mm. in transverse diameter. The volume of the tympanic cavity is approximately 0.9 cc.

The medial wall of the tympanic cavity is comprised of those structures enclosed in the optic capsule, namely, the cochlea, anteriorly with its basal turn forming the promontory, and the vestibular apparatus posteriorly. The walls of the vestibule, including the oval window, comprise a significant portion of the medial wall, while the lateral or horizontal semicircular canal forms the superomedial wall of the tympanic cavity and epitympanic recess. The lateral wall of the tympanic cavity is comprised mainly of the tympanic membrane and a recess (the epitympanic recess) above the tympanic cavity, with its lateral wall being the bony roof of the external auditory canal and a bony partition that together are called the scutum. The tympanic cavity is continuous anteriorly with the eustachian tube and posteriorly through the aditus into the mastoid antrum.

Within the tympanic cavity is the ossicular chain, consisting of the malleus, incus, and stapes. The tendons of the tensor tympani muscle and the stapedius muscle lie within the cavity. The entire cavity is lined with a low-lying mucous membrane that forms folds over the ligamentous attachments of the ossicular chain as well as the tendons. The major arteries and nerves are intimately associated with the bony walls of the tympanic cavity, while the posterior tympanic artery accompanies the chorda tympani nerve in close proximity to the pars flaccida of the tympanic membrane. The chorda tympani enters the posterior aspects of the tympanic cavity through the canaliculus of the chorda tympani in the facial canal. As it passes laterally it arches superiorly and lateral to the long process of the incus and courses medial to the long process of the malleus to exit anteriorly through the petrotympanic fissure.

In considering the soft tissues of the

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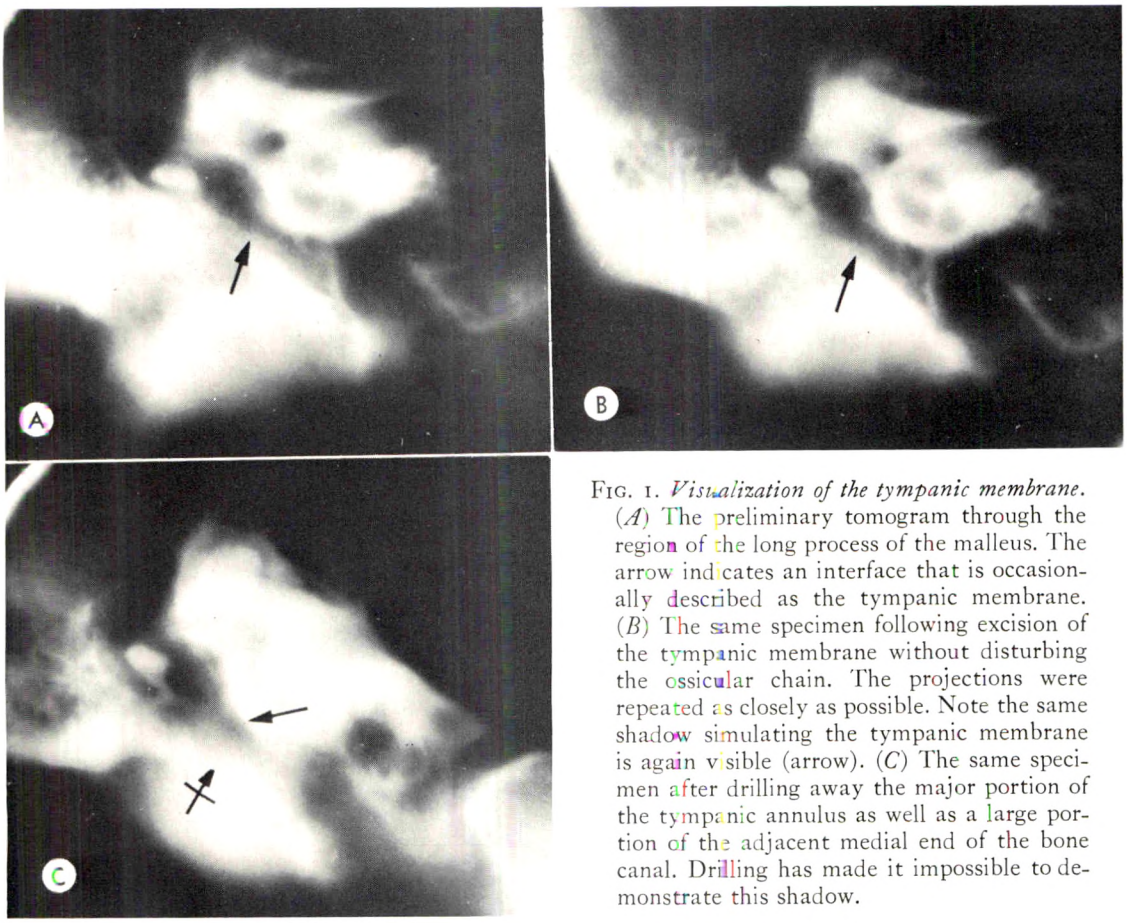


Fig. 1. *Visualization of the tympanic membrane.* (*A*) The preliminary tomogram through the region of the long process of the malleus. The arrow indicates an interface that is occasionally described as the tympanic membrane. (*B*) The same specimen following excision of the tympanic membrane without disturbing the ossicular chain. The projections were repeated as closely as possible. Note the same shadow simulating the tympanic membrane is again visible (arrow). (*C*) The same specimen after drilling away the major portion of the tympanic annulus as well as a large portion of the adjacent medial end of the bone canal. Drilling has made it impossible to demonstrate this shadow.

tympanic cavity, the tympanic membrane is most conspicuous in the depths of the external auditory canal. It is about 0.1 mm. thick and measures about 9.0 mm. in a vertical diameter with 8.0 mm. in anteroposterior diameter. The long process of the malleus is firmly attached in a vertical direction to the midportion of the tympanic membrane called the "umbo." The midportion of the drum bows medially due to the attachment of the malleus and the tensor tympani tendon. The tympanic membrane bears an important relationship to a ring of bone or tympanic annulus that contains a central sulcus for the attachment of the periphery of the tympanic membrane (Fig. 1, *A–C*). Superiorly the sulcus runs out into small points which are called the greater or lesser tympanic spines. The tympanic annulus is composed of dense cortical bone and protrudes for approximately 1.0 mm. into the lumen of the medial end of the external auditory canal.

Of the two muscles lying within the tympanic cavity, the tensor tympani is the larger, measuring approximately 2.0 cm. in length. It originates from the cartilaginous portion of the eustachian tube and courses posteriorly in the semicanalis m. tensor tympani immediately above the eustachian tube at the anterior angle of the petrous bone. Its tendon is at right angles to the belly of the muscle coursing around the cochleariform process of the medial wall of the tympanic cavity. The tendon inserts into the malleus at the junction of the neck and long process (Fig. 2, *A–C*). The tendon, through its action, pulls the malleus and tympanic membrane medially. It serves to dampen excursions of the tympanic mem-

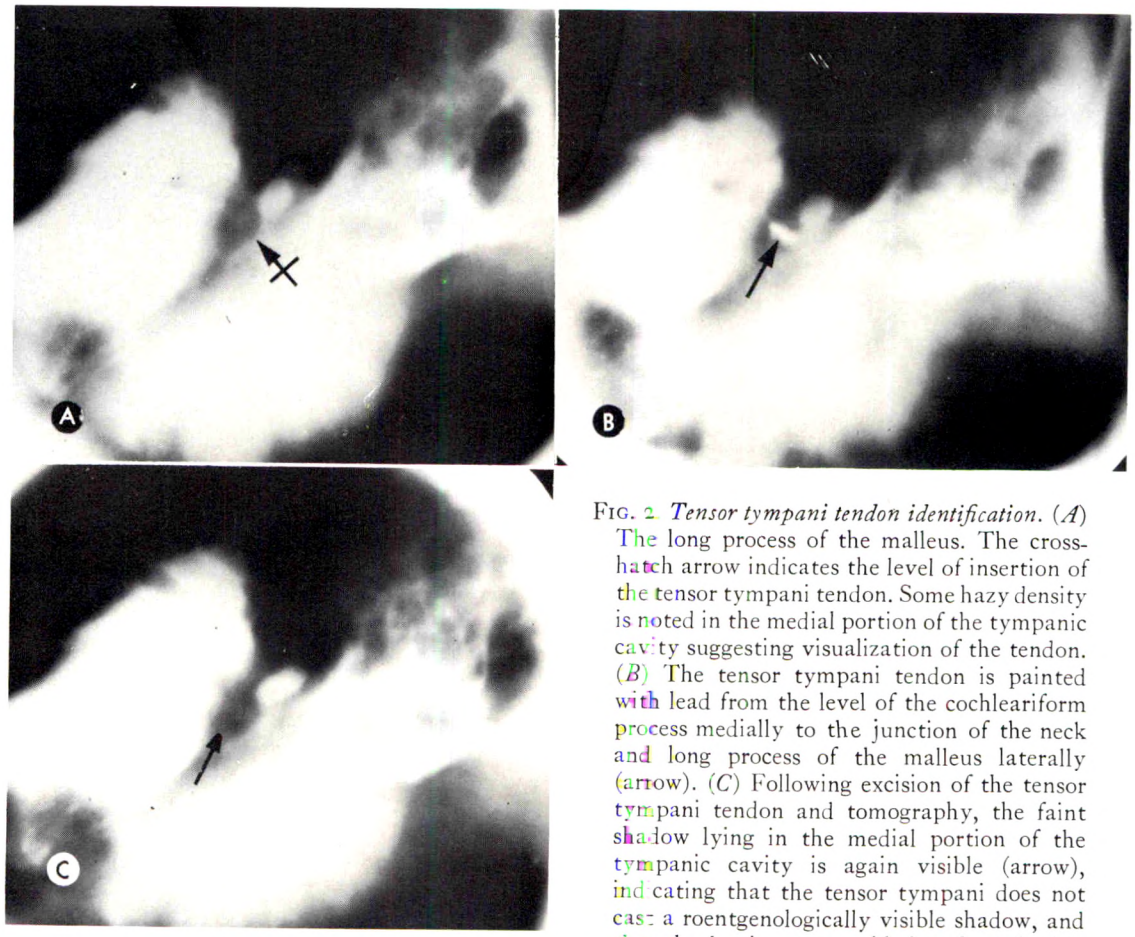


FIG. 2. *Tensor tympani tendon identification.* (*A*) The long process of the malleus. The cross-hatch arrow indicates the level of insertion of the tensor tympani tendon. Some hazy density is noted in the medial portion of the tympanic cavity suggesting visualization of the tendon. (*B*) The tensor tympani tendon is painted with lead from the level of the cochleariform process medially to the junction of the neck and long process of the malleus laterally (arrow). (*C*) Following excision of the tensor tympani tendon and tomography, the faint shadow lying in the medial portion of the tympanic cavity is again visible (arrow), indicating that the tensor tympani does not cast a roentgenologically visible shadow, and that the density presumably is a ghost shadow of bony structures such as the stapes and possibly part of the ponticulus.

brane. This tendon is approximately 1.0 mm. in diameter and lies almost parallel to the coronal plane of the skull. It angulates from above downward at approximately 15 degrees with the horizontal plane. Approximately 2.0 mm. of this tendon is embedded in a groove in the cochleariform process and 3.0 mm. lie free in the tympanic cavity.

The stapedius muscle is the smallest of all muscles and takes origin inside a volcano-shaped cone lying on the posterior inferior wall of the tympanic cavity. This cone is immediately anterior to the inferior portion of the posterior genu of the facial canal. At times the lumen of the facial canal communicates with the lumen of the cone-shaped pyramidal eminence. The tendon of the stapedius muscle is approximately 2.0 mm. in length by 0.3–0.4 mm. in diameter. It leaves the pyramidal eminence to course directly anterior to insert in the neck of the stapes adjacent to the stapedial-incudal joint (Fig. 3, *A–D*).

TECHNIQUE

The investigation of soft tissue density was divided into 2 parts: (1) the identification of normal soft tissues that cast shadows in the tympanic cavity; and (2) the simulation of pathologic processes that one would find in serous otitis media and chronic otitis media with cholesteatoma formations.

All investigations were performed on moist human temporal bone specimens roentgenographed with a Philips-Massiot

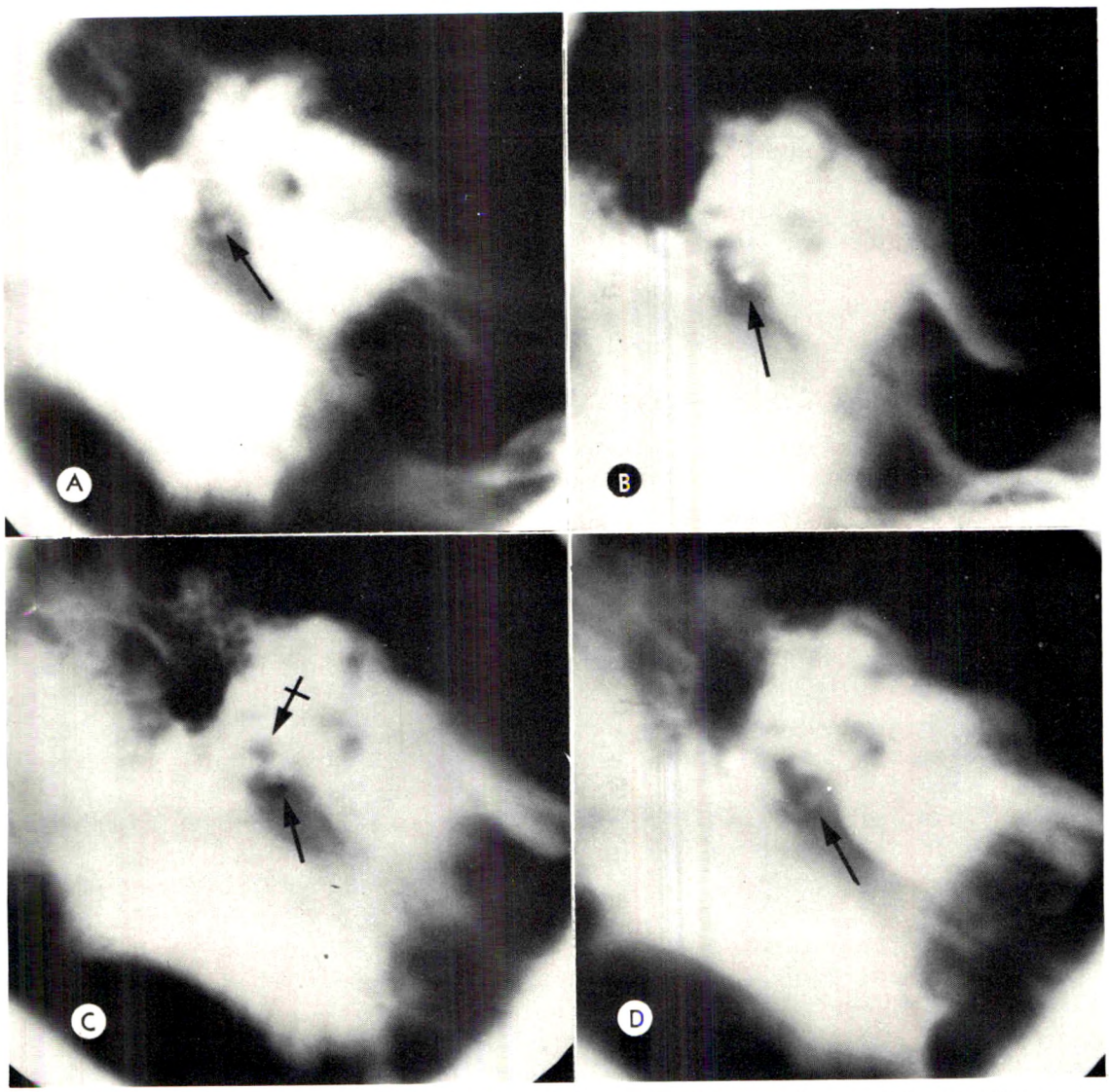


FIG. 3. *Stapedius tendon recognition.* (*A*) A shadow is in the region of the attachment of the stapedius tendon to the neck of the stapes (arrow). (*B*) The stapedius tendon has been painted with lead (arrow). (*C*) By serial section the painted stapedius tendon could be traced to the pyramidal eminence which gives origin to the stapedius muscle (arrow). The pyramidal eminence lies immediately adjacent to the posterior genu of the facial nerve (cross-hatch arrow). (*D*) Following removal of the stapedius tendon, the shadow in the medial tympanic cavity is still visible and is believed to represent the head of the stapes (arrow) and possibly "ghost shadows."

Polytome or a CGR Stratomatic tomographic device. The factors were approximately 35 kv., 15 milliamp, with a hypocycloidal or trispiral movement using a 0.6 mm. focal spot tube giving sections of approximately 1.0 mm. thickness and sections being taken at 1.0 mm. intervals. All manipulations on the temporal bone specimens were performed by removing the tegmen of the epitympanic recess using a Zeiss operating microscope to ensure accuracy of lesion placement with as little disruption of normal anatomy as possible.

The simulated pathologic states could be most accurately visualized in the Guillen projection (transorbital with the head turned 15–20 degrees toward the side under investigation). The anteroposterior projec-

tion, when used, was second-most accurate and basal projections were of limited usefulness.

Normal Structures. For identification of normal structures, a minimum of 3 specimens was used for each investigation. The tendons of the tensor tympani (Fig. 2, *A–C*) and stapedius muscles (Fig. 3, *A–D*) were tomographed before and after painting the tendon with an 87 per cent carbonated lead paint. The tendons were excised and the specimen re-tomographed to see if any shadow suspicious of tendon on the pre-painting film was still present.

The tympanic membrane was investigated by tomography before and after complete removal of the tympanic membrane. In addition, the tympanic annulus was also drilled away, followed by tomography (Fig. 1*C*).

By inspection we felt that the suspensory ligaments of the ossicles were all very short and did not cast shadows in the medial portions of the tympanic cavity. The mucosal folds are exceedingly thin as is the chorda tympani nerve. These shadows were not thought to represent significant densities in view of our results with the tensor tympani and stapedius tendons.

Pathologic States. The pathologic tissues to be simulated were as follows: (1) high protein fluid; (2) cellular debris and sebaceous material; (3) bone destruction and bone productive changes; and (4) granulation tissue. Various materials were roentgenographed and their radiodensities compared to exudative fluid, pus, cholesteatomatous material and excised surgical specimens of cholesteatomas.

Stuart's bacteriologic media was found to be the same density as high protein fluid. In a similar manner, paraffin with a 1 per cent concentration of iodine by weight gave the same densitometric opacification as surgically removed cholesteatomas. Increases of bone density were simulated by utilizing powdered bone meal in a small amount of glue, and fibrotic reactions were simulated by the addition of small bits of fascial tissue from beef. Necrosis and bone destruction could not accurately be portrayed and was simulated by drilling the appropriate areas with dental drills.

RESULTS

Normal Soft Tissue Densities. The positions of the stapedius and tensor tympani tendons could be accurately delineated with the painted specimen and at no time were nonopacified tendons visualized. The tympanic membrane did not cast a shadow. A faint haziness was frequently seen on the specimens, but this was made to disappear by grinding off the tympanic annulus and some of the adjacent bony external canal wall (Fig. 1*C*).

Simulated Pathologic Conditions. When homogeneous solutions such as the 1 per cent iodine in paraffin or the Stuart's bacteriologic media simulating cholesteatomatous cellular debris and high protein fluid, respectively, were utilized, comparison of the pre- and post-injected specimen tomograms showed only a very slight haze to the epitympanic recess and medial portions of the tympanic cavity (Fig. 4, *A* and *B*). This was present in all specimens, but the degree of change was quite minimal.

The placement of bits of fibrous tissue in the epitympanic recess and tympanic cavity again produced a haziness of a slightly greater degree than the 1 per cent paraffin or the Stuart's media specimens. Whether this change would have been sufficient to recognize in a living patient is not known at the present time. Placement of bone meal about the margins of the epitympanic recess and tympanic cavity produced the greatest degree of haze, which increased as more bone was added. We were able to obscure the shadow cast by the stapes, but the malleus and incus remained visible (Fig. 5, *A* and *B*).

The roentgenologic pattern of dense clouding of the epitympanic recess and tympanic cavity was only achieved when the trabeculated bone surrounding these cavities was drilled away and a thick layer of bone dust inserted. This had to be followed by the insertion of fibrous tissue and/

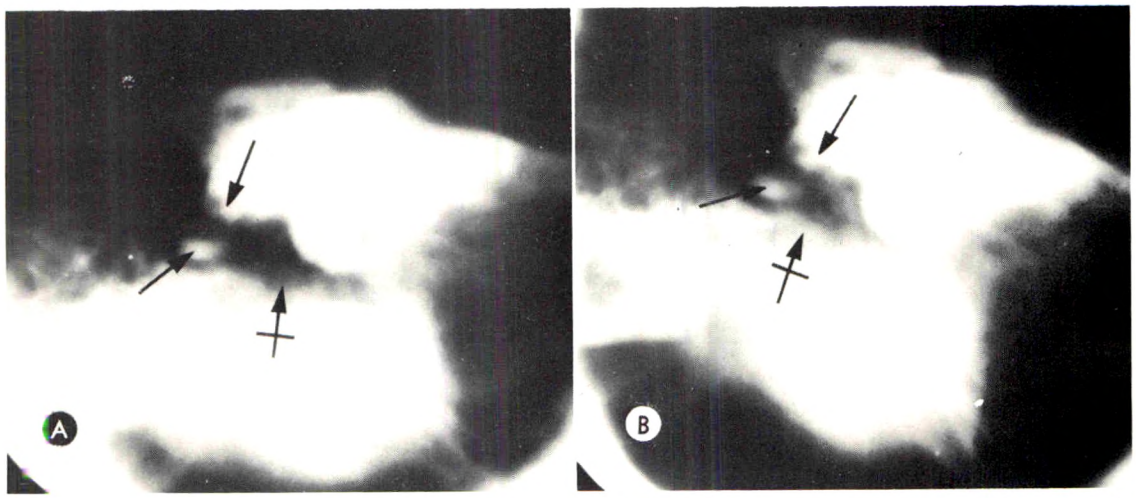


FIG. 4. *Simulated cholesteatoma with 1 per cent iodine in paraffin.* (*A*) Tomography in the pre-injected specimen shows a clear air space around the body of the incus in the epitympanic recess (arrows). The long process of the incus is clearly visible (cross-hatch arrow). (*B*) Following the injection of 0.35 cc. of 1 per cent iodine in paraffin around the ossicles and into the epitympanic recess, and filling of the tympanic cavity (arrows), the long process of the incus is still clearly visible (cross-hatch arrow). There is only a very slight haze to the air space about the ossicles in the epitympanic recess. This change was consistently seen in all of the specimens.

or 1 per cent iodine in paraffin. The insertion of heterogeneous densities into the cavities alone did not produce the characteristic roentgen appearance of cholesteatomas. The bone destruction produced by drilling as well as the bony productive changes simulated by the bone dust appear to be essential to demonstrate the roentgen

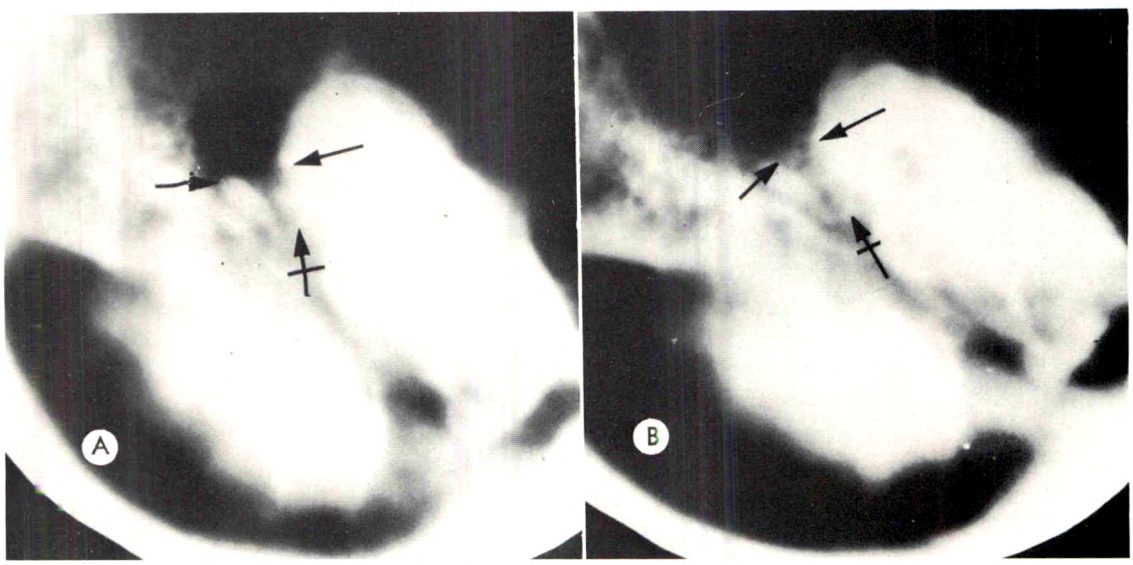


FIG. 5. *Bone dust simulating sclerosis.* (*A*) A clear epitympanic recess (arrows) in the preliminary tomograms. The long process of the incus and the stapes (cross-hatch arrow) is clearly visible. (*B*) Under direct observation through the removed tegmen of the epitympanic recess, bone dust was packed into the walls of the recess without disturbing the ossicular chain. The increase in over-all density gives a hazy appearance to the entire epitympanic recess (arrows). The shadow of the stapes (cross-hatch arrow) is faintly visible. The total picture is similar to but not exactly like a cholesteatoma.

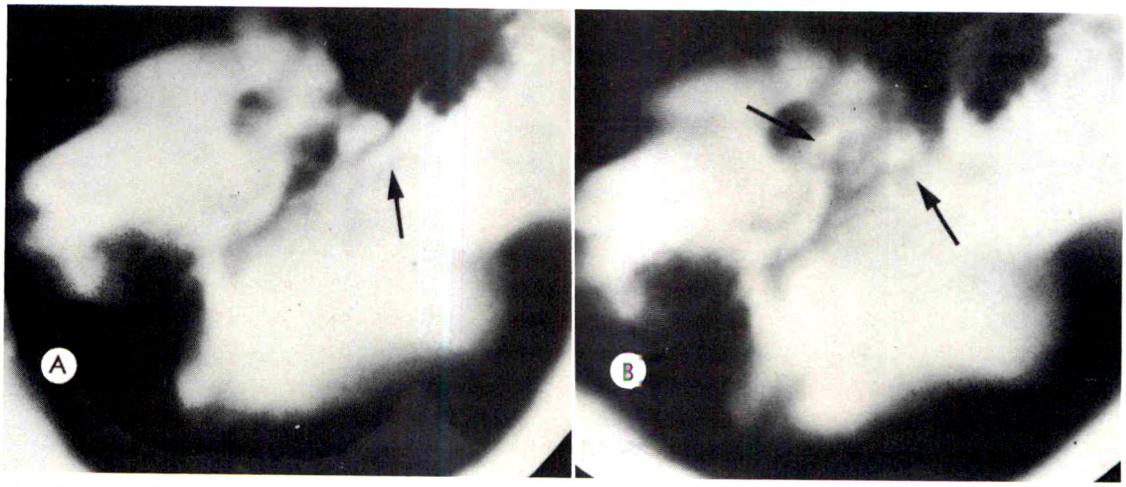


FIG. 6. *Multiple modalities to create a cholesteatoma picture.* (*A*) In the premanipulated specimen, the scutum is sharply outlined (arrow). There is a clear epitympanic recess and medial tympanic cavity. (*B*) Using a dental drill the epitympanic recess was expanded slightly by drilling away all trabecular type of bone: the resultant enlarged cavity was lined with bone dust. The scutum (arrows) was blunted by the drill. Parts of the ossicular chain were thinned with careful drilling. The epitympanic recess and tympanic cavity were packed with fibrous tissue and the intervening spaces filled with 1 per cent iodine paraffin. The combination of the bone destruction (drilling), bone production (bone dust), and heterogeneous opacification within the epitympanic recess and tympanic cavity closely simulates the roentgen picture of cholesteatoma.

appearance of cholesteatoma (Fig. 6, *A* and *B*).

DISCUSSION

Normal Structures. Obviously the validity of our results could be questioned since so much more detail is available in specimens as compared to the living patient. On the other hand, if the structures or pathologic processes cannot be identified in isolated temporal bone specimens, one should be extremely cautious in diagnosing these changes in the living patient.

Attempts were made to identify the normal soft tissues such as tendons and membranes by tomography with the plane of cut parallel to the long axis of the structure as well as perpendicular to the long axis of the structure involved. All too frequently the otic capsule became superimposed on the structure in question, which made any demonstration clearly impossible.

The shadow of the tympanic membrane has frequently been illustrated in the literature.[2–4] There is little question about its location on the roentgenogram since the long process of the malleus rests firmly attached to the umbo of the tympanic membrane and its peripheral attachment to the tympanic annulus is well known.

In viewing anteroposterior tomograms, or Guillen projections, a distinct difference of density is obvious with the tympanic cavity being more radiolucent than the region of the external canal in normal patients. The sharp delineation of these 2 densities could logically be attributed to the tympanic membrane. In over 12 specimens no difference could be detected before and after the removal of the tympanic membrane. It was thought that the tympanic annulus may have cast a shadow so the bony ridge was removed with a bone drill. This did not prove to be sufficient bone removal to alter the shadow that appeared in the region of the tympanic membrane. It was only after large amounts of bone were removed from the region of the medial-most portion of the external auditory canal that a distinct change began to take place about the region of the tympanic

membrane. It would seem logical that the shadow suspected of being due to tympanic membrane is merely the dense bone of the external auditory canal compared to the more cellular and porous bone surrounding the tympanic cavity and mastoid air cell region. This same phenomenon is probably highly significant in the cholesteatoma problem, as will be discussed later.

The cochleariform process is a poorly marginated bony protrusion from the upper portion of the promontory. It serves as the fulcrum for the tensor tympani tendon and accompanies the tendon to some extent into the tympanic cavity. Because of its irregular margins, it blends with surrounding densities to give a suggestion of visualization of the tendon of the tensor tympani muscle. By painting the tendon of the tensor tympani we were able to define how far the cochleariform process projected into the tympanic cavity. Also, following removal of the tendon, the cochleariform process was still visualized as an ill-defined shadow in the medial portion of the tympanic cavity superimposed upon the shadow of the stapes. Tomography demonstrates that this shadow disappears as one approaches the level of the oval window niche.

As best we could determine, none of the arteries or veins about the tympanic cavity could be demonstrated in non-injected specimens. There was no evidence of change following removal of the chorda tympani nerve and its accompanying vessels.

Simulated Pathologic States. At first our findings were quite disturbing in that we were not able to demonstrate a greater degree of change when high protein fluid or 1 per cent iodine in paraffin was instilled into the tympanic cavity. When one considers the over-all density of the bones of the mastoid and the small capacity of the tympanic cavity, these findings become more reasonable. Despite the small volume of air replaced by simulated lesions, we were still able to show some haziness in the epitympanic recess. The similarity in haziness caused by fibrous tissue or by packing bone meal in the wall of the epitympanic recess bears a close parallel to the changes one observes in chronic frontal sinus disease. In sinus disease one frequently sees reactive bone sclerosis about a frontal silus or maxillary antrum that causes an over-all haziness closely simulating a fluid-filled sinus. Since the pathophysiology of the mastoid air cells and the tympanic cavity is quite similar to the paranasal sinuses, one would not be surprised to see reactive bone sclerosis in the adjacent bones when a chronic inflammatory disease process is present in the lumen of the cavity. Similarly, destruction of bone septa and trabeculae with simulated reactive bone sclerosis is essential to reproducing the roentgen changes one sees with cholesteatoma (Fig. 6, *A* and *B*). Our results would confirm that on tomography a great deal of bone destruction as well as production must be present to produce the characteristic roentgen changes of cholesteatoma in the epitympanic recess and mastoid antrum. The material filling the resultant bone cavity must be heterogeneous and combined with bone destruction to produce the loss of detail of the ossicular chain that one sees in the moderately advanced cholesteatoma.

In clinical roentgenology some factors that we were unable to evaluate were the thickening of the tympanic membrane that takes place in chronic ear disease and the calcific plaques that become deposited in advanced tympanosclerosis. Unless the amount of calcium is quite marked, it is presumed that it would act similarly to small amounts of paraffin or the Stuart's bacteriologic media that cast only a faint shadow.

CONCLUSIONS

1. Many of the soft tissue shadows seen "within" the tympanic cavity are in reality bone thickenings anterior or posterior to this cavity casting their shadow in the lumen.

2. Reactive sclerosis and bone destruction are major contributors to the roentgen appearance of cholesteatomas.
3. Transudates and other homogeneous fluids within the tympanic cavity and epitympanic recess produce roentgen changes that are extremely subtle if at all discernible unless accompanied by bone reactive changes.
4. With present techniques the tendons of the tensor tympani muscle and stapedius muscle are not visible on tomography.

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REFERENCES

1. ANSON, B. J., and DONALDSON, J. A. Surgical Anatomy of the Temporal Bone and Ear. Second edition. W. B. Saunders Company, Philadelphia, 1973.
2. BERRETT, A., BRUNNER, S., and VALVASSORI, G. E. Modern Thin Section Tomography. Charles C Thomas, Publisher, Springfield, Ill., 1973.
3. BRÜNNER, S., PETERSEN, O., and SANDBERG, L. E. Tomography in cholesteatoma of temporal bone. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 588–596.
4. BUCKINGHAM, R. A., and VALVASSORI, G. E. Correlation of surgical and tomographic findings in cholesteatoma of middle ear and mastoid. *Tr. Am. Acad. Ophth.*, 1967, *71*, 673–681.
5. VIGNAUD, J., LERICHE, H., and FISCHGOLD, H. Radiographie du rocher. *La Vie Médicale*, 1970, *9*, 4075–4088.

VOL. 122, NO. 3

ARTERIOGRAPHIC TOPOGRAPHY OF ORBITAL LESIONS*

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ORBITAL lesions may be evaluated radiographically by plain film roentgenography, laminagraphy, orbital venography and cerebral angiography.[3,5,13–19,22,23,25] Comprehensive works are available detailing the bone changes in many types of intraorbital disorders.[8] The plain film roentgenograms may be of value in both localizing and characterizing the lesions. Frequently, however, the bone abnormalities are nonspecific. Orbital venography may be helpful in localizing orbital tumors;[6,24,25] however, this procedure will not provide information as to the histology of the lesion. Orbital ultrasound is becoming increasingly popular for the localization of orbital mass lesions. Orbital arteriography is of value in that it will usually provide localizing information and will, on occasion, suggest the actual histology of the tumor. It is most helpful in delineating extraorbital extensions of invasive tumors.

It is the purpose of this communication to present a topographic analysis of orbital lesions based on the arteriographic findings and to characterize the different types of displacements according to the anatomic zones within the orbit. The differential diagnosis within the various sectors will also be discussed.

TECHNIQUE

The arteriographic evaluation of orbital lesions should include at least 2 and preferably 3 projections; lateral, submentovertical, and frontal projection with the tube angled cranially approximately 10 degrees to the orbito-meatal line.[23] Stereoscopic evaluation, especially in the lateral projection, is valuable in obtaining a 3 dimensional concept.

The degree of intraorbital visualization is related both to the amount of contrast medium injected as well as to the site of injection. Although retrograde brachial and common carotid arteriography will opacify the ophthalmic artery and its branches, selective internal carotid artery injection enhances visualization. Direct magnification using a small focal spot x-ray tube, and photographic subtraction[20] add to the diagnostic accuracy of orbital arteriography. Orbital lesions should be evaluated by external carotid arteriography as well. Lesions involving the inferior portion of the orbit may be vascularized by the infraorbital artery, a distal branch of the maxillary artery.[1,2]

ANATOMY

The normal anatomy of the ophthalmic artery and its branches has been detailed by Hayreh and co-workers,[9–11] and may be summarized as follows: The ophthalmic artery complex is divided into 3 sections, the first 2 are extraorbital and consist of an intracranial and an intracanalar segment[23] (Table 1).

The *intracranial segment* is between 0.7–

TABLE I

DIVISIONS OF THE OPHTHALMIC ARTERY

(After Vignaud *et al.*[23])

A. Intracranial segment
B. Intracanalar segment
C. Intraorbital segment
1. Infraoptic portion
2. Circumneural portion
a. Supraoptic loop
b. Intraoptic loop (type 1 and 2)
3. Terminal portion

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9.4 mm. in length, arising in most cases from the anteromedial or superomedial aspect of the internal carotid artery, intradurally, approximately at the level of the anterior clinoid process.[9] Rarely, it originates more proximally from the intracavernous portion of the internal carotid artery in which case it enters the orbit through a separate canal in the optic strut.[12]

The second, *intracanalar segment*, is that portion within the optic canal that lies in relation to the inferior lateral surface of the optic nerve. Roentgenographically this portion is usually seen as a straight segment with only minimal undulation. On very rare occasions the extraorbital segment of the ophthalmic artery may be totally absent.[10] The orbital structures are then vascularized *via* collaterals through the superior orbital fissure or by other meningeal arteries.

Intraorbital segment. There have been several schemata for classifying the intraorbital segment. That presented by Vignaud *et al.*[23] is most suited for a topographic analysis of orbital masses. In this scheme the intraorbital segment is divided into 3 portions. The first or infraoptic portion runs below the optic nerve within the muscle cone and extends for approximately 10–15 mm. In all 3 projections this portion appears as a direct extension of the intracanalar segment, extending anteriorly and inferolaterally in a straight or slightly undulating course.

The second portion of the intraorbital segment crosses the optic nerve. For purposes of later discussion we have proposed the term "circumneural" for this portion of the ophthalmic artery. This circumneural portion may present in one of three different configurations. The most common type is a supraoptic loop in which case the ophthalmic artery emerges from beneath the optic nerve on its lateral aspect and ascends around the superior surface of the nerve ultimately emerging on the medial side where it continues forward perforating the muscle cone to extend along the medial wall of the orbit. On the lateral roentgenogram this is seen as the classical "bayonet" appearance (Fig. 1*A*). In the frontal view a well formed medially directed concave loop is visible (Fig. 1*B*). Less commonly this portion may present as 2 varieties of an infraoptic loop. In the first variety the ophthalmic artery emerges from below the nerve and ascends directly along the medial side of the nerve. In the lateral view the ophthalmic complex has an "L" shape rather than the more usual bayonet. In the frontal projection there is no round loop about the nerve.

In the second variety of infraoptic loop the ophthalmic artery again emerges on the medial side but in this case ascends in a semicircular direction to the superior surface of the nerve thereby forming a loop about the medial half of the nerve. In the lateral projection the "bayonet" appearance is maintained but in the frontal view the circumneural loop is concave laterally.[23]

The third intraorbital portion of the ophthalmic artery is variable and is the most tortuous. The majority of the visualized branches arise from this portion. It is often difficult to distinguish the small intraorbital branches that arise from the ophthalmic artery due to the 2 dimensional nature of the roentgenogram. There are certain exceptions. The anterior and posterior ethmoidal arteries, the largest medially directed branches, can be detected in the lateral and submentovertical projections (Fig. 1*C*). The lacrimal artery, which ascends to vascularize the lacrimal gland, can also be identified in the submentovertical and anteroposterior projections where it is seen arising near the first loop of the ophthalmic artery and extending to the superolateral quadrant of the orbit.

When prominent, the supraorbital artery can be recognized because of its egress from the orbit *via* the supraorbital canal. The main trunk of the ophthalmic artery continues as the angular artery which can be seen in all three projections. It makes a characteristic loop at the level of the trochlea of the superior oblique muscle where it continues forward and medially.

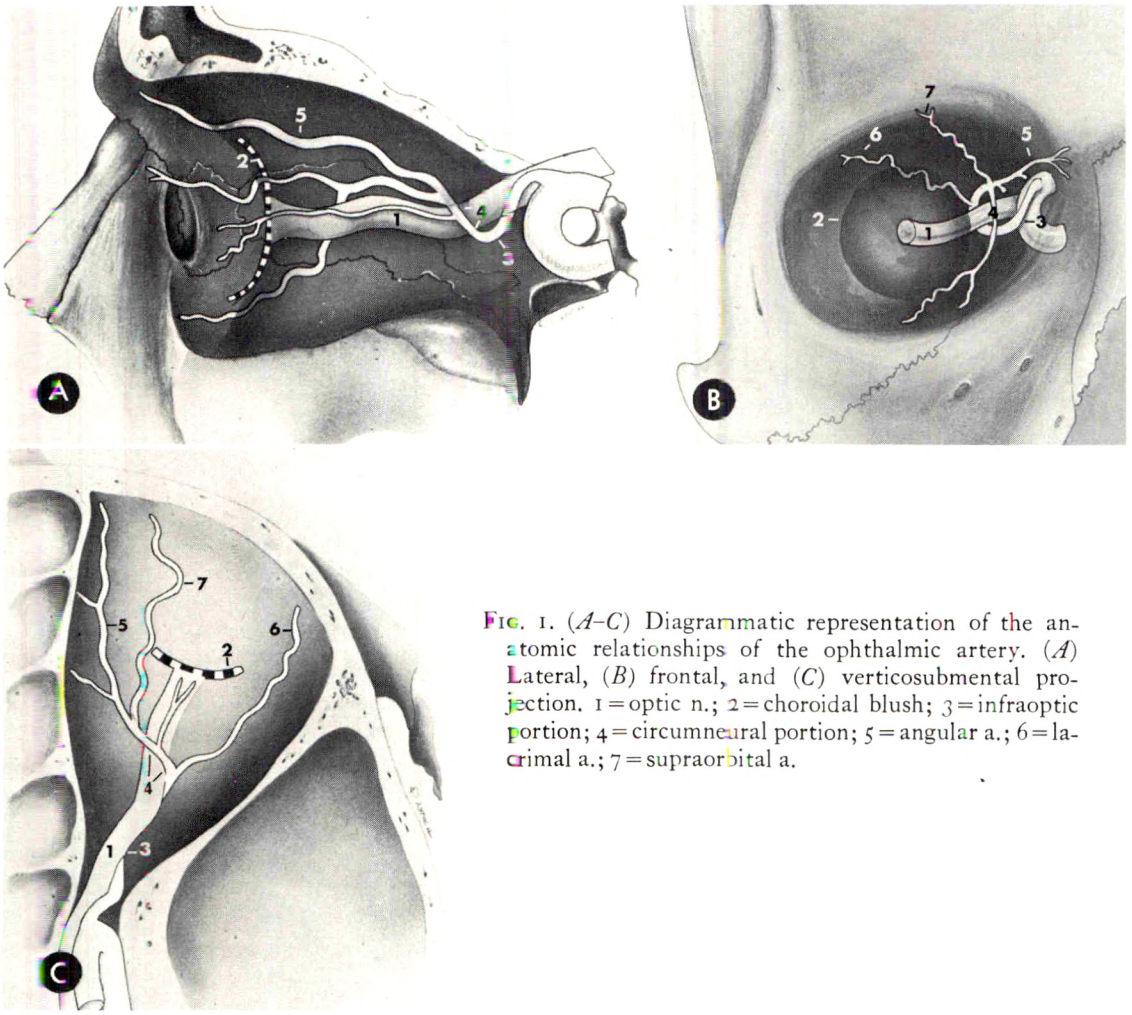


FIG. 1. (*A–C*) Diagrammatic representation of the anatomic relationships of the ophthalmic artery. (*A*) Lateral, (*B*) frontal, and (*C*) verticosubmental projection. 1 = optic n.; 2 = choroidal blush; 3 = infraoptic portion; 4 = circumneural portion; 5 = angular a.; 6 = lacrimal a.; 7 = supraorbital a.

The characteristic crescentic blush of the choroid layer of the eye was first described by Schurr[21] in 1951 and can be visualized on nearly all cerebral angiograms.[7] There are no accurate measurements for the position of the choroidal blush and marked variation in position exists. This is due in part to the effect of small degrees of rotation on the spatial relationship of the choroid to the more lateral zygomatic arches and more medial tuberculum sellae (Fig. 2, *A–C*). The crescentic blush usually appears 3 and 4 seconds after injection of the contrast medium into the internal carotid artery[4] and fades within 4 seconds. The choroidal blush may be seen in the frontal projection (Fig. 3*A*). Usually only the superior and medial surfaces are visualized but on occasion the entire circle is seen. In our experience the choroidal blush is frequently demonstrated in the submentovertical projection (Fig. 3*B*). Lateral and medial displacement as well as compression of the globe can be evaluated. The degree of proptosis is difficult to evaluate in this projection because small changes in the angulation of the head will change the relative position of the choroid.

TOPOGRAPHIC ANALYSIS

The orbit can be divided into zones depending on the direction of displacement of the major vessels and the choroidal blush (Fig. 4).

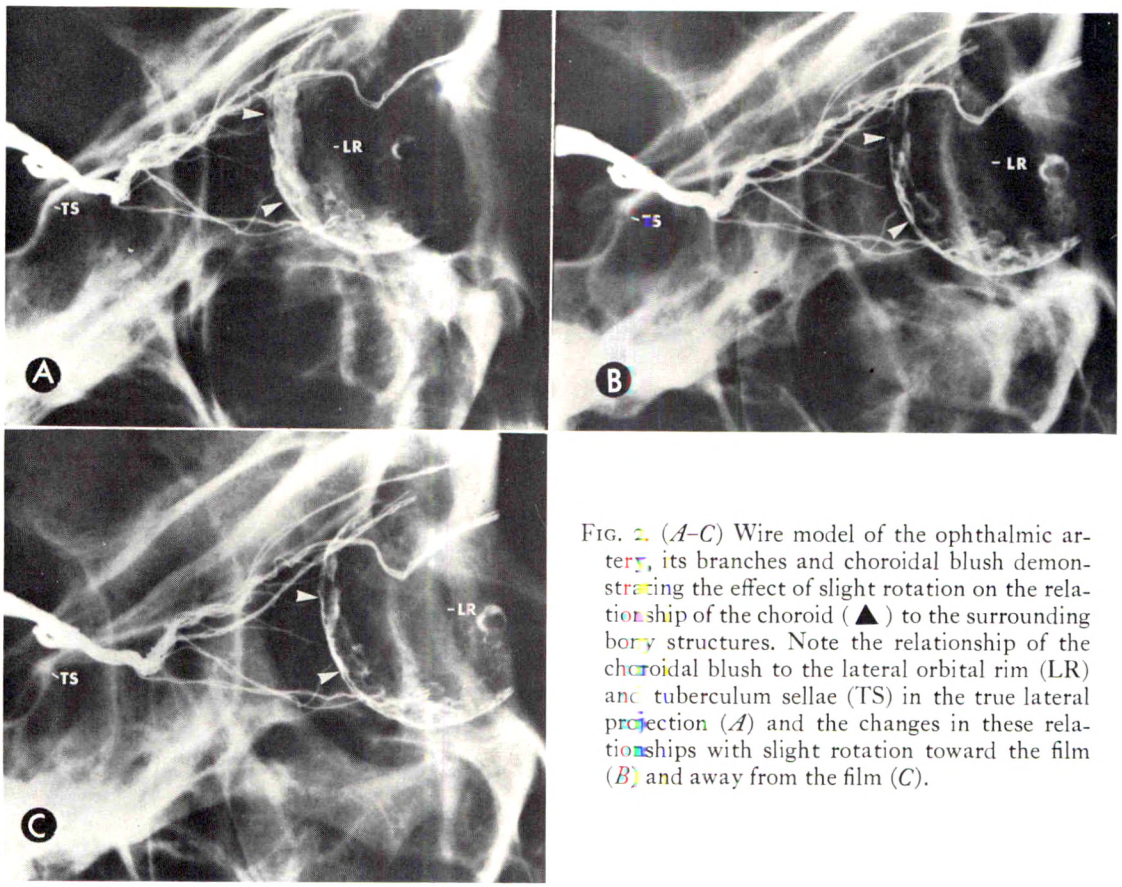


FIG. 2. (*A–C*) Wire model of the ophthalmic artery, its branches and choroidal blush demonstrating the effect of slight rotation on the relationship of the choroid (▲) to the surrounding bony structures. Note the relationship of the choroidal blush to the lateral orbital rim (LR) and tuberculum sellae (TS) in the true lateral projection (*A*) and the changes in these relationships with slight rotation toward the film (*B*) and away from the film (*C*).

The following classification based on vascular displacements was selected:

A. Superolateral lesions
B. Superomedial lesions
C. Medial lesions
D. Inferior lesions
E. Central nerve and sheath lesions
F. Central muscle cone lesions

For the purpose of this analysis the characteristic angiographic changes in each pro-

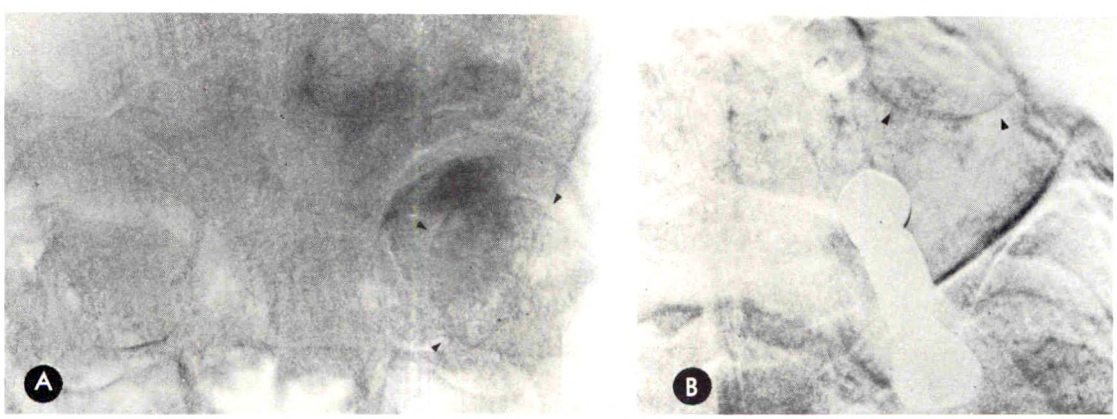


FIG. 3. (*A* and *B*) Normal choroidal blush (▲) as seen in the capillary phase of a selective internal carotid arteriogram in (*A*) the frontal and (*B*) submentovertical projections.

jection will be dealt with separately.

LATERAL PROJECTION

A great deal of information is available on the lateral roentgenogram concerning the position of intraorbital masses.

Superolateral Lesions. Lesions confined to the superolateral quadrant of the orbit are distant from the main trunk of the ophthalmic artery. Consequently, significant displacement of the ophthalmic artery is unusual. However, one would expect to find deviation of the lacrimal artery as well as the superior muscular and ciliary arteries. These vessels will appear stretched and bowed downwards indicating a vector from above (Fig. 5, *A* and *B*). The choroid will be displaced anteriorly and downward corresponding to the amount of proptosis. Lesions which cause downward bowing of the smaller orbital vessels but do not affect the main trunk of the ophthalmic artery will most likely be within the superior lateral quadrant. Masses in this sector include lacrimal gland tumors, dermoid cysts, and sphenoid ridge meningiomas.

Superomedial Lesions. Superomedial lesions distort the main trunk of the ophthalmic artery and its medial branches because of their proximity (Fig. 6, *A–D*). These vessels will appear stretched, and downwardly bowed indicating a force from above. The normal relationship of the proximal portion of the ophthalmic artery to the optic nerve and muscle cone may be maintained. Consequently the loop of the circumneural portion of the ophthalmic artery will be preserved. Large lesions will on occasion so distort the orbital content that the loop will be effaced. The choroidal blush will be displaced anteriorly and downward. The differential diagnosis includes: downward extension of intracranial

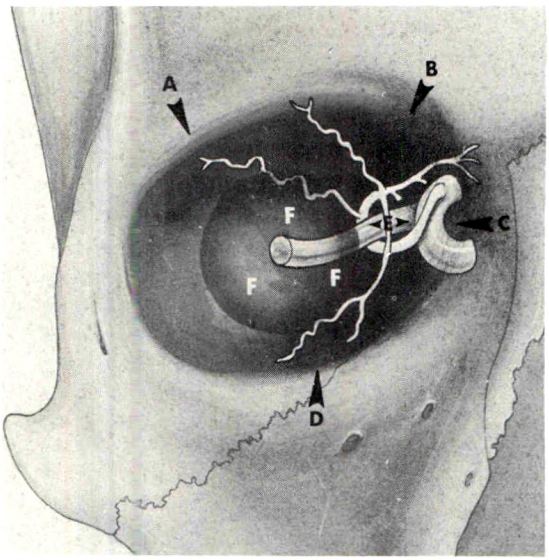


FIG. 4. Diagram of the topographic division of orbital lesions. A. Superolateral lesions; B. superomedial lesion; C. medial lesions; D. inferior lesions; E. central nerve and sheath lesion; F. central muscle cone lesion.

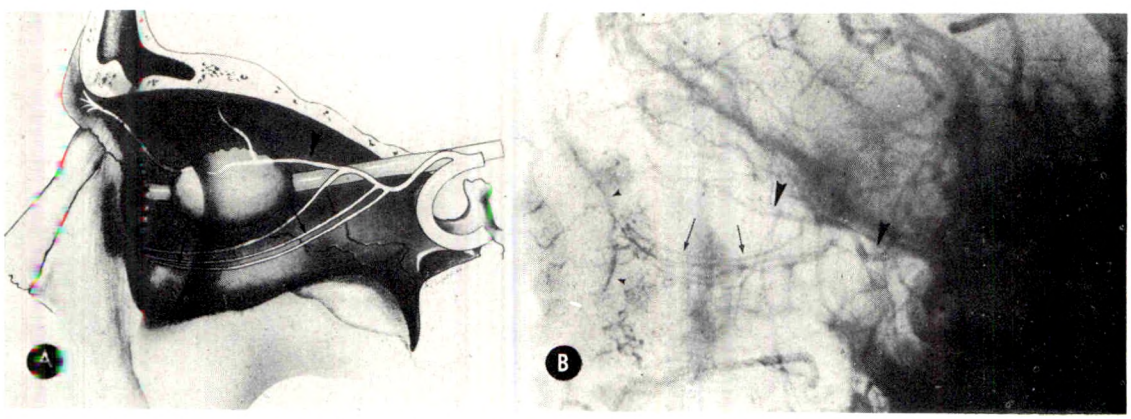


FIG. 5. (*A* and *B*) Superolateral mass lesion (dermoid) in the lateral projection. The ophthalmic artery is normal throughout its course (▲). The lacrimal artery and the superior muscular branches are stretched and displaced downward (▲). The choroidal blush is displaced forward (▲). Displacement of the superior branches with a normal ophthalmic artery is characteristic of superolateral masses.

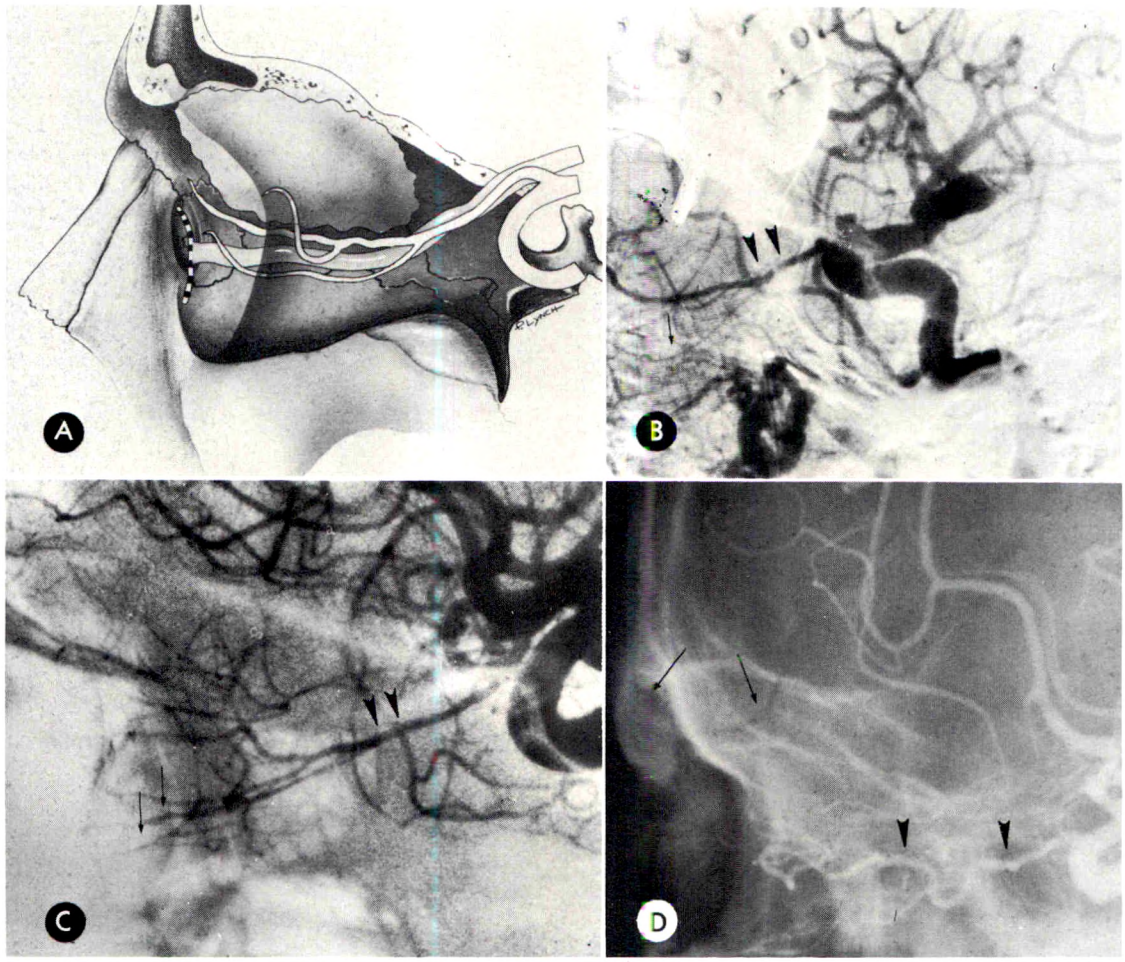


FIG. 6. Superomedial mass lesions in the lateral projection. (*A* and *B*) Mucocele of the frontal sinus displacing the ophthalmic artery (▲) downward. The angular artery and other superior branches are displaced downward (▲). (*C*) Fracture of the floor of the frontal fossa with herniation of the brain into the orbit. The entire ophthalmic arterial complex is displaced downward (▲). Fracture lines are noted within the frontal bone (▲). (*D*) Superior tumor displacing the ophthalmic artery (▲) and its distal branches (▲) downward.

lesions including meningiomas and traumatic brain herniation; frontal sinus mucocele; osteoma; or carcinoma.

Medial Lesions. Small medial lesions may be quite difficult to diagnose in the lateral projection. The majority of the mass effect is directed laterally, perpendicular to the plain of the film. Consequently, the extent of the displacement cannot be determined. There will usually be some straightening and loss of undulations of the ophthalmic artery suggesting that the vessel is being displaced along its horizontal axis (Fig. 7, *A* and *B*). The frontal and submentovertical projections are the key to diagnosis of these lesions. The choroidal blush will be normal or imperceptibly displaced. Differential diagnosis includes: osteoma of the ethmoid sinus; ethmoid tumors including carcinoma; lymphoepithelioma; and chondral tumors.

Optic Nerve or Sheath Lesions. Because of the intimate relation of the proximal portion of the ophthalmic artery to the optic nerve, a lesion of the optic nerves and their dural coverings cause characteristic angiographic changes (Fig. 8, *A–C*). The infraoptic portion of the ophthalmic artery may

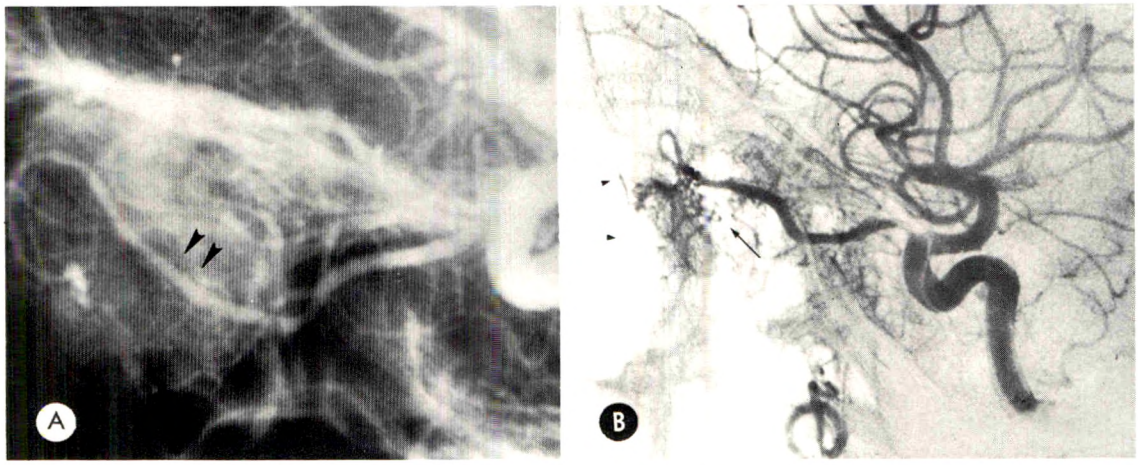
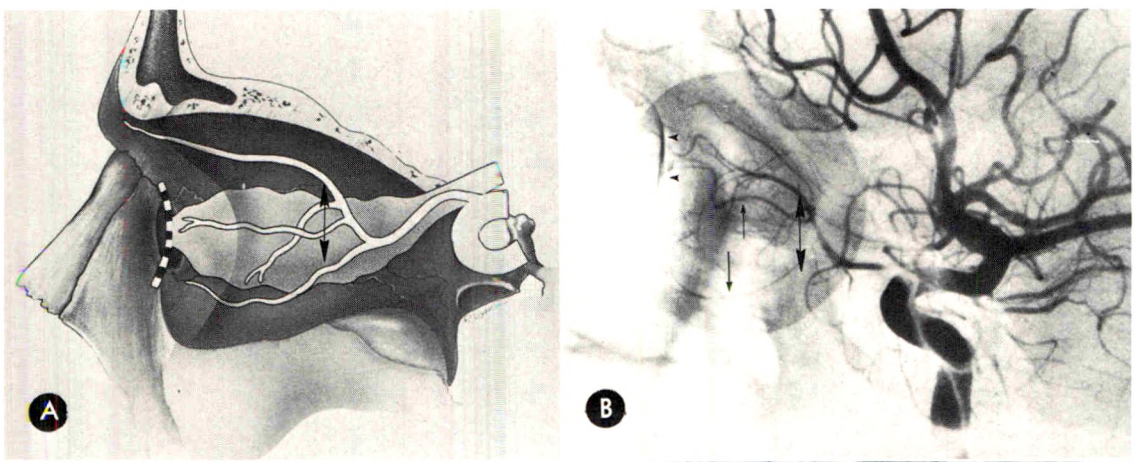


Fig. 7. Medial lesions in the lateral projection. (*A*) Osteoma of the ethmoid sinus. The terminal portion of the ophthalmic artery (▲) is stretched and bowed around the bony mass lesion. (*B*) Lymphoepithelioma of the ethmoid sinus invading the orbit. Tumor vascularity is noted (▲), however, the major trunk of the ophthalmic artery is not deviated. Lateral deviation is best demonstrated in the frontal and submento-vertical projections.



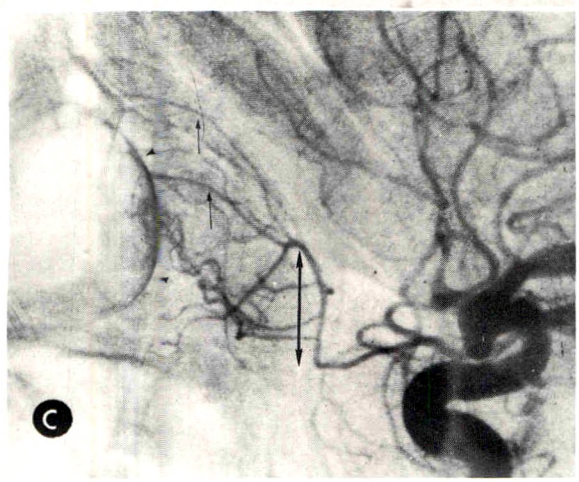


Fig. 8. (*A* and *B*) Optic nerve lesions in the lateral projection. The infraoptic portion of the ophthalmic artery is normal and the circumneural loop is widened ( ). The major branches are swept around the mass (▲) and the choroidal blush (▲) is displaced forward and flattened. The extent of the lesion is delineated by multiple stretched vessels. (*C*) In this case the infraoptic portion of the ophthalmic artery is depressed in addition to the widened circumneural loop. This glioma is considerably larger than the previous one and the vascular displacement consequently is more marked.

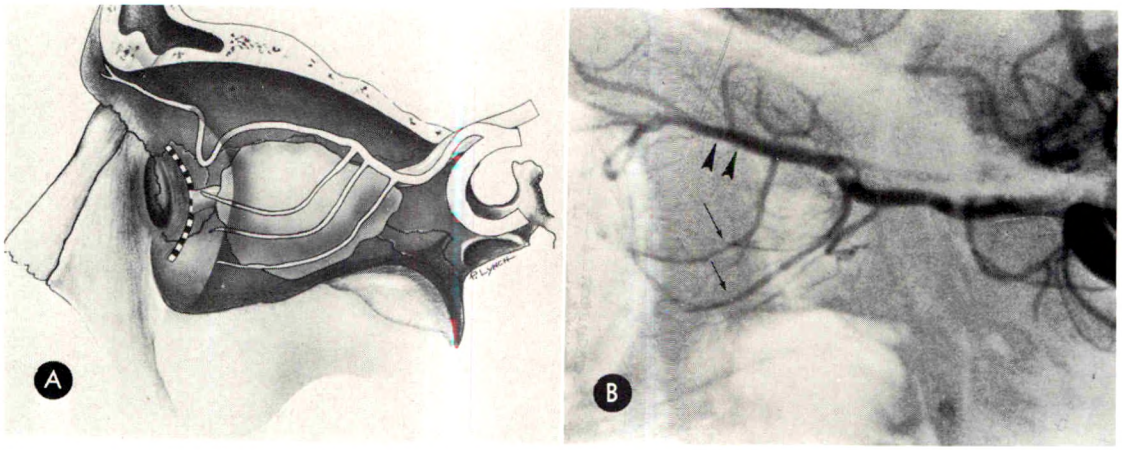


Fig. 9. (*A* and *B*) Muscle cone lesion in the lateral projection (hemangioma). The first portion of the ophthalmic artery is normal. The inferior branches (▲) and the angular artery (▲) are straightened and stretched indicating the central position of the mass.

be depressed. The major change, however, involves the arterial loop around the optic nerve. These lesions cause widening of the nerve and consequently this circumneural segment will be stretched and widened. The normal range for the width of the loop is stated to be from 3 to 7 mm.[23] The terminal portion of the artery is stretched and elevated. The muscular branches and the ciliary arteries may also be swept around the expanded nerve. The choroidal blush may be flattened, deformed, or displaced directly forward. Three lesions are of primary consideration in this area: optic nerve glioma; neurofibroma; and primary orbital meningioma. When tumor hypervascularity is absent these 3 lesions will be indistinguishable angiographically.

Muscle Cone Lesions. The intraorbital segment of the ophthalmic artery and its branches will be displaced in accordance with the exact location of the lesion (Fig. 9, *A* and *B*). The superiorly directed muscular branches can be displaced superiorly and bowed upward. The lateral branches are bowed laterally and the inferior branches are displaced inferiorly. While it may be impossible to specifically identify which muscular arteries are opacified on the arteriogram, the characteristic pattern of bowing will indicate that the mass lies centrally within the muscle cone. The relation of the ophthalmic artery to the optic nerve is maintained throughout its first and second portions and accordingly the circumneural loop will not be widened. The choroidal blush will be displaced forward and may be flattened. A number of lesions characteristically occur in this area; *i.e.*, hemangiomas; varicocele; and sarcomas.

Inferior Lesions. Large inferiorly placed masses distort the ophthalmic arterial complex in a characteristic manner (Fig. 10, *A–C*). The ophthalmic artery as well as the muscular and ciliary branches are stretched and bowed upwards. The choroidal blush will be indented posteriorly and displaced forwards. If these lesions originate within the muscle cone the lateral and medial muscular branches will be splayed apart as well as elevated. These findings are best demonstrated in the submentovertical projection (Fig. 13, *A* and *B*). Small lesions arising within the inferior portion of the orbit or invading from the maxillary sinus or nasopharynx should be evaluated by external carotid arteriography.[1,2] The infraorbital and sphenopalatine arteries and their branches commonly supply lesions in this sector. Orbital lesions extending into the maxillary sinus will cause localized inferior bowing of the infraorbital artery.[2] This vessel is characterized by its straight

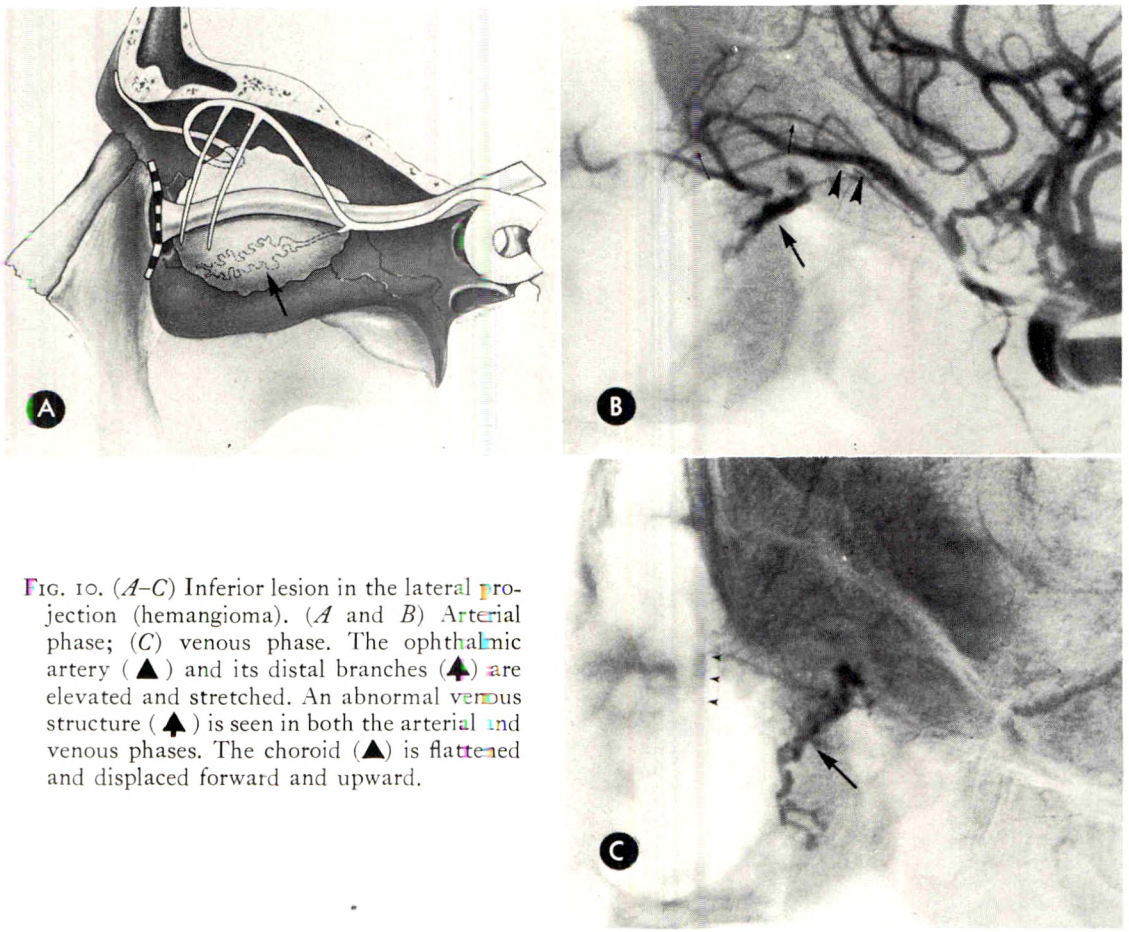


FIG. 10. (*A–C*) Inferior lesion in the lateral projection (hemangioma). (*A* and *B*) Arterial phase; (*C*) venous phase. The ophthalmic artery (▲) and its distal branches (▲) are elevated and stretched. An abnormal venous structure (▲) is seen in both the arterial and venous phases. The choroid (▲) is flattened and displaced forward and upward.

course within the infraorbital groove and canal. Its downward displacement will be found in fractures of the orbital floor with localized mass, malignant orbital tumors extending into the maxilla (rhabdomyosarcoma, etc.). The infraorbital artery will be displaced upward with spread of carcinoma from the maxillary antrum. Tumor hypervascularity arising from the infraorbital or sphenopalatine arteries may occur without major vessel displacement. This commonly occurs in cylindroma, juvenile angiofibroma and meningioma.

SUBMENTOVERTICAL PROJECTION

The submentovertical (SMV) projection complements the lateral projection better than the frontal projection. In the frontal projection the ophthalmic artery and all of its major branches course forward perpendicular to the plane of the film (Fig. 1*B*). Consequently, except for the circumneural loop the remainder of the vessel appears foreshortened. In the SMV projection the full length of each visualized vessel may be evaluated. The choroidal blush is very frequently visualized. Deviation or distortion of the back of the globe can be evaluated.

Medial and Lateral Lesions. The relationship of the intracanalar and intraorbital segments of the ophthalmic artery to the sphenoid and ethmoid sinuses is most easily determined in the SMV projection (Fig. 11, *A* and *B*). Consequently, masses arising from these structures stretch and straighten the ophthalmic artery displacing it laterally.

Laterally situated lesions and those growing through the sphenoidal wing will

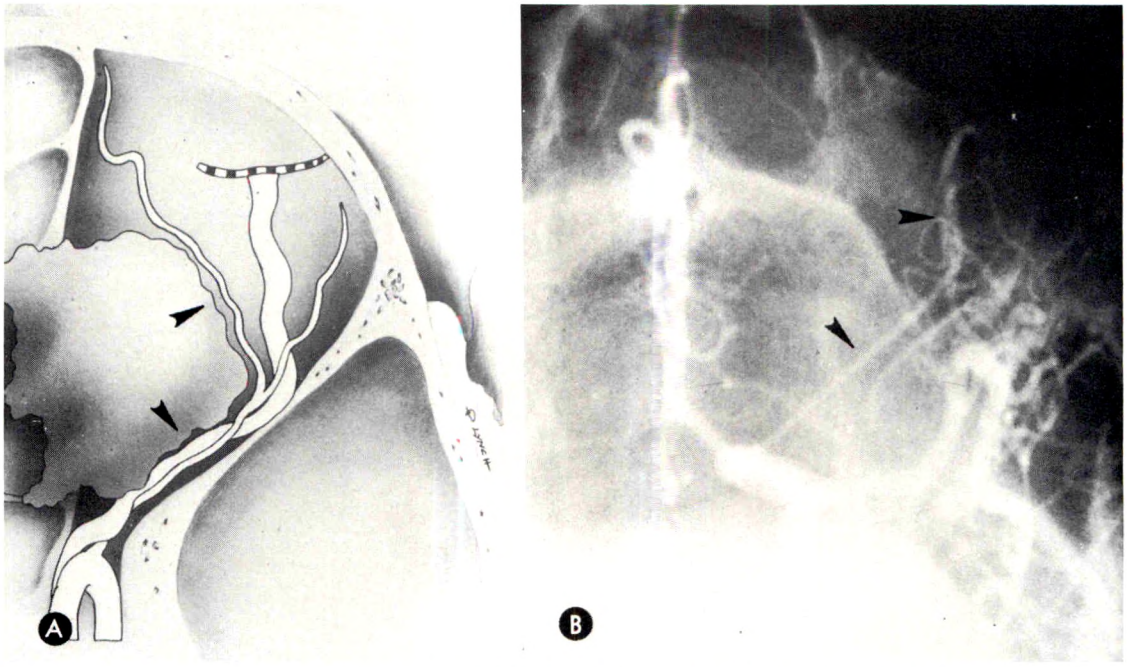


FIG. 11. (*A* and *B*) Medial lesion in the submentovertical projection. Carcinoma of the ethmoid sinus. The ophthalmic artery (▲) is stretched, displaced laterally and outlines the lateral aspect of the tumor.

displace the proximal portions of the ophthalmic artery medially.

Optic Nerve and Sheath Lesions. As previously described, lesions of the optic nerve and sheath characteristically distort the circumneural loop of the ophthalmic artery. This is demonstrated to advantage in the SMV projection (Fig. 12, *A* and *B*). The infraoptic portion is displaced laterally. The loop segment is stretched and straightened as it courses over the optic nerve. The combination of lateral deviation of the infraoptic portion of the artery and expansion of its circumneural loop is characteristic of optic nerve or nerve sheath tumors.

Muscle Cone Lesions. Masses within the muscle cone will separate and splay the distal ophthalmic artery branches toward the periphery of the orbit indicating the central location of the mass (Fig. 13, *A* and *B*). Centrally located tumor vascularity, when present, is helpful in localizing the mass. In contradistinction to optic nerve lesions, the circumneural portion of the ophthalmic artery should be normal.

FRONTAL PROJECTION

In the frontal projection alterations in topography are manifest by displacement of the ophthalmic artery complex and/or the choroidal blush in the horizontal or vertical planes.

Medial and Lateral Lesions. Medial lesions, those arising from the ethmoid region or nasal cavity, displace the infraoptic and circumneural portions of the

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FIG. 13. (*A* and *B*) Muscle cone lesion in the submentovertical projection (hemangioma). The infraoptic and circumneural portions of the ophthalmic artery are normal (▲) excluding an optic nerve glioma. The central position of the mass is suggested by the lateral displacement of the muscular and lacrimal arteries (▲). An abnormal venous channel is demonstrated (▲).

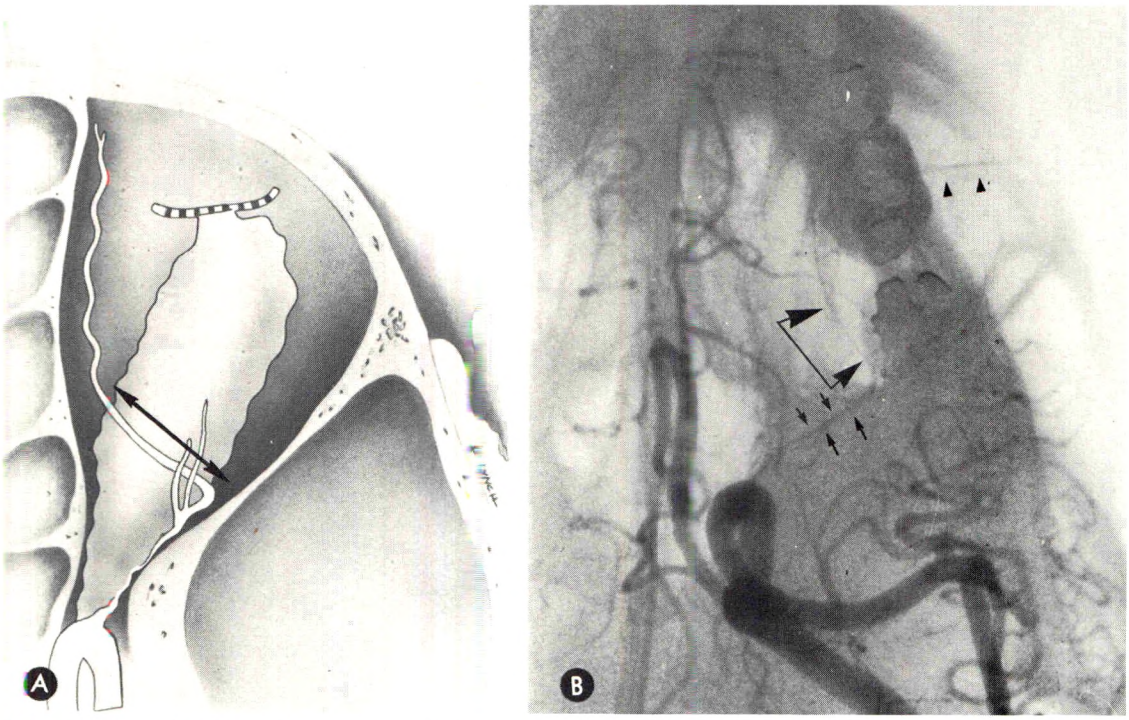
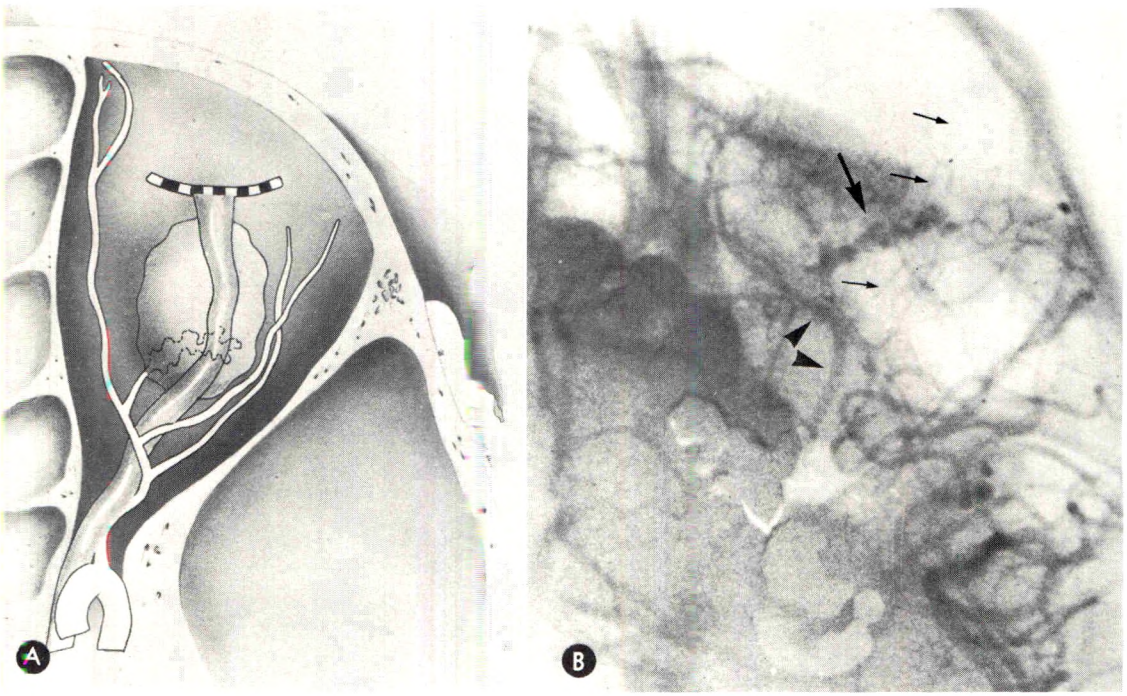


Fig. 12. (*A* and *B*) Optic glioma in the submentovertical projection. The infraoptic portion of the ophthalmic artery is displaced laterally (▲). The circumneural loop is stretched over the enlarged optic nerve (▲). The choroidal blush (▲) is displaced forward and flattened.



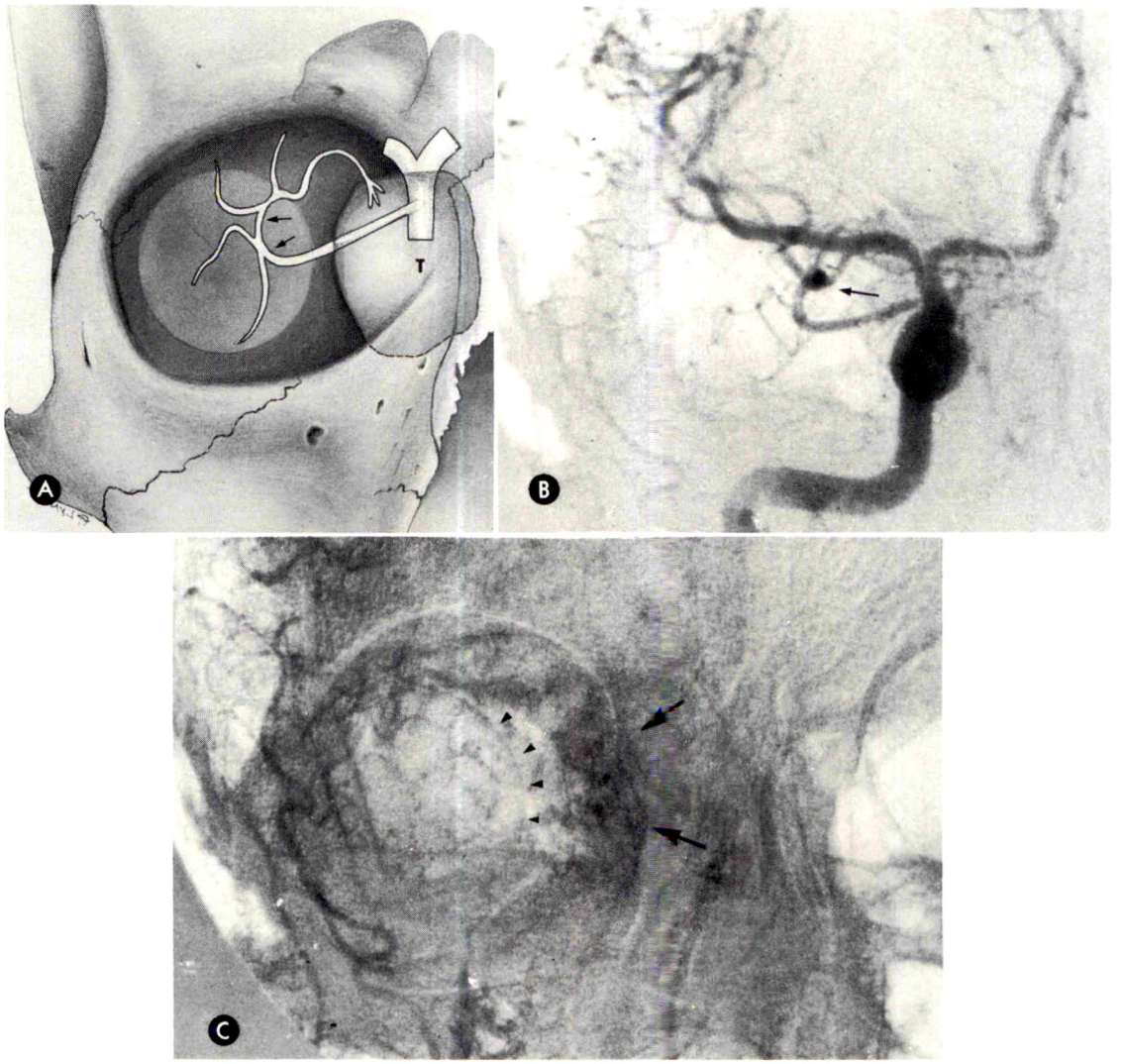


Fig. 14. (*A–C*) Medial lesion in the frontal projection (lymphoepithelioma of the ethmoid sinus). (*A* and *B*) Arterial phase. The infraoptic portion of the ophthalmic artery is stretched and the circumneural loop is displaced laterally (▲). (*C*) Venous phase. Tumor stain is present in the medial aspect of the orbit (▲) and the choroid blush is displaced laterally (▲).

ophthalmic artery laterally; however, there is usually a minimal deformity of the circumneural loop (Fig. 14, *A* and *B*). There may be lateral displacement of the choroidal blush (Fig. 14*C*).

Lateral lesions will deviate the ophthalmic arterial complex and the choroidal blush medially (Fig. 15, *A* and *B*). The infraoptic segment and loop will be displaced as well as the more distal branches. In this instance, however, one must be aware of the normal variations as the infraoptic type of ophthalmic artery will normally appear more medial than the supraoptic variety because the artery lies medial to the nerve in this instance.

Superior and Inferior Lesions. Superior masses will displace the infraoptic and loop segments downward. Anteriorly located lesions may distort the trochlear loop as well as the angular and supraorbital branches. Inferior lesions will elevate the entire complex superimposing it upon the middle cerebral artery (Fig. 16). Consequently, it will be difficult to define the ophthalmic artery in such cases in this pro-

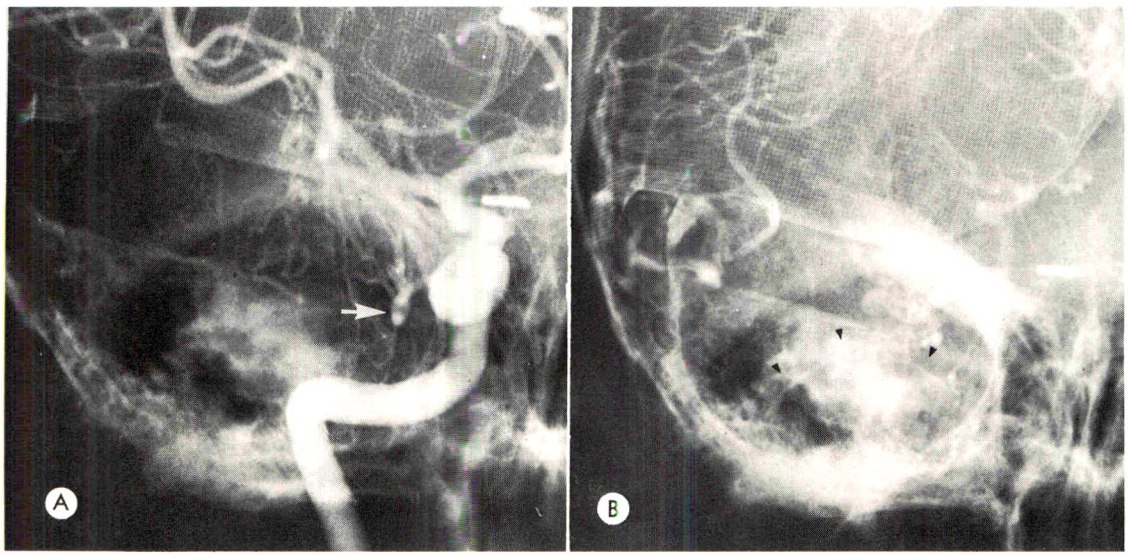


FIG. 15. (*A* and *B*) Lateral lesions in the frontal projection (meningosarcoma). The infraoptic portion of the ophthalmic artery (▲) and the choroidal blush are displaced medially (▲).

jection. Vertical displacement of the choroid is difficult to evaluate on the frontal film. Minor variations in the angle of the incident beam will cause apparent depression of the choroid. If the projection is correct, downward displacement can be appreciated.

SUMMARY AND CONCLUSIONS

A topographic analysis of the vascular alterations of orbital mass lesions is presented.

The orbit is divided into 6 zones and lesions in these zones are correlated with specific changes involving the ophthalmic artery and its branches. With this information many intraorbital lesions can be defined.

While this approach will be helpful in most cases of orbital masses, there will be instances when lesions will be so large as to defy precise localization within the orbit.

Very small tumors may demonstrate only minimal distortion or displacement of the vascularity precluding arteriographic diagnosis.

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REFERENCES

1. ALLEN, W. E., III, KIER, E. L., and ROTHMAN, S. L. G. Maxillary artery: normal arteriographic anatomy. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1973, *118*, 517–527.

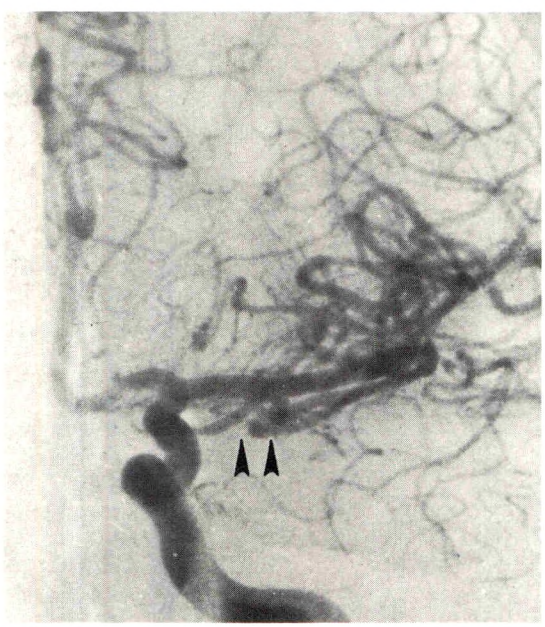


FIG. 16. Inferior lesion in the frontal projection. Hemangioma elevating the ophthalmic arterial complex ( ). The distal branches cannot be seen because of superimposition over the middle cerebral artery branches.

2. Allen, W. E., III, Kier, E. L., and Rothman, S. L. G. Maxillary artery in craniofacial pathology. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 139–142.
3. DiChiro, G. Ophthalmic arteriography. *Radiology*, 1961, *77*, 948–957.
4. DiChiro, G. Angiographic topography of choroid. *Am. J. Ophth.*, 1962, *54*, 232–237.
5. Dilenge, D., and Metzger, J. L'Angiographie de l'Artère Ophthalmique. In: L'Exploration Neuroradiologique en Ophthalmologie. Edited by P. Guillot, L. Saraux, and H. Sedan. Masson et Cie, Paris, 1966.
6. Hanafee, W. N. Orbital venography. *Radiol. Clin. North America*, 1972, *10*, 63.
7. Handel, S. F., Stargardter, F. L., Glickman, M. G., and Newton, T. H. Frequency of ocular choroidal blush during arteriography. *Neuroradiology*, 1973, *6*, 50–55.
8. Hartmann, E., and Gilles, F. Roentgenologic Diagnosis in Ophthalmology. J. B. Lippincott Company, Philadelphia, 1959.
9. Hayreh, S. S., and Dass, R. Ophthalmic artery. Part I. Origin, and intracranial and intracanalicular course. *Brit. J. Ophth.*, 1962, *46*, 65–68.
10. Hayreh, S. S., and Dass, R. Ophthalmic artery. Part II. Intraorbital course. *Brit. J. Ophth.*, 1962, *46*, 165–185.
11. Hayreh, S. S. Ophthalmic artery. Part III. Branches. *Brit. J. Ophth.*, 1962, *46*, 212–247.
12. Kier, E. L. Embryology of normal optic canal and its anomalies: anatomic and roentgenographic study. *Invest. Radiol.*, 1966, *1*, 346–362.
13. Krayenbühl, H. Diagnostic value of orbital angiography. *Brit. J. Ophth.*, 1958, *42*, 180–190.
14. Krayenbühl, H. Value of orbital angiography for diagnosis of unilateral exophthalmos. *J. Neurosurg.*, 1962, *19*, 289–301.
15. Lloyd, G. A. S. Technique for arteriography of orbit. *Brit. J. Radiol.*, 1960, *42*, 252–255.
16. Lloyd, G. A. S. Radiological investigation of proptosis. *Brit. J. Radiol.*, 1970, *43*, 1–18.
17. Lombardi, G. Radiology in Neuro-ophthalmology. Williams & Wilkins Company, Baltimore, 1967.
18. Lombardi, G. Orbital pathology and contrast media. *Radiol. Clin. North America*, 1972, *10*, 115–128.
19. Lombardi, G., and Passerini, A. Ophthalmic artery in axial view. *Acta radiol.* (Diag.), 1969, *9*, 379–382.
20. Rothman, S. L. G., Kier, E. L., Allen, W. E. III, and Barauskas, L. Simple technique for photographic image enhancement of subtraction films. *Radiology*, 1973, *107*, 457–459.
21. Schurr, P. H. Angiography of normal ophthalmic artery and choroidal plexus of eye. *Brit. J. Ophth.*, 1951, *35*, 473–478.
22. Takashashi, M., Lombardi, G., Passerini, A., and Obno, S. Primary intraorbital meningiomas: roentgenologic study. *Neuroradiology*, 1973, *5*, 95–101.
23. Vignaud, J., Clay, C., and Aubin, M. L. Orbital arteriography. *Radiol. Clin. North America*, 1972, *10*, 30–61.
24. Vignaud, J., Clay, C., and Bilaniuk, L. T. Venography of orbit: analytical report of 413 cases. *Radiology*, 1974, *110*, 373–382.
25. Zizmor, J., and Lombardi, G. Atlas of Orbital Radiography. Aesculapius Publishing Co., Birmingham, 1973.

VOL. 122, NO. 3

THE ROENTGENOLOGIC DEMONSTRATION OF SINUSITIS*

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IT is obvious that a correct diagnosis has to be established before correct treatment of a particular disease can be instituted. However, the diagnosis, especially in routine medical care, often lacks reliability. Instead, the patient is often treated "as if" he suffered from a particular disease or in order "to be prepared." The need for correct diagnosis is especially evident if therapy differs between a disease and related conditions. This is the case in acute sinusitis. The difference between an uncomplicated rhinitis and a sinusitis is indistinct from a pathophysiologic and clinical viewpoint. However, an uncomplicated rhinitis, in general, does not require any major therapy, but the sinusitis often requires antibacterial treatment. Furthermore, if secretion is present in the diseased sinus, healing often requires uncomfortable drainage measures: *i.e.*, irrigation, and a 10 day's treatment of antibiotics. Both these measures can apparently be omitted in the treatment of uncomplicated rhinitis. The establishment of reliable clinical methods to differentiate between the two conditions is obviously needed.

The common clinical means to establish the diagnosis of sinusitis are often too unreliable (the patient's history, inspection of the nasal mucosa, diaphanoscopy), or are too uncomfortable for the patient (sinucopy, or diagnostic irrigation). The roentgenologic examination, on the other hand, is both painless and safe, and its importance for the establishment of a diagnosis of sinusitis has been ascertained by many authors.[5,9,12,14,15,18]

Roentgenologic sinus examination, in general, contains 4 standard projections: the occipito-frontal (OF); the occipito-mental (OM); the lateral (L); and the axial (A). For the demonstration of fluid, a horizontal beam has to be used with the patient in a sitting position. The different projections show the different sinuses and are routinely made on suspicion of sinusitis. The clinically most important feature to be demonstrated roentgenologically in sinusitis is the occurrence of fluid in the sinus, indicating impaired drainage and possible infected secretion in the sinus. The frequency of inflammatory changes varies greatly among the sinuses, most frequently affecting the maxillary sinus and least frequently, the sphenoidal sinus. The confidence of demonstrating secretion by the 4 routinely adopted projections is poor, when controlled with subsequent irrigation.[4–6,9,13,18] In general, the interval between the roentgenologic examination and the subsequent irrigation either was not given by the above authors or amounted to several days. In a similar investigation using immediate irrigation after the roentgenologic examination, the lack of confidence of the 4 standard roentgenograms in demonstrating sinus secretion could also be confirmed.[1,7] These authors also showed that the confidence of demonstrating secretion in maxillary sinuses could be increased from 24 to 88 per cent in all not completely opaque sinuses by adding a fifth projection, the occipito-mental side (OMS), with the patient's head recumbent and the diseased side downwards. The secretion then moves from the alveolar recess, where it is hard to detect, to the zygomatic recess, where it is easily observable. Pathologic findings in the frontal sinuses can be examined in a similar manner, adopting the OF view, with head tilted (OFS).

The aim of the present investigation was to examine the reliability of routine roent-

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genologic methods in an attempt to simplify the roentgenologic examination, preferably with increased roentgenologic confidence.

MATERIAL AND METHOD

The sinus roentgenograms of 300 randomly selected patients referred for suspected sinusitis were evaluated. All patients had been examined by the 4 standard views: OF, OM, L, and A. In addition, the above mentioned occipito-mental and occipito-frontal projections with the cheek down (OMS, OFS) had been performed in all cases with any maxillary or frontal sinus changes except complete opacity. The OM projection was checked first, since it gives the best view of the most commonly diseased sinuses, the maxillary, and findings were noted. Then additional views were checked and changes from the preliminary findings noted.

The present investigation was limited to adult patients, 15 years or older. Since the ethmoidal cells lying closest to the nasal cavity are involved in the infectious process very early in rhinitis and sinusitis,[10] and since uncomplicated changes here seldom require specific therapy, the ethmoidal cells were not included in the roentgenologic evaluation. The complete results are given elsewhere, including an examination of healthy controls and an estimation of the economical savings made possible with the new roentgenologic sinus procedure.[2] The results are usually limited to the straight roentgenologic findings.

RESULTS

The evaluation of the sinus roentgenograms is given in Table I. Thirty-two per cent of the patients with clinical suspicion of sinusitis had normal roentgenograms. Fifty-six per cent had changes confined to the maxillary sinuses only, 1 per cent to the frontal sinuses only, and 11 per cent to both maxillary and frontal sinuses. None of the 300 patients showed changes in the sphenoidal sinus. These figures clearly demonstrate the great variation between the different sinuses.

The types of the roentgenologic findings are given in Table II. In the maxillary sinuses, mucous membrane thickening only, mucous membrane thickening in combination with secretion, and complete opacity were approximately of equal occurrence (15 per cent each), whereas other changes were unusual (3.5 per cent). The frontal sinuses were missing in 3 to 4 per cent.

The confidence of the different roentgenologic projections in comparison with the basic OM projection is given in Table III. It appears that the OM view has a high degree of diagnostic reliability for demonstrating maxillary changes. In some cases with mucous membrane thickening, but without secretion in the OM view, secretion appeared in the OMS projection (Fig. 1, *A* and *B*; and 2, *A* and *B*). In sinusitis confined to the maxillary sinus, the additional 3 projections never disclosed any different roentgenologic findings. In some cases, the OM view failed to show both frontal sinuses due to technical reasons, and in other cases the frontal sinuses were too

TABLE I

ROENTGENOLOGIC EXAMINATION OF 300 PATIENTS*

Changes	No.	Per Cent
None	97	32
In one maxillary sinus	88	29
In both maxillary sinuses	80	27
In one frontal sinus	2	<1
In both frontal sinuses	1	<1
In one maxillary and one frontal sinus	12	4
In one maxillary and both frontal sinuses	2	<1
In both maxillary and one frontal sinus	14	5
In both maxillary and both frontal sinuses	4	1
	300	100

* Occipito-frontal (OF); occipito-mental (OM); lateral (L); axial (A); and sometimes occipito-frontal (OFS), and occipito-mental (OMS) views with cheek downward.

TABLE II

PERCENTAGE OF TYPES OF SINUS CHANGES

	Right Maxillary	Left Maxillary	Total Maxillary	Right Frontal	Left Frontal	Total Frontal	Sphenoidal
No changes	47	55	51	89	91	90	0
Mucous membrane thickening (MMT)	13	17	15	3	2	2.5	0
Secretion	2	<1	1	2	2	2	0
MMT+secretion	16	15	15	<1	<1	<1	0
MMT+polyp	<1	1	<1	0	0	0	0
Polyp or cyst	4	3	3.5	0	0	0	0
Complete opacity	17	12	14.5	2	1	1.5	
Frontal sinus missing				4	3	3.5	
Number of sinuses	300	300	600	300	300	600	300

large for satisfactory evaluation in the OM projection only. When the frontal sinuses could be evaluated on the OM view, there were only 5 cases where the preliminary finding had to be corrected after examining the OF projection. The lateral (L) and axial (A) projections never gave additional information to the preliminary roentgenologic findings in sinusitis.

DISCUSSION

The number of roentgenologic examinations per capita is constantly increasing. The use of medical x-ray film increased 44 per cent in the United States from 1958 to 1963 compared with an increase of population of 14.3 per cent. According to official statistics in the U.S.A., the index of annual medical x-ray film consumption set at 100

TABLE III

TYPE OF MAXILLARY CHANGES IN THE FIRST EVALUATED PROJECTION (UPRIGHT, OM) IN COMPARISON WITH THE SUBSEQUENTLY EVALUATED PROJECTION (SIDE, OMS)

The results after evaluation of OM+OMS projections	Evaluation after the first evaluated projection (OM)							
	No changes	MMT[1]	Secretion	MMT+ secretion	MMT+ cyst or polyp	Cyst or polyp	Complete opacity	Total
No changes	302	—	—	—	—	—	—	302
MMT[1]	—	89	—	1	—	—	—	90
Secretion	1	—	2	—	—	—	—	3
MMT+secretion	—	44	—	48	—	—	—	92
MMT+polyp	—	1	—	—	3	—	—	4
Polyp	—	—	—	—	—	21	—	21
Complete opacity	—	1	—	—	—	1	86	88
Total	303	135	2	49	3	22	86	600

Agreement

MMT[1] = mucous membrane thickening.

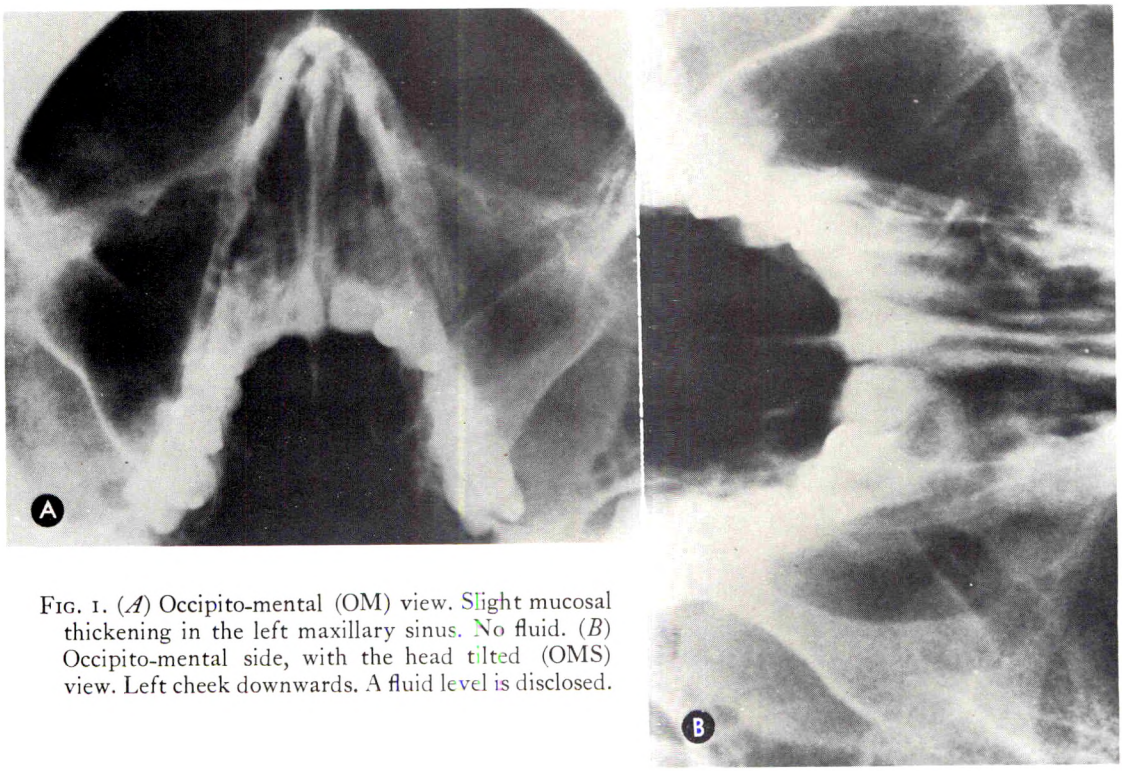


FIG. 1. (*A*) Occipito-mental (OM) view. Slight mucosal thickening in the left maxillary sinus. No fluid. (*B*) Occipito-mental side, with the head tilted (OMS) view. Left cheek downwards. A fluid level is disclosed.

in 1947 had increased to 230 by 1963 (U.S. Department of Health, 1966). The apparent consequences of these facts are economical for the individual as well as for society. Additionally, the consequences of the continuously increasing radiation pollution should not be neglected. This has lately been emphasized by Margulis,[11] who concluded that: *Radiologists and physicians should dispense with routine roentgen examinations and only perform or order those that are medically indicated in the particular patients*. This statement has particular importance in investigations which are time consuming and expensive or where the frequency of the disease is so high as to require many examinations. The latter is the case with sinusitis.

Sinusitis is a fairly common disease constituting 1.5 per cent of all outpatient visits[8] in Sweden, and between 4 and 7 per cent of visits by otolaryngologic patients.[3,16,17] The only reliable and simple means of establishing the diagnosis is by roentgenography. The findings of the present investigation clearly indicate that the roentgenologic method can be simplified, and 2 of the routinely adopted projections, the axial and lateral, can be eliminated from routine procedures in the diagnosis of sinusitis.

The present investigation demonstrates that in clinically suspected sinusitis cases, the OM view has a high degree of confidence. If it is negative, no more views are needed. In cases of pathologic changes in the maxillary sinuses, an additional OMS view with the diseased side down is the easiest and best way of finding small amounts of fluid and to differentiate between fluid and a polyp or cyst of the sinus (Fig. 3, *A* and *B*). It was also found that the OF projection seldom added information to an OM view of the frontal sinuses, when these were roentgenologically demonstrable. Only if the centering of the beam is inadequate, or if the frontal sinuses are large, or if there are suspected roentgenologic changes on an initial OM view, are the OF view and in some cases the OFS view

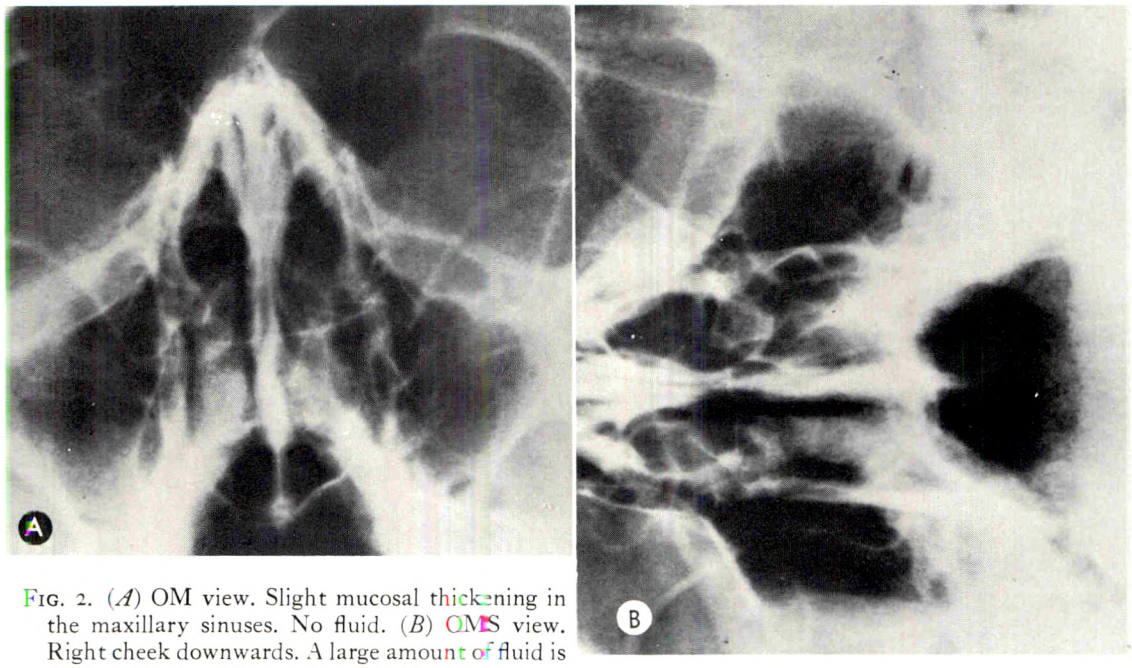


Fig. 2. (*A*) OM view. Slight mucosal thickening in the maxillary sinuses. No fluid. (*B*) OMS view. Right cheek downwards. A large amount of fluid is disclosed in the right maxillary sinus.

needed. There was never any additional roentgenologic information obtained from the lateral and axial projections concerning the diagnosis of sinusitis. The axial view is necessary mainly when, in cases of complete opacity of the maxillary or frontal sinuses, the bony walls have to be studied. Consequently, it appears that the number

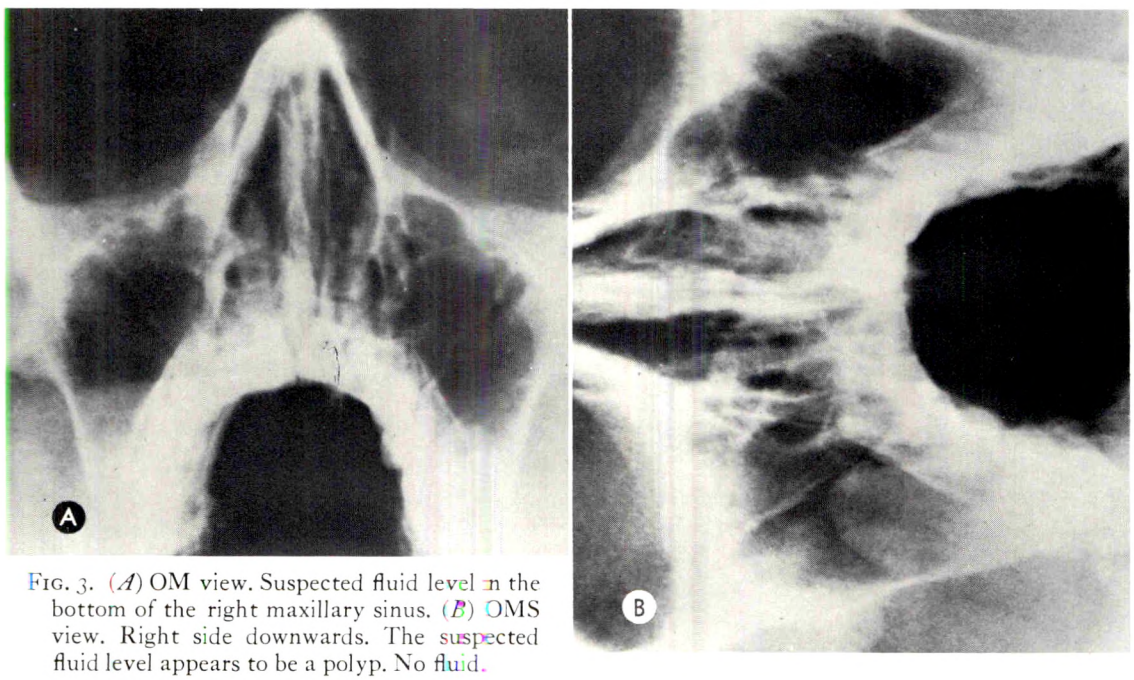


Fig. 3. (*A*) OM view. Suspected fluid level in the bottom of the right maxillary sinus. (*B*) OMS view. Right side downwards. The suspected fluid level appears to be a polyp. No fluid.

TABLE IV

ACUTE SINUSITIS PROJECTIONS (MAXIMUM)*: SUGGESTED NEW METHOD NEEDED FOR DIAGNOSTIC PURPOSES

Site of changes	Roentgenologic Projection						
	OM	OMS Unilat.	OMS Bilat.	OF	OFS Unilat.	OFS Bilat.	Maximum No. of Roentgenograms
No changes (32%)	+						1
Changes in one maxillary sinus (29%)	+	+					2
Changes in both maxillary sinuses (27%)	+		+				3
Changes in one frontal sinus (<1%)	+			+	+		3
Changes in both frontal sinuses (<1%)	+			+		+	4
Changes in one maxillary sinus and one frontal sinus (4%)	+	+		+	+		4
Changes in one maxillary sinus and both frontal sinuses (<1%)	+	+		+		+	5
Changes in both maxillary sinuses and one frontal sinus (5%)	+		+	+	+		5
Changes in both maxillary sinuses and both frontal sinuses (1%)	+		+	+		+	6

* In those cases where the sinus shows complete opacity in the OM or OF projections, projections with diseased side downward (OMS and OFS) need not be done (43 per cent of all maxillary sinus and 21 per cent of all frontal sinus changes). They may be replaced by the axial (A) projection for determination of the thickness of the bony sinus wall.

of projections for demonstrating sinusitis can be diminished by a considerable degree (from 5.14 to 2.3 roentgenograms per patient) with the new suggested method. A proposal on the roentgenologic performance in suspected sinusitis is given in Table IV. This methodologic simplification presupposes close communication between the referral physician and the radiologist concerning the patient's symptoms and, further, an immediate evaluation of the initial roentgenograms for possible additional sinus projections. According to Margulis,[11] routine views for every roentgenologic examination should be reduced to a minimum and additional views be obtained only as indicated on the basis of a few primary views. Our investigations show that this can be done in roentgenologic diagnosis of sinusitis with increased diagnostic confidence.

CONCLUSION

In the roentgenologic examination of cases of clinically suspected sinusitis, the occipito-mental view should be the basic projection, since it discloses the maxillary sinuses more confidently than the other projections and, additionally, gives reliable information on the frontal sinuses. If the findings are normal, no further projections are needed. Any roentgenologic changes in the maxillary sinus except complete opacity require additional occipito-mental views with the head tilted down towards the diseased side.

The occipito-frontal projection, including side projection, demonstrates the frontal sinuses and the ethmoidal sinuses and should remain a routine projection in suspected frontal sinus changes and in those cases where the evaluation of the frontal sinuses is difficult in the occipito-mental projection.

The lateral projection can be eliminated, since inflammatory changes of the sphenoidal sinuses apparently are rare. If the patient is suspected of sphenoidal sinusitis, the projection obviously should be adopted.

The axial projection does not demonstrate any further changes than those found in the occipito-mental and the occipito-mental side projection. Furthermore, it is the most uncomfortable for the patient and could consequently be dismissed from the routine examination and reserved for those cases where there are doubts about the thickness of the bony wall to the

frontal and maxillary sinuses, *e.g.*, in completely opaque sinuses in the occipitomental and/or occipito-frontal view.

The adoption of this routine procedure not only diminishes time and x-ray film consumption, but has the further advantage of increased reliability in demonstrating the inflammatory roentgenologic sinus change by including the occipitomental side view.

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REFERENCES

1. Axelsson, A., Grebelius, N., Chidekel, N., and Jensen, C. Correlation between raciological examination and irrigation findings in maxillary sinusitis. *Acta oto-laryng.*, 1970, *69*, 302–306.
2. Axelsson, A. Produktvärdering av sinusröntgenundersökningar. SPRI-project, No. 7104, 1974.
3. Bablik, L. Möglichkeiten und Grenzen der antibiotischen Therapie der Ohren-, Naser-, und Halskrankheiten. *HNO*, 1958, *6*, 321–330.
4. Ballantyne, J. C. On relationship between radiological appearances and proof-puncture findings in suspected cases of infection of maxillary antrum. *J. Laryng. & Otol.*, 1946, *61*, 306–310.
5. Ballantyne, J. C., and Rowe, A. K. Some points in pathology, diagnosis, and treatment of chronic maxillary sinusitis. *J. Laryng. & Otol.*, 1949, *63*, 337–342.
6. Buch, A. Non-diagnosed maxillary sinusitis. *Acta oto-laryng.*, 1949, Suppl. 77, pp. 1–17.
7. Chidekel, N., Jensen, C., Axelsson, A., and Grebelius, N. Diagnosis of fluid in maxillary sinus. *Acta radiol.* (Diag.), 1970, *10*, 433–440.
8. Härnqvist, M., and Lindholm, B. Öppen sjukvård i Göteborg 1969. SPRI-project No. 7009, 1972.
9. Hinde, R. T. Review of diagnostic problem in one hundred cases of chronic maxillary sinusitis. *J. Laryng. & Otol.*, 1950, *64*, 687–695.
10. Kristensen, H. K. Nordisk lärobok it Oto-rhino-laryngologi. Munksgaard, 1958.
11. Margulis, A. R. Lessons of radiobiology for diagnostic radiology. Caldwell Lecture. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *117*, 741–756.
12. Mattila, K., and Westerholm, N. Round shadows in maxillary sinuses. *Odontologisk Tidskrift*, 1968, *76*, 121–136.
13. McNeil, R. A. Comparison of findings on transillumination, x-ray and lavage of maxillary antrum. *J. Laryng. & Otol.*, 1963, *77*, 1009–1013.
14. Pendergrass, E. P., Schaeffer, J. P., and Hodes, P. J. The Head and Neck in Roentgen Diagnosis. Charles C Thomas, Publisher, Springfield, Ill., 1956.
15. Sanders, S. H. Confused problem of sinus disease in today's practice of otolaryngology. *Tr. Am. Acad. Ophth.*, 1964, *68*, 456–469.
16. Teichert, Chronische sinusitis und Allergie. *HNO*, 1968, *16*, 266–267.
17. Tzschoppe, A., Gresenz, P., Roth, U., and Heidemüller, B. Das poliklinische Krankengut des Hals-Nasen-Ohren Arrtes. *Deutsche Gesundh. Wes.*, 1970, *25/17*, 654–657.
18. Vuorinen, P., Kauppila, A., and Pulkkinen, K. Comparisons of results of roentgen examination and puncture and irrigation of maxillary sinus. *J. Laryng. & Otol.*, 1962, *76*, 359–364.

ANGIOGRAPHY OF EPENDYMOMAS OF THE SPINAL CORD AND FILUM TERMINALE

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EPENDYMOMAS are the most frequent intramedullary tumors of the spinal cord and filum terminale. In the unrivaled series of cases collected at the Mayo Clinic over a period of 45 years of 301 intramedullary tumors, 169 (more than 50 per cent) were ependymomas.[11]

Arteriography of the spinal cord has been rewarding in some types of intramedullary tumors, particularly the hemangioblastomas which often present with pathognomonic angiographic features.[1,3] In the glioma group, however, the findings have been meager.[2,5] Fauré *et al.*[7] reported the displacement of the anterior spinal artery in a 13 year old girl with a thoracic cord astrocytoma. Goldenberg[8] identified pathologic vessels in a case of cervical cord astrocytoma Grade III in a 47 year old male. This has been the only reported observation of abnormal angiographic vascularization in a cord glioma.

Two cases of dorsolumbar ependymoma have been studied angiographically by Djindjian.[6] In one of these 2 patients the enlarged posterolateral spinal arteries were "... displaced further from the midline than normally giving the appearance of a widened cord." In this and in the second patient dilated retromedullary veins, probably the posterior spinals, were noted in the area of the tumor. No pathologic vessels, however, were observed in these 2 ependymomas whose finer histology was not mentioned.

MATERIAL

The present study deals with the angiographic findings which we have observed in 4 cases of ependymoma of the spinal cord, filum terminale and cauda equina. The patients were all males, ages 32, 23, 13, and 11 years. Two of our ependymomas involved the conus and the cauda, one the lower thoracic cord and conus, and the fourth extended for the entire length of the cord. Each one of the 4 patients came to our observation after explorative surgery had been carried out. At operation, an attempt at removal of the tumor had been made in 3 cases, whereas in the fourth patient the cord had only been exposed, and the final diagnosis had not been made. This last patient was in fact referred to us as a possible case of arteriovenous malformation of the spinal cord. In 2 of the patients radiotherapy had also been used prior to our observation and angiographic evaluation. Myxopapillary type ependymoma[9] was the final histologic diagnosis in 3 cases. In the fourth patient, an 11 year old boy with involvement of the entire cord, the tumor was described differently by 3 neuropathologists: cellular type ependymoma with Grade III malignancy,[10] ependymoblastoma, and malignant subependymoma.

Plain spine roentgenograms were within normal limits in 1 patient (the oldest), whereas widening of the spinal canal and scoliosis were found in 3 cases. Total or subtotal subarachnoidal block with expansion of the cord above or below were shown by pantopaque myelography. The positive contrast medium, which was introduced by the lumbar and/or cisternal routes, distributed itself in a pattern characteristic for intramedullary lesions.

ANGIOGRAPHIC FINDINGS

The arteriography was carried out by the selective technique.[4] The angiographic evaluation was limited to the thoracolumbar

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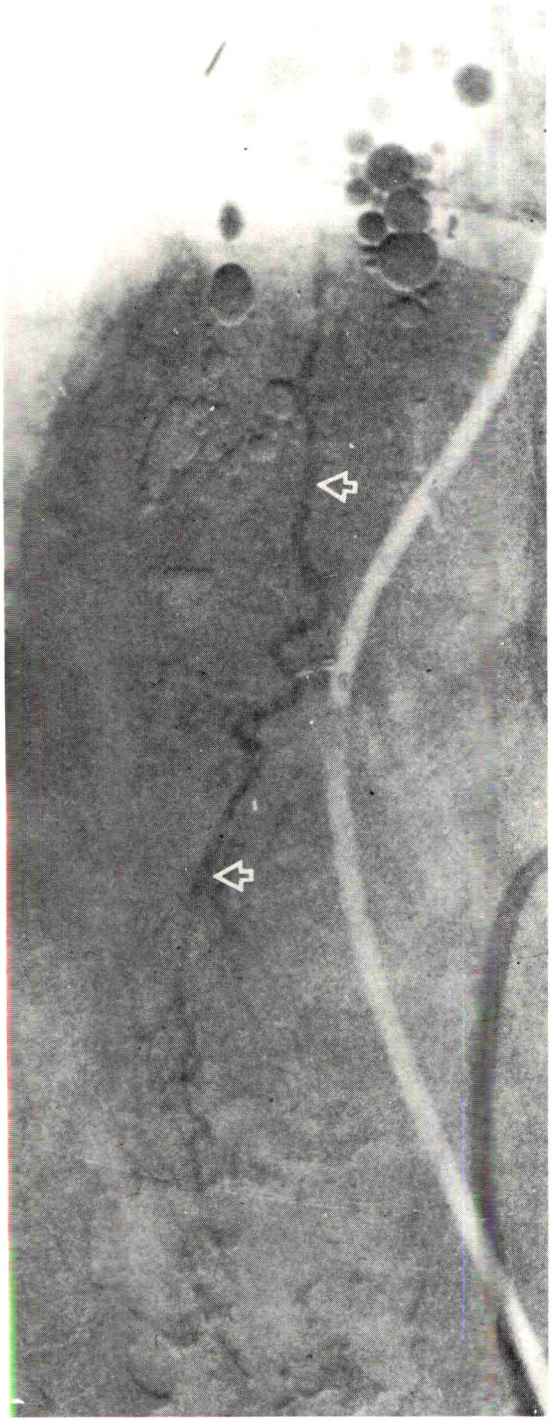


FIG. 1. Markedly enlarged anterior spinal artery (upper arrow). Its tortuosity is not necessarily pathologic in a young patient. Displacement of its inferior part (lower arrow) is due to scoliosis, but also to tumoral mass effect. Recurrent myxopapillary ependymoma.

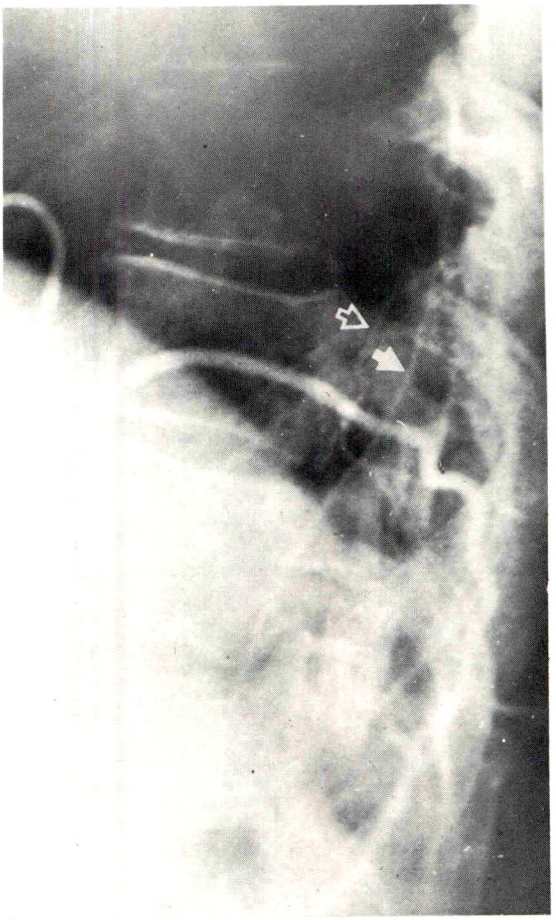


FIG. 2. Massive posterior displacement of anterior spinal artery (solid arrow) in case of recurrent myxopapillary ependymoma of the lower cord. Artery of Adamkiewicz (open arrow) is located in front of anterior spinal, rather than posterior to it as in normal cases.

area, even in the patient in whom, eventually, involvement of the entire cord was demonstrated. The abnormal angiographic findings were of 6 types: (1) enlarged cord arteries; (2) displacement of these arteries; (3) pathologic vessels; (4) striking visualization of the anastomotic loop(s) of the conus as an expression of increased communication from the anterior to the posterolateral arteries; (5) large tortuous draining veins; and (6) slow circulation.

The anterior spinal artery was markedly enlarged in the 3 cases of myxopapillary ependymoma (Fig. 1). This artery was displaced away from the midline in 2 and

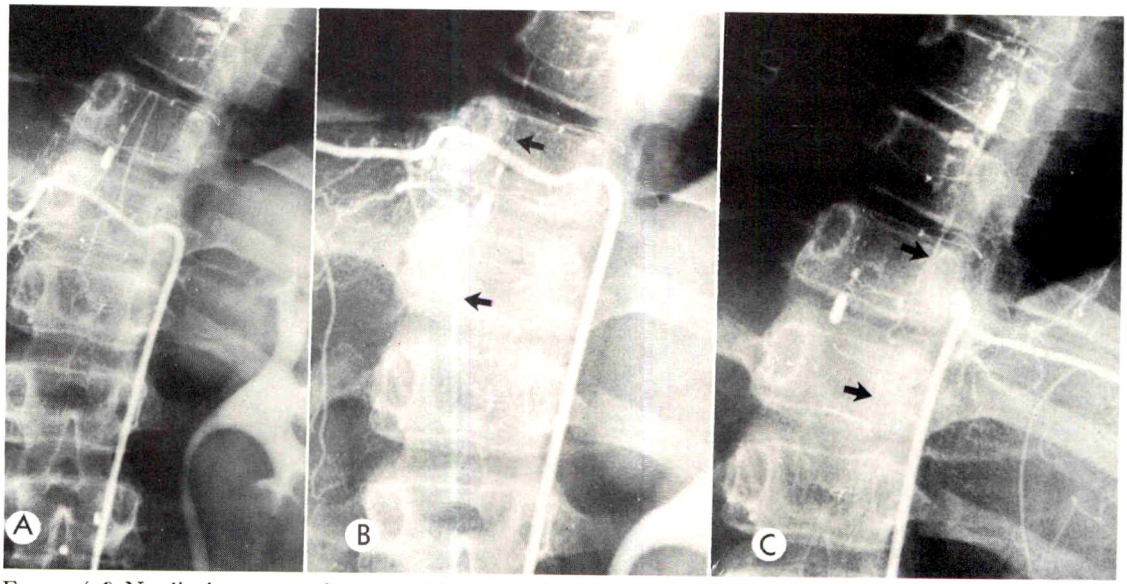


FIG. 3. (*A*) No displacement of artery of Adamkiewicz (originating from XI right intercostal) and anterior spinal artery is present. The (*B*) right and (*C*) left posterolateral spinal arteries, each filling from separate contributors (X right and X left intercostals), are displaced laterally (arrows). At surgery cystic fluid collection at this site was found. Ependymoblastoma.

markedly posterior (Fig. 2) in 1 of these patients. In the young boy with ependymoblastoma, widened posterolateral spinal arteries were displaced laterally indicating expansion of the cord (Fig. 3). Pathologic tumoral vessels were shown in 1 of the patients with myxopapillary ependymoma (Fig. 4). Unusually clear visualization of

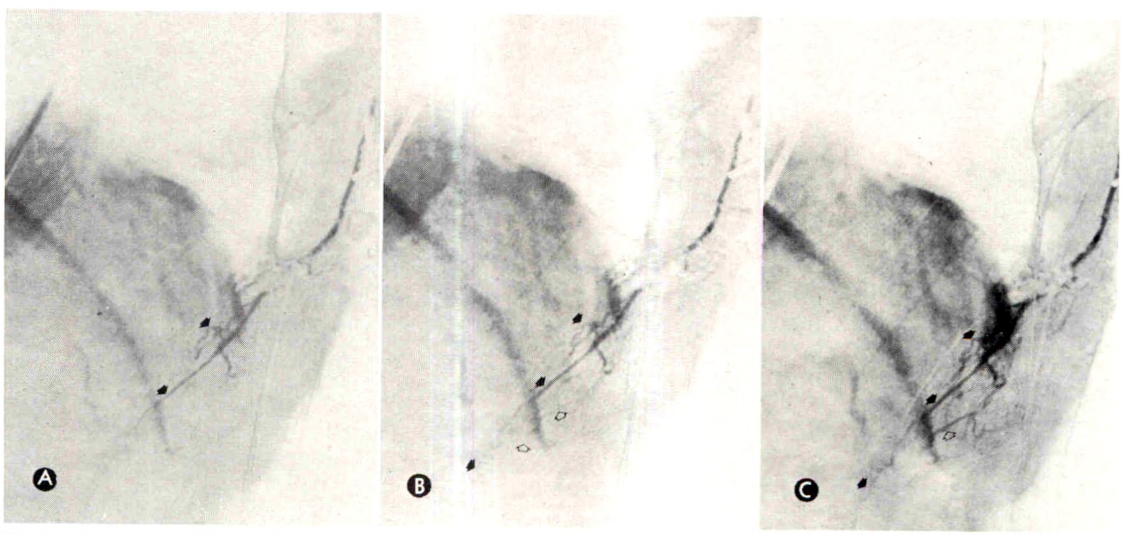


FIG. 4. Lateral serial arteriograms. Patient markedly kyphoscoliotic. (*A*) Pathologic tumoral vessels (upper arrow). Anterior spinal artery (lower arrow). (*B*) Pathologic tumoral vessels (upper solid arrow), anterior spinal artery (center solid arrow) and artery of filum terminale (lower solid arrow). Anastomotic loop(s) and posterolateral spinal arteries (open arrows). (*C*) Pathologic tumoral vessels (upper solid arrow), anterior spinal artery (center solid arrow) and artery of filum terminale (lower solid arrow). Posterolateral spinal arteries (open arrow). Recurrent myxopapillary ependymoma.

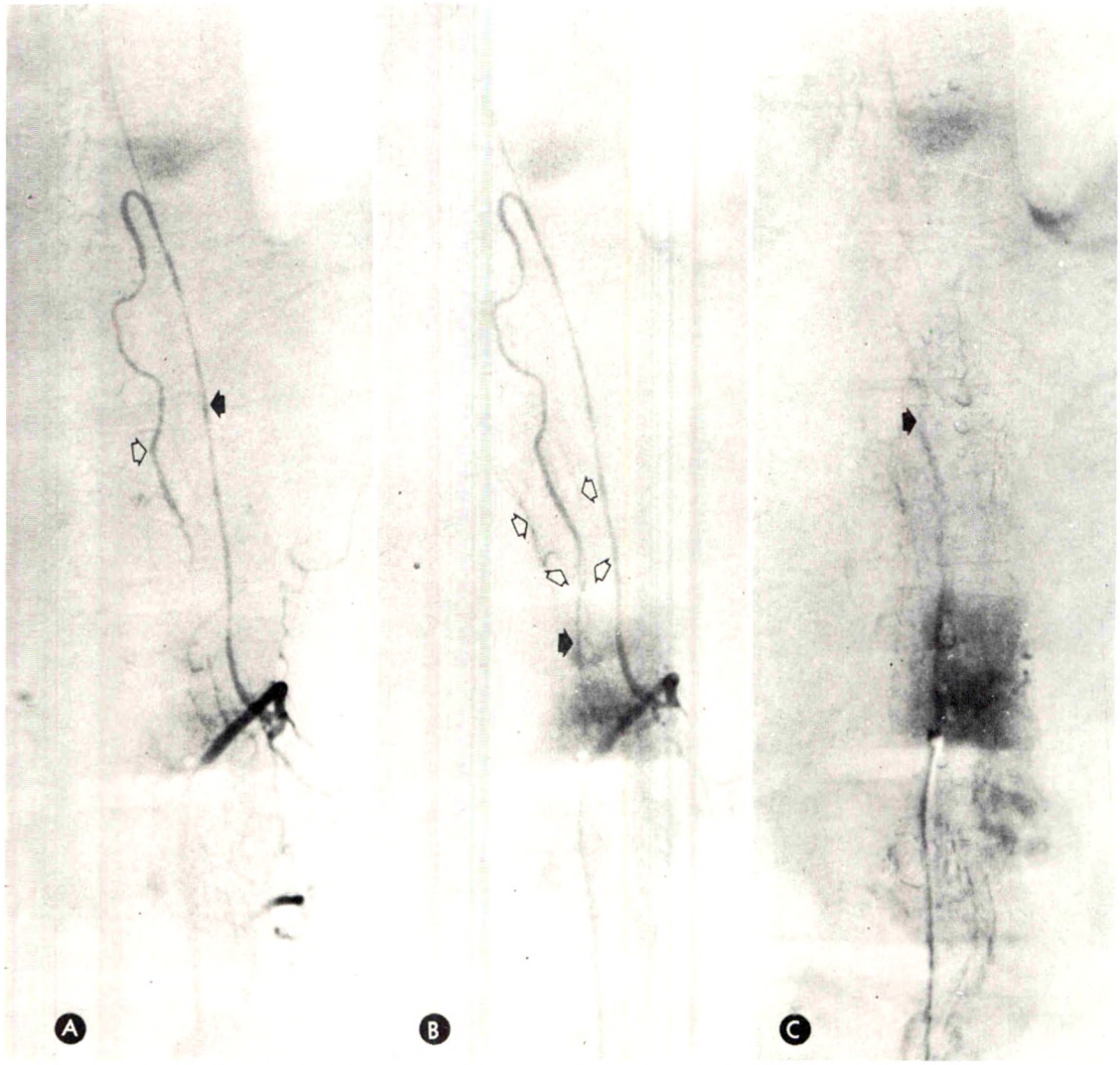


Fig. 5. Serial arteriograns. (*A*) Artery of Adamkiewicz (solid arrow). Large anterior spinal artery (open arrow). Its sinuosity is not necessarily pathologic in a young patient. (*B*) Anastomotic loops and right and left posterolateral arteries (open arrows) clearly outline the conus which probably is distorted on its axis. Large artery of the filum terminale (solid arrow). (*C*) Large, tortuous draining vein(s) (arrow). Myxopapillary ependymoma.

the anastomotic loop(s) around the conus (Fig. 4; and 5) and marked slow circulation were present in the 3 cases of myxopapillary ependymoma. Large, tortuous draining veins were present in 2 of these 3 cases.

DISCUSSION

The above described angiographic findings in ependymomas of the spinal cord, filum terminale and cauda do not appear to be pathognomonic. Some features, however, such as the marked enlargement of the spinal arteries and the striking demonstration of the anastomotic loop(s) are quite characteristic.

It may be of some interest to make a comparison with our angiographic findings in 4 other patients with gliomas other than ependymomas, namely low Grade I or II astrocytomas. In 1 cervical astrocytoma we could not demonstrate the cord vasculature, whereas in 2 cervicothoracic astrocy-

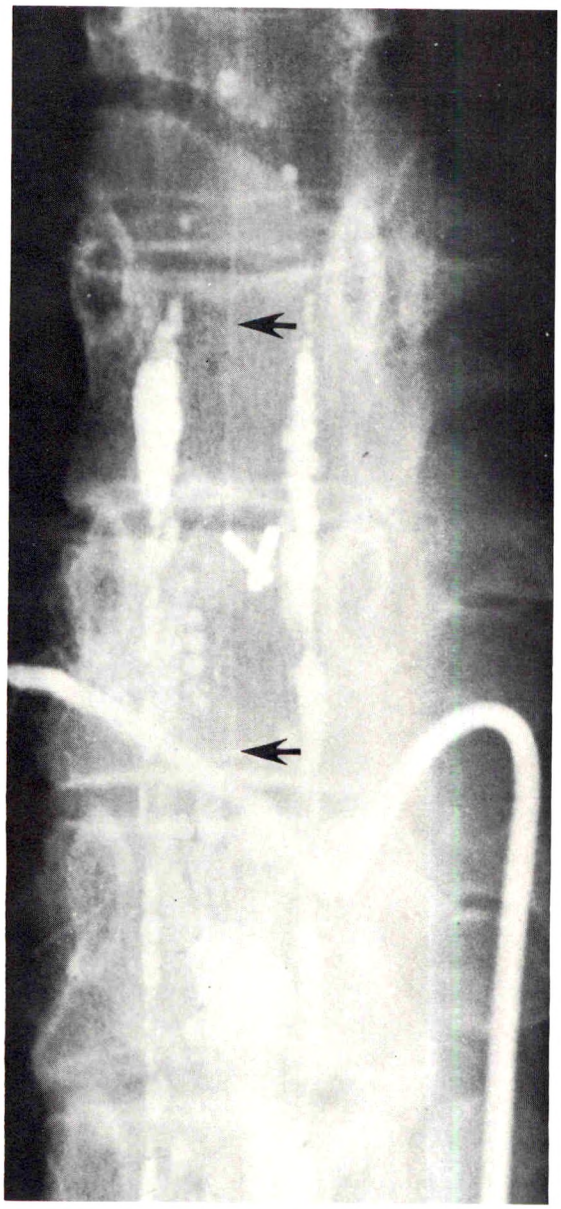


FIG. 6. Normal thoracic anterior spinal artery (arrows) at the site (laminectomy) of cervico-thoracic astrocytoma Grade II.

tomas, the cord vessels appeared normal (Fig. 6). In a case of thoracolumbar astrocytoma the vessels were also normal.

Considering that the removal of the ependymomas, particularly if the operating microscope is used, is more often successful than in cases of astrocytomas,[12] the surgeon may find the above angiographic information of some significance. Arteriography may also offer additional knowledge about the tumor extent and particularly regarding the location of the large vessels in relationship to the neoplastic mass. We have noted that the main arteries are frequently displaced. The angiograms may help in differentiating between infiltrating and encapsulated tumors, as well as in establishing whether large cysts are contained in the cord. The splaying of the posterolateral spinal arteries seen in 1 of our cases corresponded to an area where a large cystic collection was found at surgery. It is too early to make statements regarding the possible value of the pathologic vessels for the specific diagnosis of ependymoma or for the differential diagnosis between the various types of ependymomas. Our case of myxopapillary ependymoma with pathologic vessels was a recurrent tumor after attempted surgical removal and radiotherapy.

SUMMARY

The angiographic findings in 4 cases of spinal cord ependymoma, three of the myxopapillary type, are reported.

Enlargement and displacement of the anterior spinal artery, striking visualization of the anastomotic loop(s) around the conus, and, in 1 case, pathologic vessels have been observed.

The findings, although not pathognomonic, are significant, particularly considering that they were not evident in 4 other patients with cord gliomas other than ependymomas.

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REFERENCES

1. DI CHIRO, G. Combined retino-cerebellar angiomatosis and deep cervical angiomas: case report. *J. Neurosurg.*, 1957, *14*, 685–687.
2. DI CHIRO, G. Recent successes and failures in radiographic and radioisotopic angiography of spinal cord. *Brit. J. Radiol.*, 1972, *45*, 553–560.

3. Di Chiro, G., and Doppman, J. L. Differential angiographic features of hemangioblastomas and arteriovenous malformations of spinal cord. *Radiology*, 1969, *93*, 25–30.
4. Di Chiro, G., Doppman, J., and Ommaya, A. K. Selective arteriography of arteriovenous aneurysms of spinal cord. *Radiology*, 1967, *88*, 1065–1077.
5. Di Chiro, G., and Wener, L. Angiography of spinal cord: review of contemporary techniques and applications. *J. Neurosurg.*, 1973, *39*, 1–29.
6. Djindjian, R. Angiography of the Spinal Cord. University Park Press, Baltimore, 1970.
7. Fauré, C., Lefebvre, J., and Debrun, G. La vascularization artérielle normale et pathologique du renflement lombaire de la moëlle épinière chez l'enfant: l'artère d'Adamkiewicz. *Ann. de radiol.*, 1967, *10*, 129–140.
8. Goldenberg, M. Glioma of spinal cord demonstrated by angiography. *J. Canad. A. Radiologists*, 1970, *21*, 113–115.
9. Kernohan, J. W., and Fletcher-Kernohan, E. M. Ependymomas: study of 109 cases. *Proc. A. Res. Nerv. & Ment. Dis.*, 1937, *16*, 182–209.
10. Mabon, R. F., Svien, H. J., Kernohan, J. W., and Craig, W. M. Ependymomas. *Proc. Staff Meet. Mayo Clin.*, 1949, *24*, 65–71.
11. Sloof, J. L., Kernohan, J. W., and MacCarty, C. S. Primary Intramedullary Tumors of the Spinal Cord and Filum Terminale. W. B. Saunders Company, Philadelphia, 1964.
12. Yasargil, M. G. Vertebral column and spinal cord lesions. In: Microsurgery Applied to Neurosurgery. Edited by M. G. Yasargil. Georg Thieme Verlag, Stuttgart, 1969, pp. 167–178.

TRAUMATIC DEFECTS OF THE PARS INTERARTICULARIS OF THE LUMBAR VERTEBRAE

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IN the recent literature there have been relatively few cases reported of trauma causing defects in the pars interarticularis (isthmus) of the lumbar vertebrae.

Roche[16] was probably the first to report a case in 1948. Wiltse in 1957[22] reported a similar case and again in 1962,[23] when 2 of 3 fractures in 1 patient were described. Sullivan and Beckell[20] reported 3 cases in 1960 with separation of the pars interarticularis due to severe trauma which healed with immobilization. Hadley[6–8] reported 1 in 1963 and 2 earlier, Melamed[13] 1 in 1964, and Schneider and Melamed in 1957.[19] Russell and Nakata[18] reported 1 case in 1968, and Beeler[2] 1 in 1970. Beller and Kirsch[3] reported spondylolisthesis in 2 cases at the level above a lumbosacral fusion in 1964. Anderson[1] also reported spondyloschisis following spinal fusion in 1956. This particular sequel to a lumbar fusion was reported by Harris and Wiley in 1963.[9]

There has been a sprinkling of other reports[4,11,15,17] and undoubtedly there are many more unreported cases.

This report is of 2 cases, both of which had intact isthmus demonstrated on one side before the injury, followed by an isthmian defect bilaterally after injury.

REPORT OF CASES

CASE I. A 16 year old boy injured his lower back playing soccer 1 year before. Roentgenographic examination was reported negative. He was treated by whirlpool and local analgesics and had no more difficulty until 1 year later when he developed low back pain, especially on the right side while running. This became progressively worse. The findings were: fairly good range of back motion; tenderness to right L5; limitation of straight leg raising on the right at 65°, no limitation on the left. There was hypesthesia of the lateral aspect of the right foot, and a suggestion of decreased right ankle jerk. He was admitted to the hospital and roentgenograms were taken of the lower back on March 6, 1972, which showed no defects of pars interarticularis on the right, but a suggestion on the left (Fig. 1, *A–C*). On March 15, 1972, bilateral isthmian defects were demonstrated, but no displacement of the vertebrae (Fig. 2, *A* and *B*). A myelogram at that time was negative. Patient was treated with a brace and he has improved but has developed pain on heavy lifting. On June 6, 1972, roentgenograms showed the isthmian defects to be more definite and a beginning first degree spondylolisthesis had developed (Fig. 3, *A–C*). He continued to improve symptomatically and on June 11, 1973 had very little, if any, back pain. Physical examination was negative. Roentgenograms, however, revealed bilateral defects, probably undergoing fibrous union, and the first degree spondylolisthesis was now more definite (Fig. 4, *A–C*).

CASE II. A 17 year old white male, active in sports, felt well until September 1969, at which time he twisted his back while playing football, with pain lasting 2 to 3 days. In October 1969 he again twisted his back, with pain for 1 to 2 days. In November 1969 there was another football injury with immediate severe pain in the lower back. He was unable to walk for several days. Roentgenograms showed a hair line defect of the left L5 pars interarticularis and none on the right (Fig. 5, *A–C*). The patient was advised to give up active participation in sports, but ignored the advice. Roentgenograms on January 6, 1970 showed a broad defect bilaterally of the pars interarticularis (Fig. 6, *A–C*). As of May 13, 1970, the patient had continued to be active in sports. Only slight spasm was noted at this time and there was no clinical disability. In January 1971, tomograms of the pars interarticularis showed nonunion without forward slippage. In June 1973 the pa-

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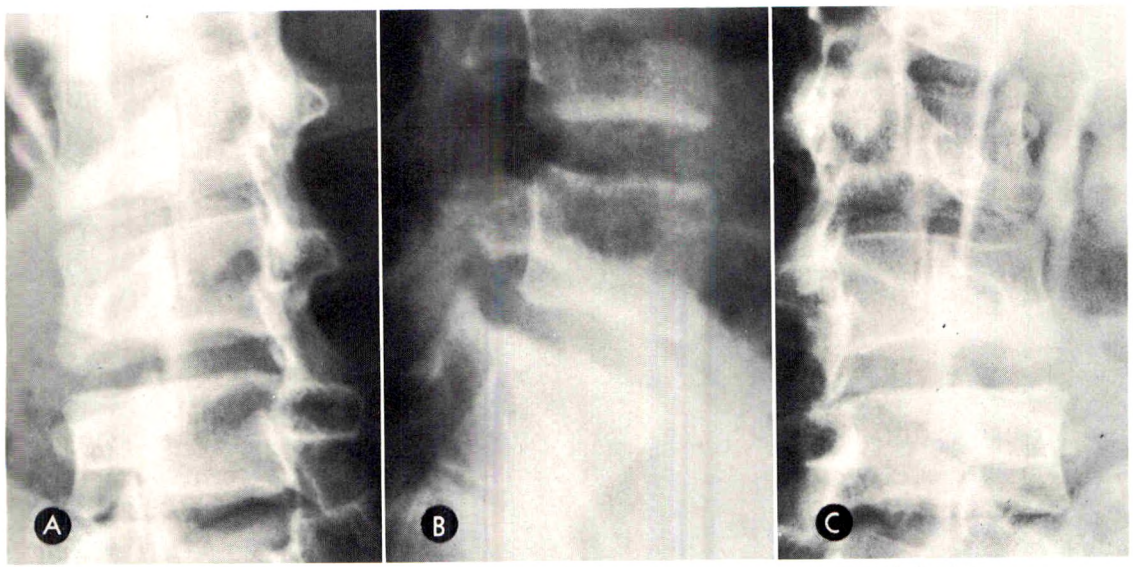


FIG. 1. Case 1. (*A–C*) *March 6, 1972.* First roentgenograms show no isthmian defect on the right and a suggestion of one on the left.

tient continued to be active in sports and had occasional recurrent back pain. Roentgenograms on June 29, 1973, showed continued nonunion and a suggestion of slight forward slipping of L_5 (Fig. 7, *A–C*).

DISCUSSION

In the early literature few authors entertained the idea that defects in the pars interarticularis were due to trauma. They

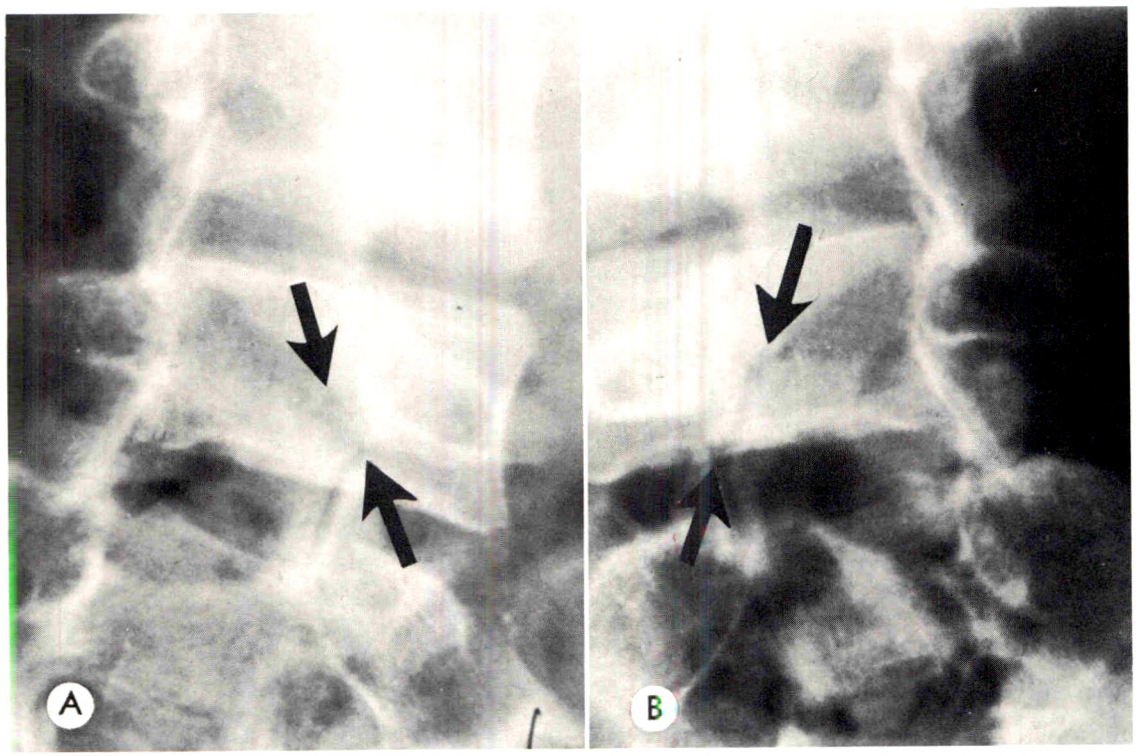


FIG. 2. Case 1. (*A* and *B*) *March 15, 1972.* Bilateral isthmian defects are demonstrated.

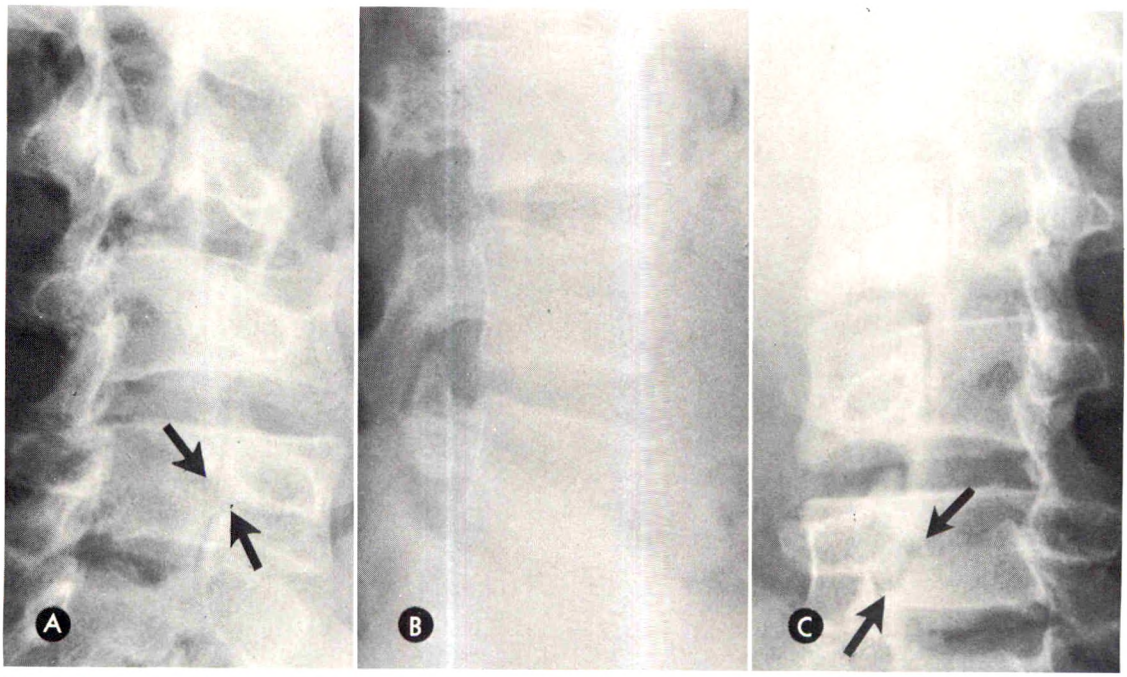


FIG. 3. Case 1. (*A–C*) *June 6, 1972.* Isthmian defects have become more definite and there is now a very minimal forward slipping of L5 on S1 not present on March 6, 1972.

usually considered it a congenital defect, but this theory has been discarded because of lack of demonstration of the lesion in the fetus and newborn and its presence in the adolescent and adult in a fairly constant proportion of individuals, 5–6 per cent.[21] Excluding violent injury in which the cause of a luxation or fracture luxation of the vertebra is obvious, Brocher's theory,[7] in regarding them as dysplasias of the vertebral arch analogous to dysplasias of hip, is pertinent. In this situation, which does not become evident until after birth, the defect in the pars interarticularis represents a frequent but not necessary occurrence. Certainly, changes similar to a March fracture and the changes occurring in bone from certain repeated stresses such as described by Looser,[12] Milkman,[14] and Brandt[4] must be considered as a possible

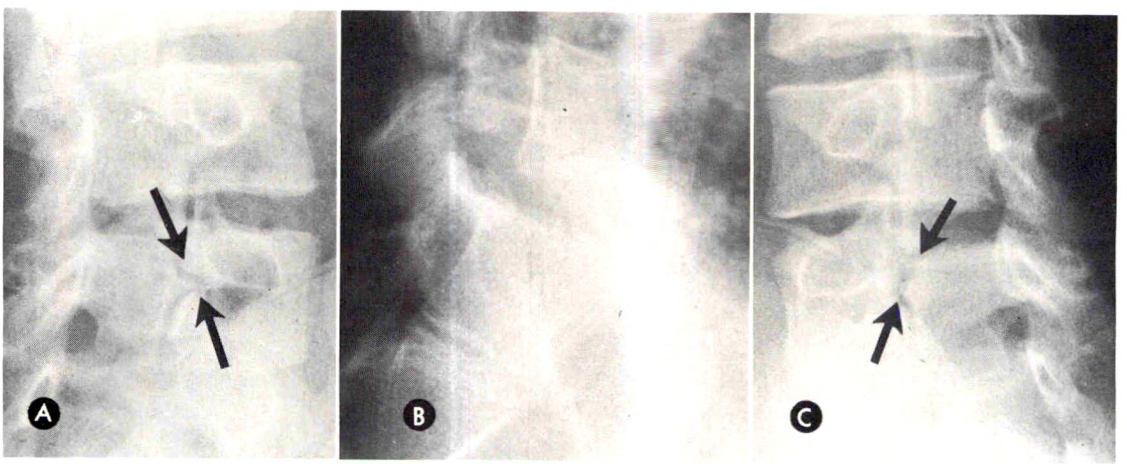


FIG. 4. Case 1. (*A–C*) *June 11, 1973.* The isthmian defects show no healing. The edges are smooth, and there is now first degree spondylolisthesis.

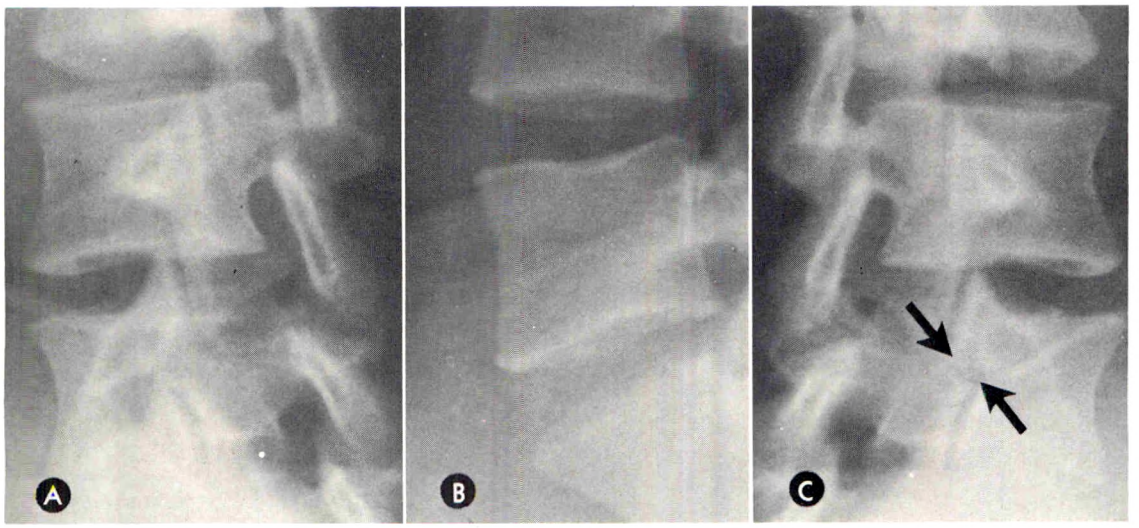


FIG. 5. Case II. (*A–C*) *November 24, 1969.* After several instances of trauma, an isthmian defect is suspected on the left, none on the right.

cause. The alteration in the mechanism of motion following lumbar spine fusion has on occasion been followed by spondylolisthesis at the level above with defects in the isthmus.[1,3,9]

The isolated cases of fractures of the pars interarticularis with or without slipping in the literature follow, in most instances, the same pattern as the 2 cases reported here: adolescent athletic males active in sports and with repeated instances of minor injuries to the back during their growing period. Only the happenstance of having had roentgenographic examination of the lumbar spine before the fractures occurred proves the cause and effect in this particular group of cases.

CONCLUSIONS

1. Spondylolisthesis has been shown not to be a congenital defect.
2. Evidence points to the causes as being:
   a. possible dysplasia of the vertebral arch analogous to dysplasia of the hip
   b. Changes similar to a March fracture with repeated injuries
   c. Altered mechanism following spinal fusion
   d. Severe trauma

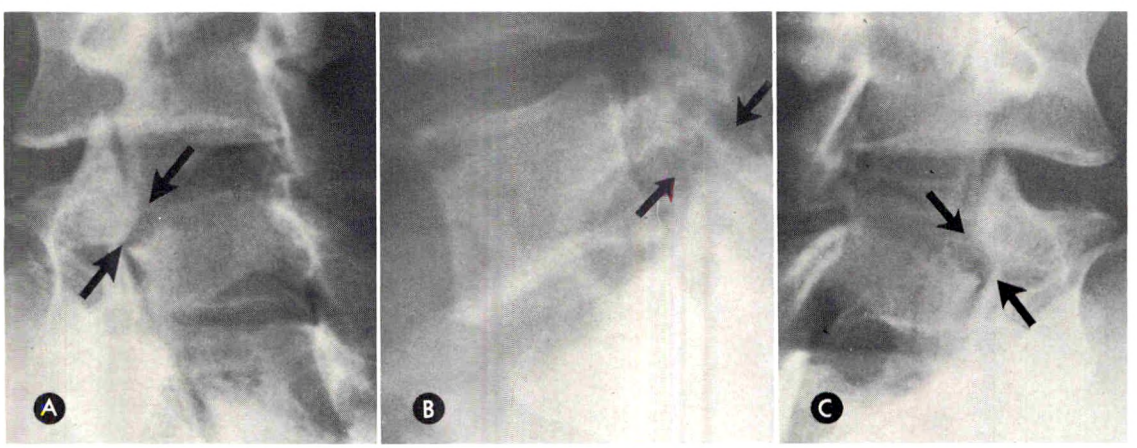


FIG. 6. Case II. (*A–C*) *January 6, 1970.* The defects are now broad and definite bilaterally. There is no forward displacement.

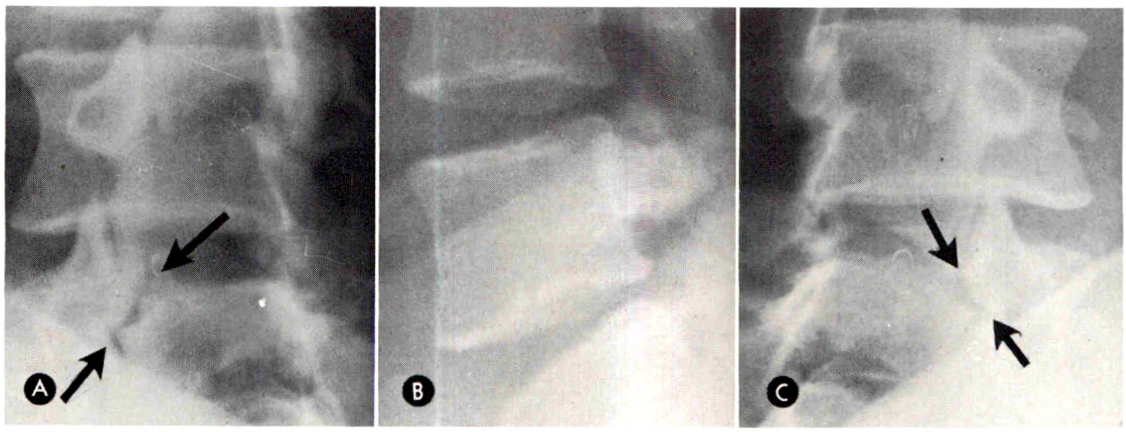


FIG. 7. Case II. (*A–C*) *June 29, 1973.* The defects are still plainly visible. In comparing the spot, lateral roentgenogram of L5 with the original one on November 24, 1969 a slight forward displacement can be detected.

3. Two cases are reported which probably fall under category 2 b.

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REFERENCES

1. ANDERSON, C. E. Spondyloschisis following spine fusion. *J. Bone & Joint Surg.*, 1956, *38-A*, 1142–1145.
2. BEELER, J. W. Further evidence on acquired nature of spondylolysis and spondylolisthesis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 796–798.
3. BELLER, H. E., and KIRSCH, D. Spondylolisthesis and spondylolysis following back fusions. *South M. J.*, 1964, *57*, 783–786.
4. BRANDT, G. Creeping fractures: transformation zones, overload injuries. *Ergebn. d. Chir. u. Orthoped.*, 1941, *33*, 1–59.
5. GIUNTOLI, L., and GUARESCHI, B. Spondylolisthesis: medico-legal problems of spondylolisthesis in its roentgenological aspects. *Radiol. med.*, 1962, *48*, 833–860.
6. HADLEY, L. A. Bony masses projecting into spinal canal opposite break in neural arch of fifth lumbar vertebra. *J. Bone & Joint Surg.*, 1955, *37-A*, 787–797.
7. HADLEY, L. A. Fatigue fracture of fifth lumbar neural arch. In: Clinical Orthopedics. Edited by A. F. De Palma, Editor-in-Chief. J. B. Lippincott Company, Philadelphia, 1953.
8. HADLEY, L. A. Stress fracture with spondylosis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *90*, 1258–1262.
9. HARRIS, R. I., and WILEY, J. J. Acquired spondylolysis as sequel to spine fusion. *J. Bone & Joint Surg.*, 1963, *45-A*, 1159–1170.
10. KOHLER, A., and ZIMMER, E. A. Borderlands of the Normal and Early Pathologic in Skeletal Roentgenology. Grune & Stratton, Inc., New York, 1956, pp. 362–367.
11. LAMBERT, R. G., and BILLINGS, E. L. Traumatic spondylolisthesis. Paper read at the Annual Meeting of the Western Orthopedic Association, Coronado, California, 1960.
12. LOOSER, E. Über Spätrachitis und Osteomalacie. *Deutsche Ztschr. Chir.*, 1930, *152*, 210–357.
13. MELAMED, A. Fractures of pars interarticularis of lumbar vertebra. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 584–586.
14. MILKMAN, L. A. Multiple spontaneous idiopathic symmetrical fractures. AM. J. ROENTGENOL. & RAD. THERAPY, 1934, *32*, 622–634.
15. NEWMAN, P. H., and STONE, K. H. Etiology of spondylolisthesis. *J. Bone & Joint Surg.*, 1963, *45-B*, 39–59.
16. ROCHE, M. B. Bilateral fracture of pars interarticularis of lumbar neural arch. *J. Bone & Joint Surg.*, 1948, *30-A*, 1005–1008.
17. ROWE, G. G., and ROCHE, M. B. Etiology of separate neural arch. *J. Bone & Joint Surg.*, 1959, *41-A*, 303–320.
18. RUSSELL, W. J., and NAKATA, H. Spondylolysis following trauma: case report. *Radiology*, 1968, *91*, 973–974.
19. SCHNEIDER, C. C., and MELAMED, A. Spondylolysis and spondylolisthesis: case report clarifying etiology of spondylolysis. *Radiology*, 1957, *69*, 863–866.
20. SULLIVAN, C. R., and BECKELL, W. H. Problem of traumatic spondylolysis: report of three cases. *Am. J. Surg.*, 1960, *100*, 698–708.
21. TAILLARD, W. Etiology of spondylolysis. *Schweiz. med. Wchnschr.*, 1963, *93*, 301–304.
22. WILTSE, L. L. Etiology of spondylolisthesis. *Clin. Orthoped.*, 1957, *10*, 48–58.
23. WILTSE, L. L. Etiology of spondylolisthesis. *J. Bone & Joint Surg.*, 1962, *44-A*, 539–560.

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THE APLASTIC HYPOPLASTIC LUMBAR PEDICLE*

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UNILATERAL aplastic and hypoplastic lumbar pedicles are a rare finding thought to represent varying degrees of the same anomaly. Only 2 previous cases of hypoplastic lumbar pedicle have been recorded.[2] This anomaly does not seem to be uncommon in the cervical region.[1,3–10]

Roentgenographic recognition of this anomaly is made from plain film roentgenography and tomography, and distinction from lytic processes of the affected pedicle or osteoblastic lesions of the hypertrophied contralateral pedicle should be made.

The purpose of this communication is to present 2 cases with this rare anomaly.

REPORT OF CASES

CASE I. A 12 year old Caucasian male with a complaint of abdominal pain was referred to this hospital for a plain roentgenogram of the abdomen. Physical examination was entirely normal. Plain film roentgenography demonstrated a faintly visualized left pedicle of the first lumbar vertebra and a slight but definite increase in thickness of the cortical rim of the right pedicle of the same vertebra. This was confirmed by coned views in the anteroposterior projection and tomograms (Fig. 1, *A* and *B*) and oblique projections (Fig. 2, *A* and *B*). The lateral tomograms revealed a localized enlargement of the intervertebral foramen produced by narrowing of the affected pedicle (Fig. 3). Despite the lack of symptoms, a myelography was performed, which revealed no significant abnormalities.

CASE II. A 26 year old Caucasian male was admitted because of chronic vague back pain, with no neurologic deficit. Physical examination was essentially normal except for minimal muscular spasm in the lumbar region. Plain

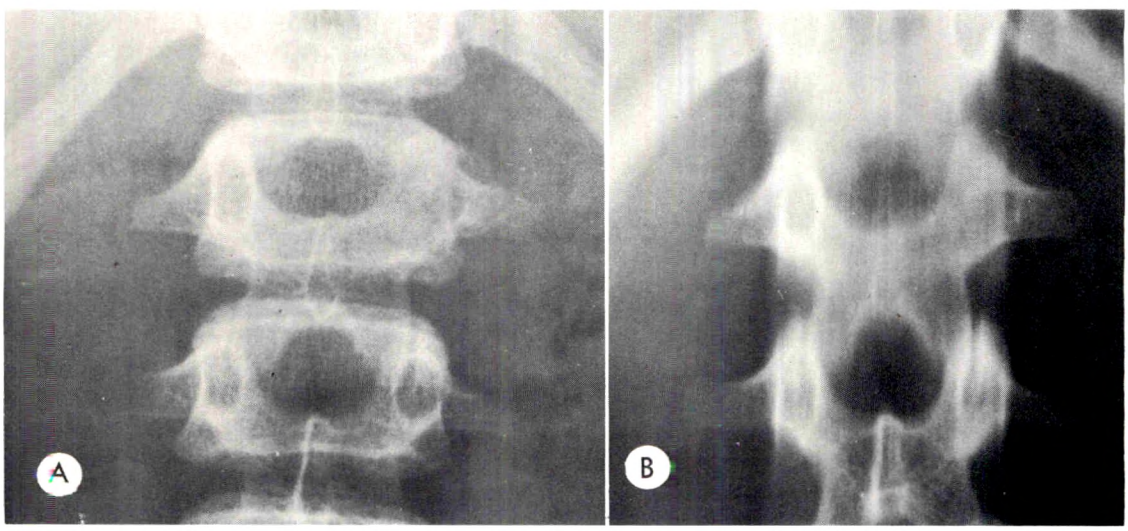


FIG. 1. Case 1. Anteroposterior projections. (*A*) Plain film roentgenogram and (*B*) tomogram demonstrate faint visualization of the left pedicle of the first lumbar vertebra and increased density of the opposite pedicle.

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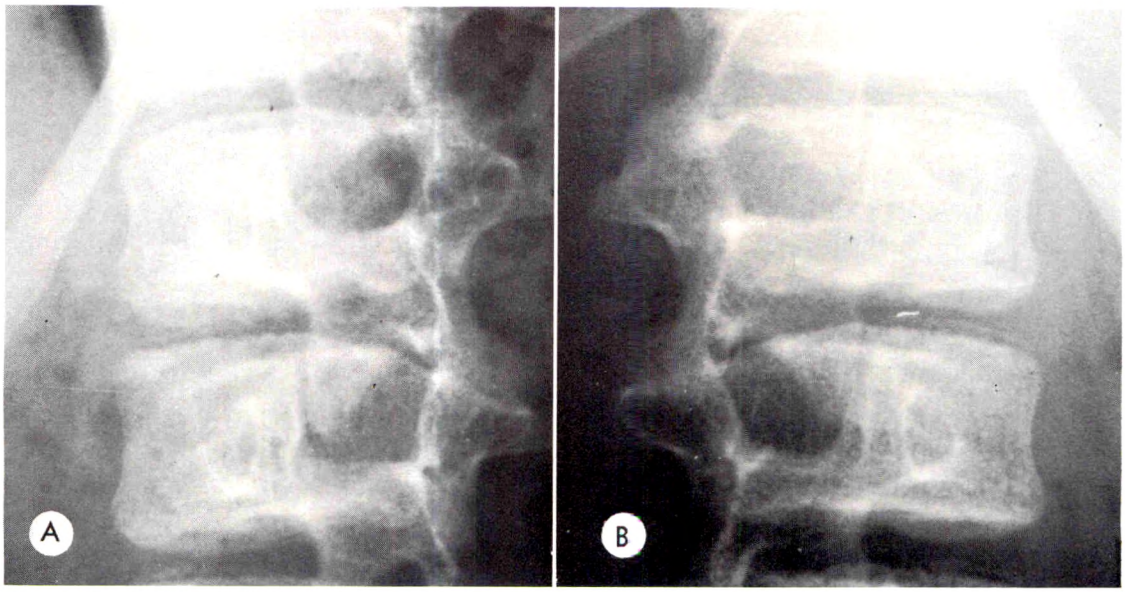


FIG. 2. Case 1. (*A* and *B*) Oblique projections show again the poor visualization of the left pedicle.

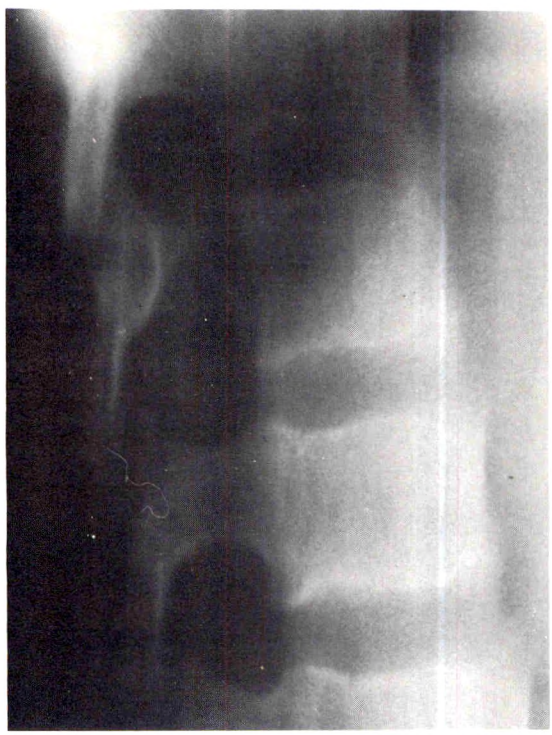


FIG. 3. Case 1. Lateral tomogram reveals a localized enlargement of the intervertebral foramen due to marked narrowing of the left hypoplastic pedicle.

film roentgenography of the lumbar spine revealed marked sclerosis of the left pedicle of the second lumbar vertebra simulating an osteoblastic process and absence of the right pedicle (Fig. 4; and 5, *A* and *B*).

DISCUSSION

Plain roentgenography and tomography permitted the diagnosis of unilateral hypoplastic lumbar pedicle in 2 patients. In Case 1, only a faint cortical rim of the hypoplastic pedicle was demonstrated. Hypertrophy of the cortex of the opposite pedicle was a subtle, but definite finding. Constriction of the affected pedicle could be appreciated on tomography, producing marked enlargement of the neural foramen which led us to perform a myelography to rule out an intraspinal process. In Case 11, there was actually an aplasia of the affected pedicle, and, of course, neither a cortical rim nor a constricted pedicle was demonstrated since none was present. The contiguous normal vertebral elements appeared abruptly terminated. The contralateral pedicle and lamina were markedly hypertrophied.

Unilateral hypoplastic or unilateral

aplastic lumbar pedicles are thought to represent varying degrees of the same anomaly, *i.e.*, an aborted attempt at formation of a vertical cleft in the vertebral arch. In the 2 previously reported cases and the 2 cases presented here, the anomalous pedicle was at L_1 in 2 cases, and at L_2 in 2 cases. Three of the patients were male and 1 female. In 2 of the patients, the anomaly was detected in the third decade (22 and 26 years of age), while the other 2 patients were in the prepubertal age group (12 and 6 years of age). It is of interest to note that those in the first category developed marked contralateral compensatory hypertrophy which simulated osteoblastic process, whereas in the younger age group, they presented with quite subtle, almost equivocal compensatory changes.

There was absence of neurologic signs and symptoms. In our Case 11, the symptom of back pain could be attributable to the anomaly.

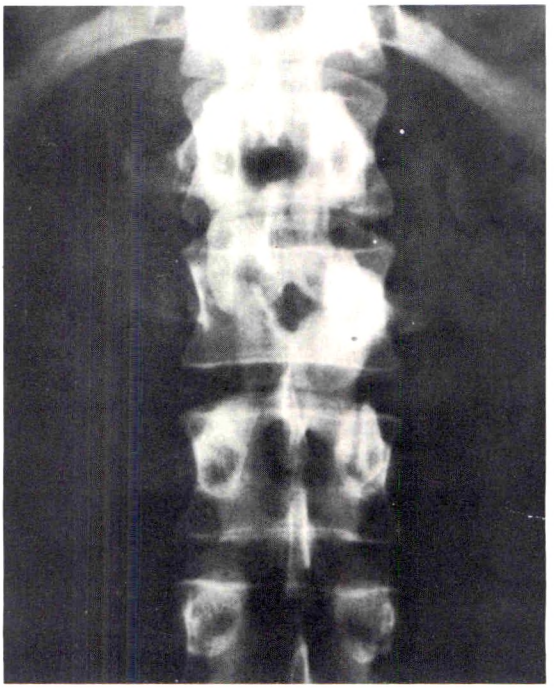


FIG. 4. Case 11. Anteroposterior projection reveals a marked increased density of the left pedicle of the second lumbar vertebra due to hypertrophy and aplastic right pedicle.

SUMMARY

One case of unilateral hypoplastic and 1 case of aplastic lumbar pedicles are pre-

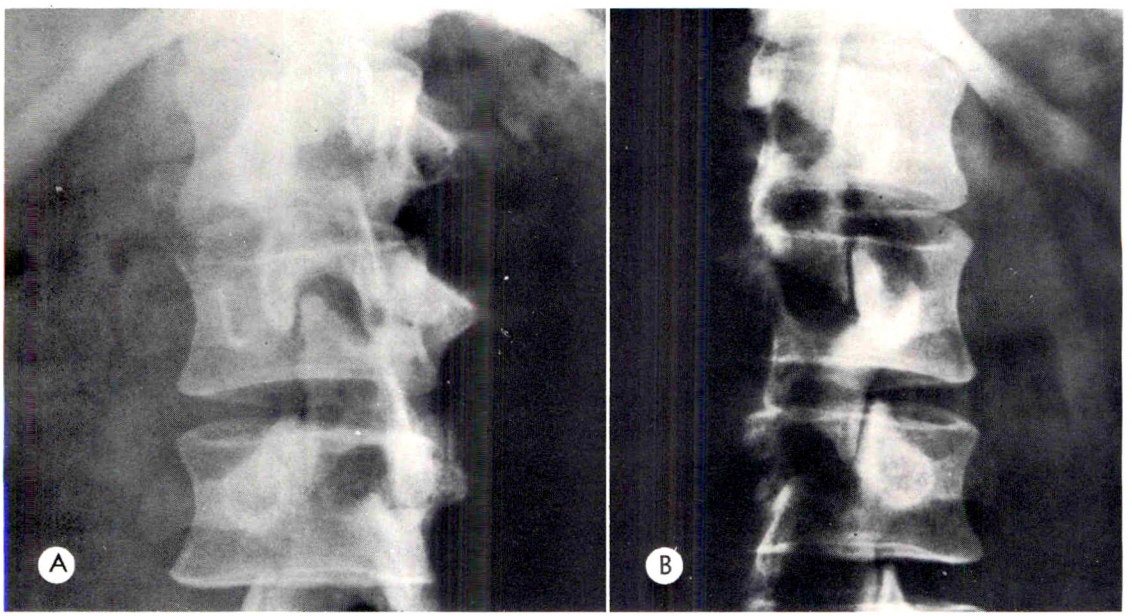


FIG. 5. Case 11. (*A* and *B*) Oblique projections show again the marked hypertrophy of the left pedicle and aplasia of the right.

sented. This anomaly should be recognized as it could be taken for a destructive lesion and the hypertrophy of the contralateral pedicle could be confused with an osteoblastic process.

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REFERENCES

1. Abel, M. S. Moderately severe whiplash injuries of cervical spine and their roentgenologic diagnosis. *Clin. Orthoped.*, 1958, *12*, 189–208.
2. Bardsley, J. L., and Hanelin, L. G. Unilateral hypoplastic lumbar pedicle. *Radiology*, 1971, *101*, 315–317.
3. Gaizler, G., Sr., and Gaizler, G., Jr. Fehlen einer Bogenwurzel und der Halswirbelsäule. *Fortschr. a. d. Geb d. Röntgenstrahlen u. d. Nuklearmedizin*, 1963, *99*, 421–423
4. Grasser, H., and Klein, E. Differentialdiagnose seltener Wirbelsäuleveränderungen. *Ztschr. Orthop.*, 1957, *88*, 550–553.
5. Hadley, L. A. Congenital absence of pedicle from cervical vertebra. Am. J. Roentgenol. & Rad. Therapy, 1946, *55*, 193–197.
6. Louyot, P., Gaucher, A., Laxenaire, M., and Combebias, J. F. Agénésie du pédicule droit de la sixième vertèbra cervicale et syndrome radiculaire C7 par hernie discale. *Rev. du Rhumatisme*, 1966, *34*, 276–279.
7. Oestreich, A. E., and Young, L. W. Absent cervical pedicle syndrome: case in childhood. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 505–510.
8. Steinbach, H. L., Boldrey, E. B., and Sooy, F. A. Congenital absence of pedicle and superior facet from cervical vertebra. *Radiology*, 1952, *59*, 838–840.
9. Wilson, C. B., and Norrell, H. A., Jr. Congenital absence of pedicle in cervical spine. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 639–647.
10. Zatz, L. M., Burgess, P. W., and Hanbery, J. W. Agenesis of pedicle in cervical spine. *J. Neurosurg.*, 1963, *20*, 564–569.

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ANGIOGRAPHIC FEATURES OF ARTERIAL LESIONS IN NEUROFIBROMATOSIS

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VASCULAR lesions due to neurofibromatosis have been reported in 29 patients.[1–15,17,18,20–23] In all of them renal artery stenosis was present. In only a few were the celiac, mesenteric or iliac arteries involved or a coarctation of the abdominal aorta described. Angiographic investigations were performed in 28 of the 29 cases, but no specific angiographic patterns have been described.

Recently, we studied angiographically 3 young patients with vascular neurofibromatosis. The renal arteries were the only site of involvement in 2 of them, but in the third, multiple vascular lesions occurred, including renal, superior mesenteric, external iliac and thoracic outlet arteries, pulmonary arteries and the thoracic aorta. To the best of our knowledge no similar case has been reported before.

We were also impressed by the angiographic patterns observed in all cases. A certain similarity in the angiographic pattern was present which seems to be different from the known angiographic features in other diseases and we believe that they may be considered as highly suggestive for neurofibromatosis.

REPORT OF CASES

CASE I.* An 11 year old boy was admitted to the hospital for investigation of hypertension. The physical examination revealed subcutaneous nodules palpable in the middle part of the right thigh and left ankle and café au lait spots all over the body. Blood pressure was 180/110 mm. Hg. Laboratory test results were within normal limits.

Renal angiography revealed a smooth severe concentric narrowing of the proximal segment of the left renal artery continuing into a gradually increasing funnel-shaped dilatation (Fig. 1).

Corrective surgery was performed and the blood pressure returned to normal limits.

CASE II. A 16 year old boy had been suffering from hypertension for 9 years; he was admitted to the hospital for the investigation of hypertension. The physical examination revealed multiple café au lait spots of varying size and a blood pressure of 170/100 mm. Hg. Intravenous urography was normal. Renal angiography revealed bilateral renal artery stenosis. The stenotic segment of the proximal main left renal artery showed an elongated funnel-shaped appearance with smooth contours continuing into a gradually increasing dilatation. Collateral circulation *via* a branch of superior capsular artery was also present (Fig. 2). The right renal artery revealed generally a similar type of stenosis with funnel-shaped dilatation but of less severity. Moreover, 2 saccular aneurysms with narrow pedicles bulged from the dilated segment of the main right renal artery. The



FIG. 1. Case I. Renal angiography reveals severe concentric narrowing (arrow) of the proximal segment of the left renal artery, continuing into a gradually increasing funnel-shaped dilatation.

* This case was reported previously in detail by Wallis *et al.*[23]

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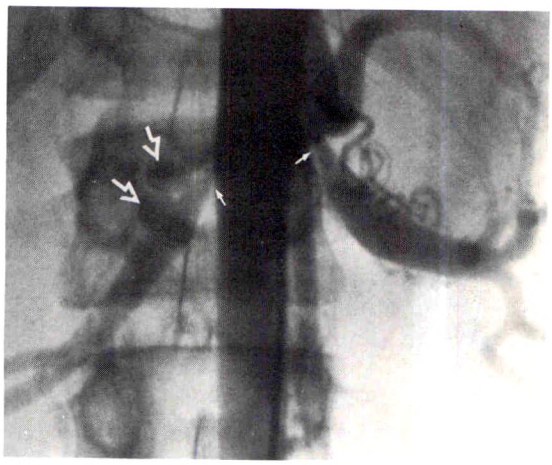


FIG. 2. Case II. Renal angiography reveals bilateral renal artery stenosis (small arrows) and funnel-shaped post-stenotic dilatation. Two saccular aneurysms bulging from the main right renal artery are also seen (open arrows).

patient was discharged on symptomatic antihypertensive therapy.

On follow-up his complaints consisted of occasional bouts of headache and his blood pressure ranged between 130–150/90–100 mm. Hg.

CASE III. A female child of 1 year was admitted with marked hypertension and restlessness, 2 days after falling from her carriage. On physical examination multiple congenital malformations were noted: amputation of all toes of left foot, the upper end of the left lower extremity was thicker as compared to the right, a large hemangioma on the left shoulder extending to the back and multiple café au lait spots over her trunk, abdomen and left arm were observed. The femoral pulses were weak, left more than the right, the blood pressure on her upper extremity was 230/160 mm. Hg and on the right lower extremity 160/90 mm. Hg; on the left lower extremity it could not be measured. A systolic murmur was heard on the left parasternal region. The angiographic examination revealed tubular coarctation of the upper thoracic aorta and multiple stenosed vascular lesions of different severities of the following arteries: left pulmonary artery, innominate artery, left common carotid artery, left subclavian artery, left external iliac artery, celiac artery, superior mesenteric artery, and both renal arteries (Fig. 3, *A* and *B*; 4; 5; and 6). The angiographic appearance of the involved arteries had in common a more or less elongated stenotic segment, gradually increasing in size into a funnel-shaped channel. The latter features were markedly prominent in the renal and left external iliac arteries.

Surgery was considered but the child succumbed and no postmortem examination was available.

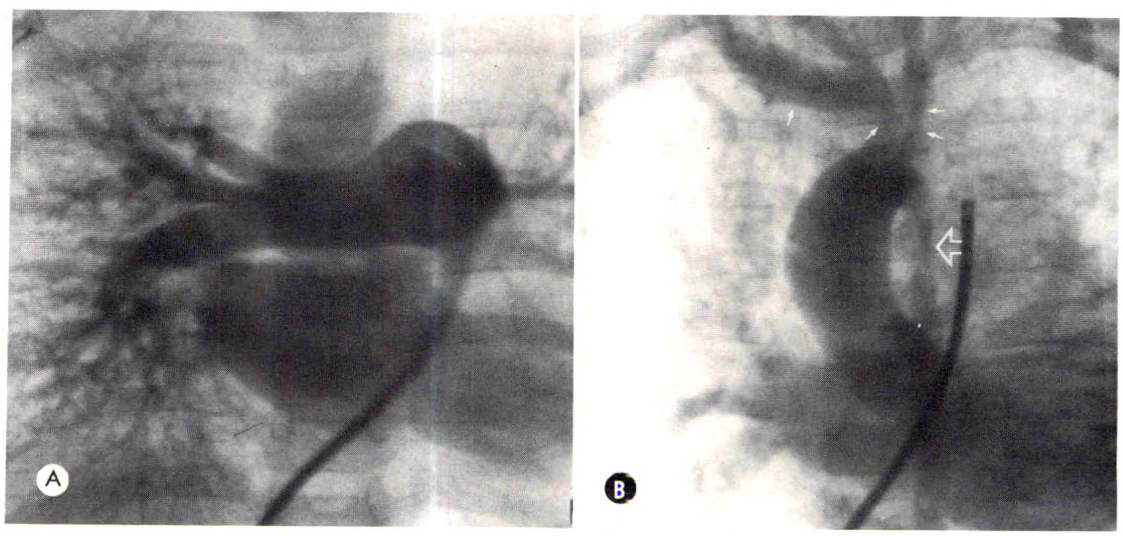


FIG. 3. Case III. (*A* and *B*) Pulmonary angiography. Early phase (*A*): marked hypoplasia of the left pulmonary artery. Levo phase (*B*): tubular coarctation of the upper thoracic aorta (open arrow), stenosis of the innominate artery with funnel-shaped post-stenotic dilatation and stenoses at the origin of the left common carotid and left subclavian arteries are seen (small arrows).

DISCUSSION

The incidence of blood vessel involvement in neurofibromatosis remains unknown at the present time. However, as additional cases are reported it is becoming increasingly evident that the association between renal artery stenosis, renovascular hypertension in young patients and neurofibromatosis is more than coincidental. Reubi[19] claims that vascular neurofibromatosis is always a part of the disease and that if one looks for vascular changes systematically they will be found in all cases.

In the cases reported in the literature the angiograms revealed involvement of different arteries:[1–15,17,18,20–23] renal artery stenosis was present in all cases, unilateral or bilateral; renal artery aneurysm was observed in 4 and coarctation of the abdominal aorta in 6 cases. In the latter cases (with coarctation of the abdominal aorta) stenoses of the celiac axis and the origin of the superior mesenteric artery were also present. Iliac artery stenosis was encountered in only 1 case. The age of the patients at the time of diagnosis ranged from 1½ to 28 years; 18 were less than 12 years old.

In the first review of ten patients by Halpern and Currarino,[13] 4 were less than 12 years old and none of them had coarctation of the abdominal aorta, while in 5 of the older patients (12, 19, 21, 25, 26 years old) coarctation of the abdominal aorta was present. The pathologic findings in the latter cases were neurofibromas, abundant fibrous adventitial tissue or highly differentiated ganglioneuromatous tissue adherent to the aortic wall. In the additional 19 cases reported later,[1,3,4,7,9,10,12,15,17,18,22,23] 14 were below the age of 12 years and in only 1 of the older patients was coarctation of the abdominal aorta described.[18] It seems that coarctation of the abdominal aorta is diagnosed mainly in those patients

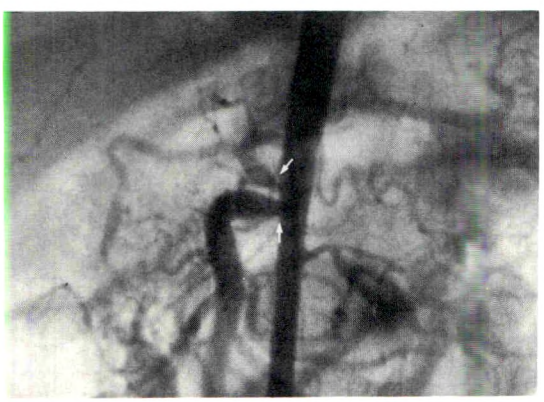


Fig. 4. Case III. Stenosis at the origin of superior mesenteric and celiac artery (arrows).

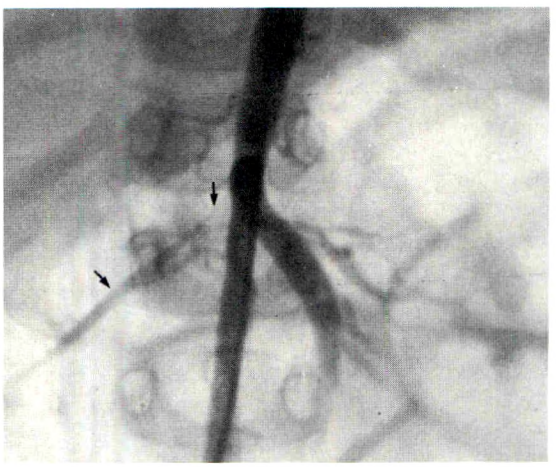


Fig. 5. Case III. Stenosis and elongated funnel-shaped post-stenotic dilatation of the main right renal artery (arrows).

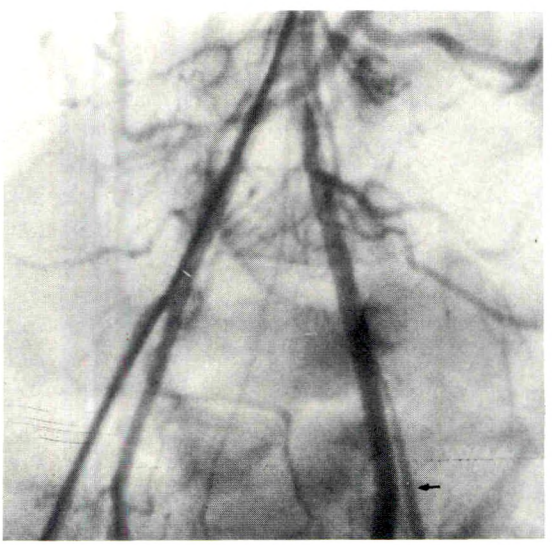


Fig. 6. Case III. Stenosis and elongated funnel-shaped post-stenotic dilatation of the left external iliac artery (arrow).

in whom the angiographic examination is performed in an advanced stage of the disease.

The angiographic feature of the aortic branches involved in the present 3 cases, revealed a certain similarity by showing an elongated, smoothly bordered stenotic segment gradually increasing its size into a funnel-shaped channel. A similar angiographic pattern can be identified on the angiograms in 14 out of 18 patients reported below the age of 12 years.[1,4,5,10,12,13,17,18,22] This distinctive angiographic pattern is different from the various types of fibromuscular dysplasia widely documented in the literature.[16,18] An eventful explanation of this specific angiographic pattern can be seen in the pathologic findings observed in vascular neurofibromatosis. Thickening, disorganization and/or atrophy of the media and nodular fibrosis of the adventitia were described in small arteries[19] and the renal arteries in 28 of the reported cases. The affected renal arteries were involved diffusely, in some of them even the intrarenal branches.[13,22,23] The poststenotic funnel-shaped appearance present in the majority of patients in the younger age group can be related to the diffuse involvement of the arterial wall which prevents the formation of the usual poststenotic dilatation. However, if localized fragmentation and atrophy of the media are present, saccular aneurysms with a narrow pedicle may occur as reported before[13] and also observed in 1 of our patients (Case 11). Further pathologic observations with angraphic correlation are necessary in order to prove this concept.

Involvement of the pulmonary artery, the thoracic outlet arteries and coarctation of the thoracic aorta has not been reported before in neurofibromatosis. These widespread arterial lesions support the view of Reubi,[19] that vascular involvement is a part of neurofibromatosis. It can be assumed that by increasing the use of angiography more cases with such widespread involvement will be detected.

SUMMARY

Three cases of vascular neurofibromatosis were studied angiographically.

Renal artery involvement was present in all. In 1 patient multiple vascular lesions including thoracic outlet arteries, thoracic aorta, pulmonary artery, celiac, superior mesenteric and iliac arteries were also present. Angiography revealed in the involved arteries a smoothly bordered stenotic segment continuing into an elongated funnel-shaped channel.

The presence of similar features in another 12 cases reported in the literature implies that the described pattern may be considered as suggestive of vascular neurofibromatosis.

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REFERENCES

1. Allan, T. N. K., and Davies, E. R. Neurofibromatosis of renal artery. *Brit. J. Radiol.*, 1970, *43*, 906–908.
2. Bloor, K., and Williams, R. T. Neurofibromatosis and coarctation of abdominal aorta with renal artery involvement. *Brit. J. Surg.*, 1963, *50*, 811–813.
3. Bourke, E., and Gatenby, P. B. B. Renal artery dysplasia with hypertension in neurofibromatosis. *Brit. M. J.*, 1971, *3*, 681–682.
4. Cornell, S. H., and Kirkendall, W. M. Neurofibromatosis of renal artery: unusual cause of hypertension. *Radiology*, 1967, *88*, 24–28.
5. Debré, R., Royer, P., and Mozziconacci, P. L'hypertension artérielle grave par une anomalie unilatéral de l'artère chez l'enfant. *Bull. Acad. nat. méd.*, 1957, *141*, 421–429.
6. Debré, R., Royer, P., Fauré, C., Pellerin, D., and Hebib, R. L'aneurysme congénital de l'artère rénale avec hypertension artérielle chez l'enfant. *Arch. franç. pédiat.*, 1957, *24*, 1–19.
7. Diekmann, L., Huther, W., and Pfeiffer, R. A. Ungewöehnliche Erscheinungsformen der Neurofibromatose im Kindesalter. *Zt. Kinderheilk.*, 1967, *101*, 191–222.
8. Dux A., and Thurn, P. Das aneurysma der Arteria renalis. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1962, *96*, 471–478.

9. Farkas, A., Heyman, I., Griffel, B., and Firstater, M. Hypertension caused by vascular lesions in neurofibromatosis. *J. Israel M. A.*, 1973, *84*, 390–393.
10. Fleming, M. P., and Miller, W. E. Renovascular hypertension due to neurofibromatosis: report of case. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 452–454.
11. Glenn, F., Keefer, E. B. C., Speer, D. S., and Dotter, C. T. Coarctation of lower thoracic and abdominal aorta immediately proximal to celiac axis. *Surg., Gynec. & Obst.*, 1952, *94*, 561–569.
12. Grad, E., and Rance, C. P. Bilateral renal artery stenosis in association with neurofibromatosis: report of two cases. *J. Pediat.*, 1972, *80*, 804–808.
13. Halpern, M., and Currarino, G. Vascular lesions causing hypertension in neurofibromatosis. *New England J. Med.*, 1965, *273*, 248–252.
14. Hanson, J., Ikkos, D., Johansson, L., Rudhe, U., and Senning, A. Coarctation of abdominal aorta: case report and description of successful surgical treatment. *Acta chir. scandinav.*, 1959, Suppl. 245, 315–322.
15. Heberer, G., Engelking, R., and Eigler, F. W. Diagnostische und Therapeutische Besonderheiten bei einigen Hochdruckkranken mit Nierenarterienstenosen. *Deutsche med. Wchnschr.*, 1967, *92*, 581–587.
16. Kincaid, O. W., Davies, G. D., Hallerman, F. J., and Hunt, J. C. Fibromuscular dysplasia of renal arteries. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 271–282.
17. Leuman, E. P., Bauer, R. P., Slaton, P. E., Biglieri, E. G., and Holliday, M. A. Renovascular hypertension in children. *Pediatrics*, 1970, *46*, 362–370.
18. Mena, E., Bookstein, J. J., Holt, J. F., and Fry, W. J. Neurofibromatosis and renovascular hypertension in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *118*, 39–45.
19. Reubi, F. Neurofibromatoses et lésions vasculaires. *Schweiz. Med. Wchnschr.*, 1945, *75*, 463–465.
20. Senning, A., and Johansson, L. Coarctation of abdominal aorta. *J. Thoracic & Cardiovasc. Surg.*, 1960, *40*, 517–523.
21. Siguier, F., Dupperat, B., Betourne, C. L., and Godeau, P. L'association neurofibromatose-hypertension artérielle. *Semaine hôp.* Paris, 1961, *37*, 1491–1495.
22. Smith, C. J., Hatch, F. E., Johnson, J. G., and Kelly, B. J. Renal artery dysplasia as cause of hypertension in neurofibromatosis. *A.M.A. Arch. Int. Med.*, 1970, *125*, 1022–1026.
23. Wallis, K., Deutsch, V., and Azizi, E. Hypertension in case of von Recklinghausen's neurofibromatosis. *Helvet. paediat. acta.*, 1970, *25*, 147–153.

INTEGRATED ISOCENTRIC DIAGNOSTIC SYSTEM FOR NEURORADIOLOGY*

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THE constant improvements in diagnostic radiologic techniques provides the impetus for development of new equipment to implement these techniques. The performance of many neuroradiologic procedures has been significantly enhanced by the recent development of an integrated isocentric system (Omnitome*). This system has been designed primarily for encephalography, but also provides significant advantages during the performance of angiography and air myelography. The prototype was installed at the Medical College of Virginia in November of 1972 for clinical trials.

The present communication will delineate the author's experience with this unit along with an outline of the advantages and techniques employed.

THE OMNITOME SYSTEM

The principal feature of the Omnitome (Fig. 1, *A–C*) is the fully isocentric system maintaining the head in alignment for filming or fluoroscopy regardless of the patient's position. The chair is designed to provide 480° of continuous rotation with decubitus use available if indicated. Room height is minimized by automatic movement of the unit up or down on a fixed wall pedestal according to the position of the unit. There is lateral, longitudinal and vertical adjustment of the chair relative to the isocenter as well as rotation about the long axis of the patient. This allows for precise adjustment of the patient to the isocenter at the beginning of the procedure and maintains versatility of adjustment for any patient motion during the procedure. Patient fixation is achieved by thigh pads, crossed shoulder straps, and calf straps. Head adjustment and fixation is obtained by a chin rest with straps across the vertex of the head. There is a C-arm (Fig. 1) which provides for continuous 300° rotation about the isocenter in a plane vertical to the wall pedestal with tomographic capabilities at any position. Grossman linear tomography may be obtained with a choice of 8°, 15°, 30° and 45° arcs. A unique feature of the Omnitome system is the ability to perform tomography at a varying angle of incidence. The L-arm (Fig. 1) will also rotate 270° about an axis parallel to the wall pedestal with consequent alteration in the site of rotation of the C-arm. Thus the C-arm always rotates on an axis perpendicular to the wall axis. This provides the ability for tomography with horizontal, vertical, or intermediate sweeps and allows blurring of the object at 90° to its densest part, thus reducing undesired shadows. These movements of the C-arm and L-arm remain independent of the movements of the chair arm. The chair arm rotates on the same plane as the L-arm but outside of the L-arm arc. This preserves chair adjustment and allows the chair to be parked in a remote position when not in use. An image intensifier and television system is incorporated into the C-arm with a choice of field size of 4.5, 6 or 9 inches. The tube has a 0.3 and 1.0 mm. focal spot. A variable source-image distance of 97 to 127 cm. is available by motorized movement of the image intensifier.

The Omnitome system is also equipped with a phototimed 105 mm. photospot camera. This provides for either single

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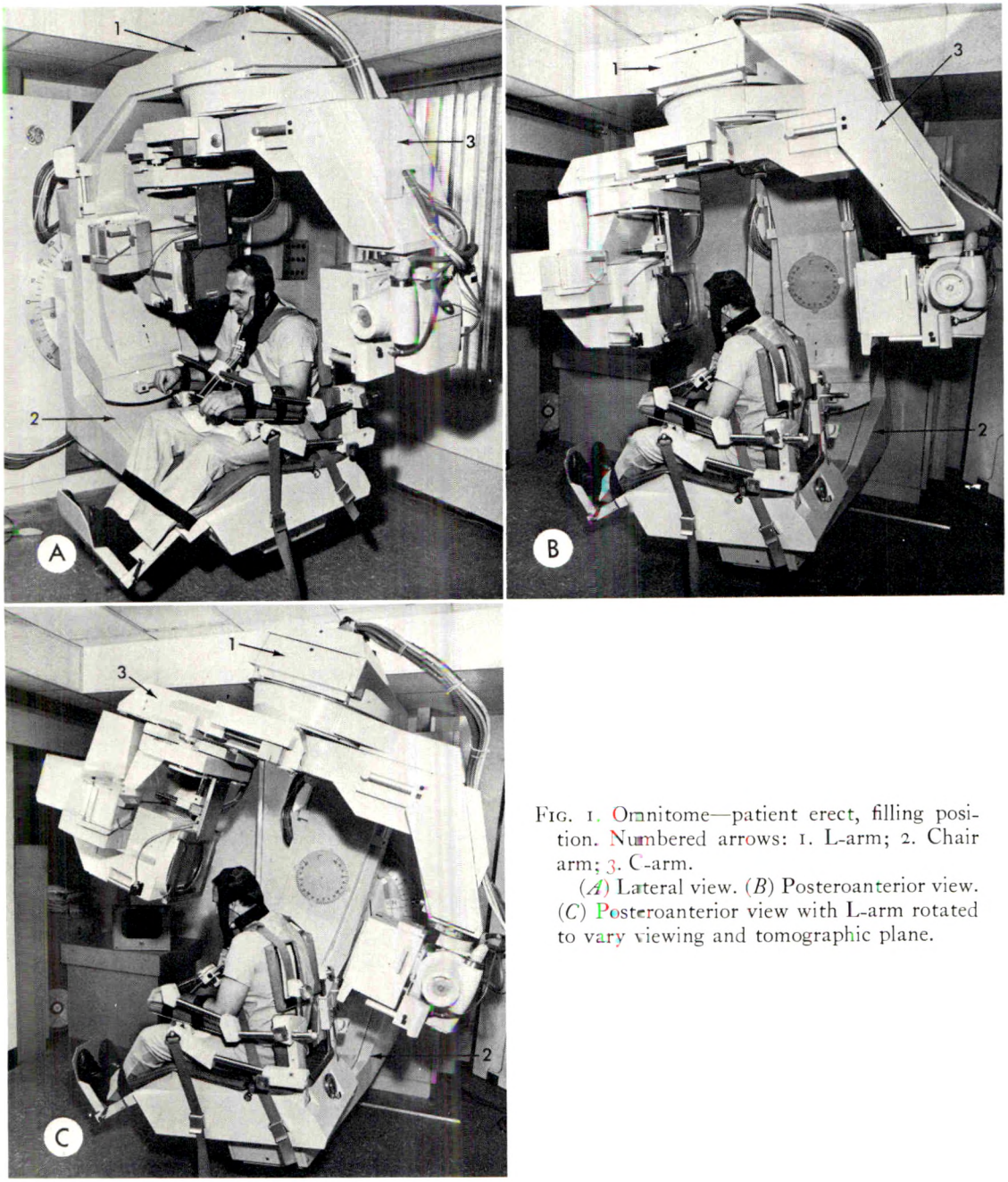


Fig. 1. Omnitome—patient erect, filling position. Numbered arrows: 1. L-arm; 2. Chair arm; 3. C-arm.
(*A*) Lateral view. (*B*) Posteroanterior view. (*C*) Posteroanterior view with L-arm rotated to vary viewing and tomographic plane.

frame or multiple frames up to 12 per second with fluoroscopic control. Tomography is also available with the use of the photospot camera. The field size available is narrowed minimally due to the curvature of the input phosphor of the image intensifier system. This, however, still provides for up to 6–7 inches of clear field size.

The remote control panel provides for the radiographic techniques and television monitor as well as for all the motions of the chair and Omnitome system. Photospots may be obtained from the remote control panel with a television screen monitor. There is also a hand controlled portable system with the same patient positioning

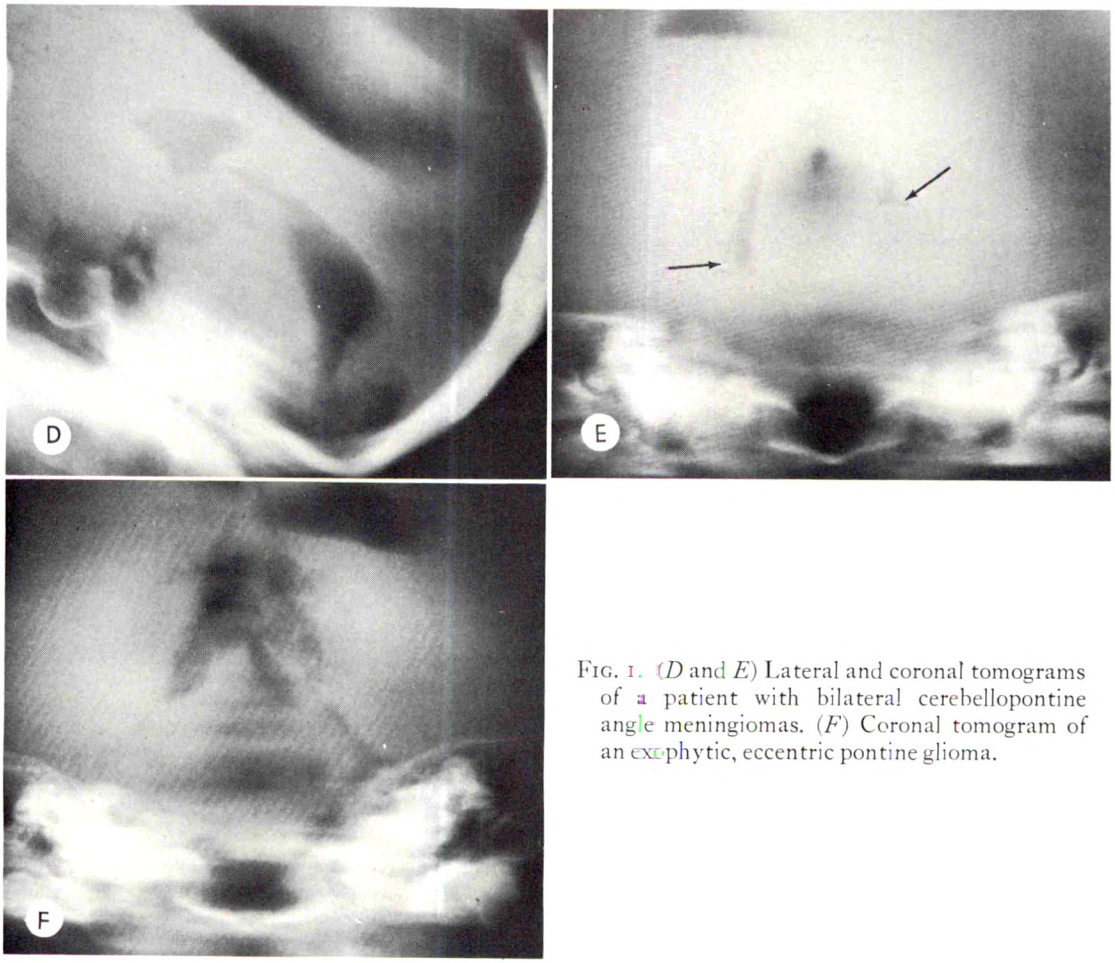


FIG. 1. (*D* and *E*) Lateral and coronal tomograms of a patient with bilateral cerebellopontine angle meningiomas. (*F*) Coronal tomogram of an exophytic, eccentric pontine glioma.

controls as on the control panel and a television monitor adjacent to the patient. This enables one to exactly position for filming or during the installation of air with closer monitoring of the patient.

An accessory table is used for angiography and myelography and will be described later. The encephalographic chair is motor driven to a parked position during these procedures.

PNEUMOENCEPHALOGRAPHY

The patient is initially positioned in the chair (Fig. 1*A*) with the head slightly flexed as for a standard pneumoencephalography. The isocenter is established with vertical and horizontal light beams and fluoroscopy. Air is injected under fluoroscopic control to obtain ventricular and cisternal filling. The basal cisterns may be filled by rotation of the chair obviating the need to remove the head from the head holder. Following the instillation of air, zonograms of an 8 or 15 degree angle can be obtained with standard films or on the photospot camera (Fig. 3). After adequate air instillation, one can obtain standard frontal and lateral tomograms with a 30 or 45 degree arc. The coronal tomograms may be obtained in the angle of incidence desired by rotation of the L-arm (Fig. 1, *B* and *C*). There is a range of about 40° available for the standard tomogram which usually enables the tomograms to be obtained in a coronal plane approximating the vertical alignment of the brain stem. Further air is then instilled under fluoroscopic control as deemed necessary to complete the pro-



FIG. 2. Omnitome—patient supine and decubitus.
(*A*) Lateral view. (*B*) Lateral view with L-arm rotated to alter plane of tomographic sweep. (*C*) Postero-anterior view. (*D*) Decubitus position.

cedure and the patient can be brought to the supine position. The L-arm may again be rotated, so that linear tomography may be obtained at different angles of incident relative to the anterior third ventricle. This may be done in the lateral of the frontal projection to enable one to tomograph exactly in the plane desired with vertical or horizontal blurring of the suprasellar cisterns (Fig. 2). Balancing of the lateral ventricles may then be performed under fluoroscopic control.

After completion of filming of the anterior horns, the patient can be rotated to the erect and prone position for further filming as desired with the photospot camera or with standard filming. The conventional pneumoencephalography usually includes standard films in the erect and supine positions with the remainder of the film series obtained on the 105 mm. photospot camera (Fig. 3). This enables one to obtain multiple different projections and angulations throughout the desired series as well as intermediate positions. The time required is markedly reduced and positioning can be performed under fluoroscopic control. Following the prone study of the occipital horns, the patient can be continued through a complete forward somersault to the supine position, enabling complete filling of the temporal horns. Decubitus positioning of the patient is also available for study of the temporal horns individually if necessary (Fig. 2*D*). One can also obtain tomography of the temporal horns perpendicular to their long axis by rotation of the L-arm, obviating movement of the patient's head.

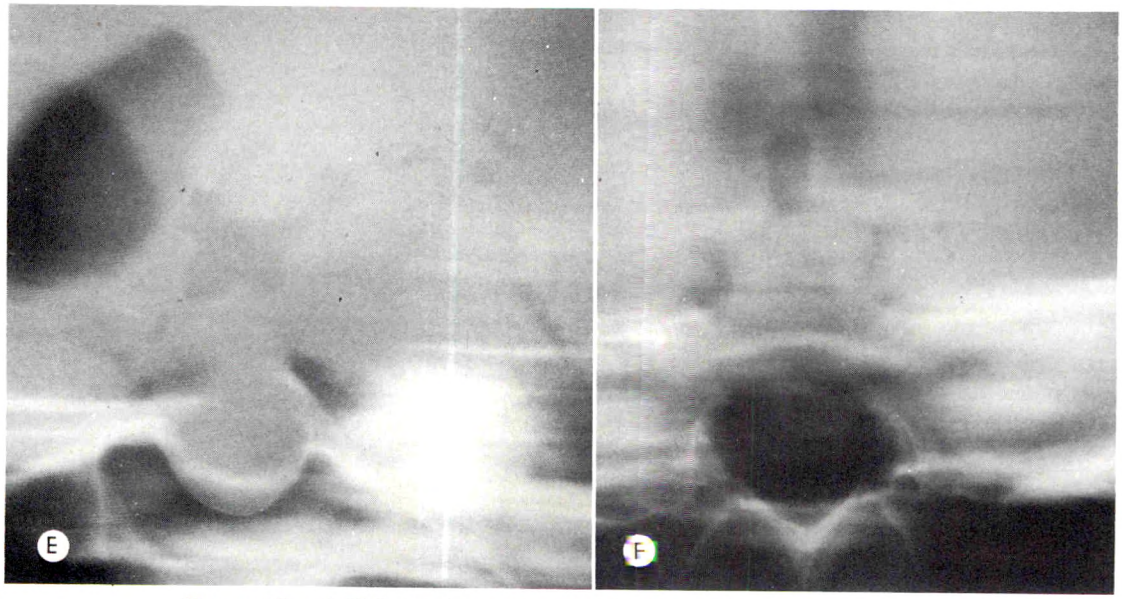


FIG. 2. (*E* and *F*) Lateral and coronal tomograms of a suprasellar mass.

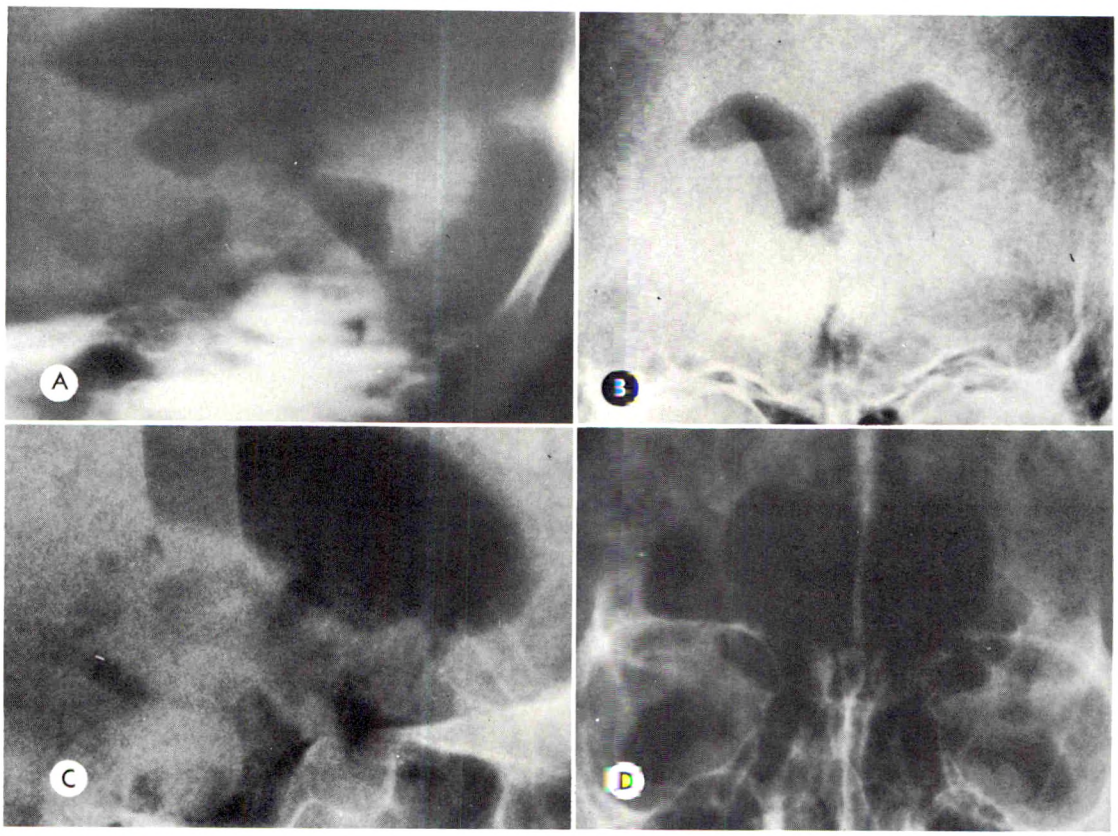


FIG. 3. Encephalography—105 mm. films.

(*A*) Lateral zonogram of 4th ventricle. (*B*) Frontal view with patient erect. (*C*) Lateral view with patient supine. (*D*) Frontal view of temporal horns.

AIR MYELOGRAPHY

For air myelography, an accessory table is utilized. The table (Fig. 4) provides for 20° of Trendelenburg positioning along with 15° of elevation of the head. The patient is supine in the Trendelenburg position with shoulder straps attached to the foot of the table. A lateral cervical puncture at the C-1, C-2 interspace is performed under fluoroscopic control and increments of oxygen exchanged for cerebral spinal fluid (Fig. 4*B*). Following complete filling of the subarachnoid space, as visualized under fluoroscopy, one can obtain lateral and frontal tomograms (Fig. 4, *B–D*). Lateral layer selection is controlled by a micrometric vernier with a travel of 20 cm. The angle sweep of the tomographic arm is usually one parallel to the cervical spine. This has been found advantageous in maintaining the ghosting of the vertebral bodies ventral to the air and spinal cord contour. However, one is also able to rotate the system so that a vertical sweep of the tomographic arm is available relative to the spinal axis if desired.

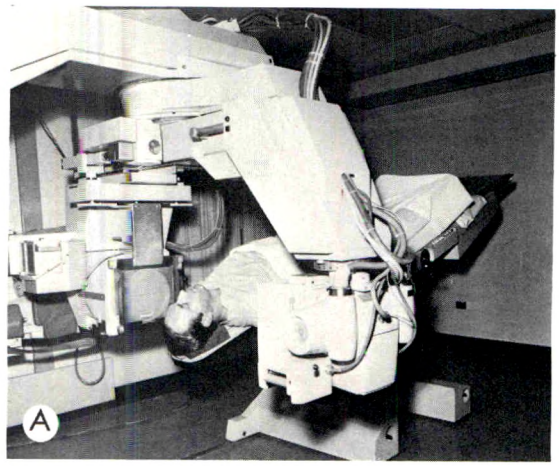


Fig. 4. Air myelography.
(*A*) Patient in 20° Trendelenburg position.

The table can then be raised to the horizontal position or the head-up position as desired to alter the cord size, as in cases of syringomyelia. Patient positioning is, therefore, simplified and their discomfort is reduced in the more comfortable supine position. The lateral puncture is easily per-

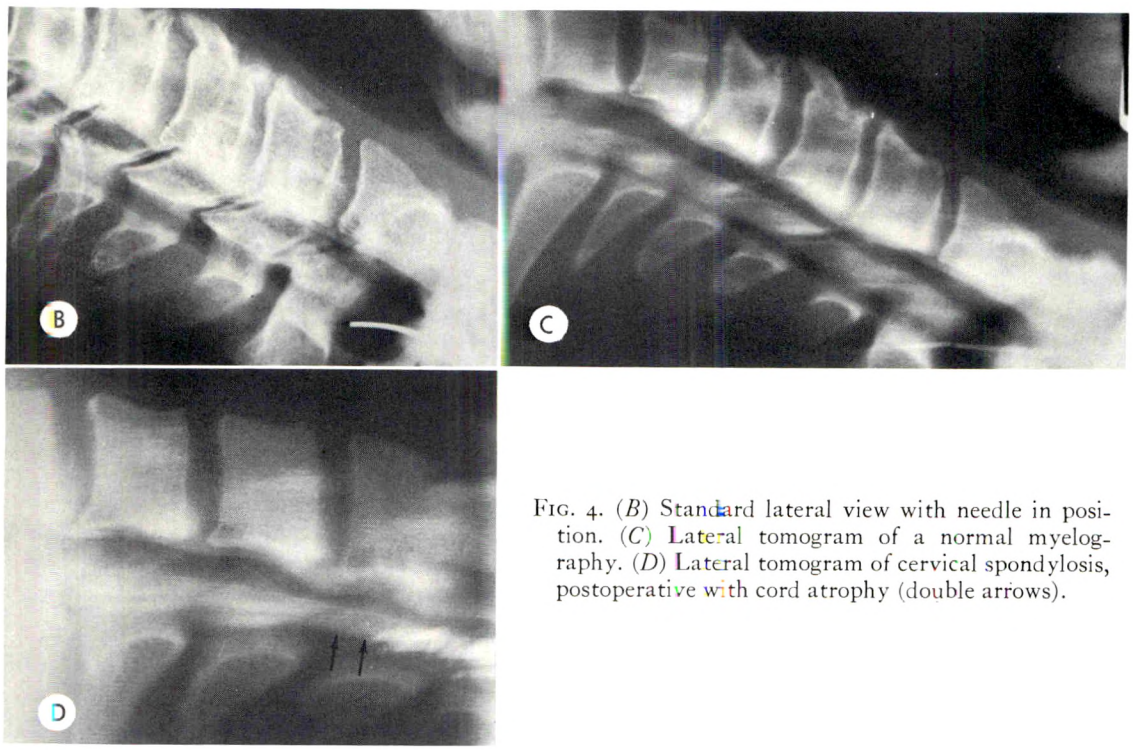


Fig. 4. (*B*) Standard lateral view with needle in position. (*C*) Lateral tomogram of a normal myelography. (*D*) Lateral tomogram of cervical spondylosis, postoperative with cord atrophy (double arrows).

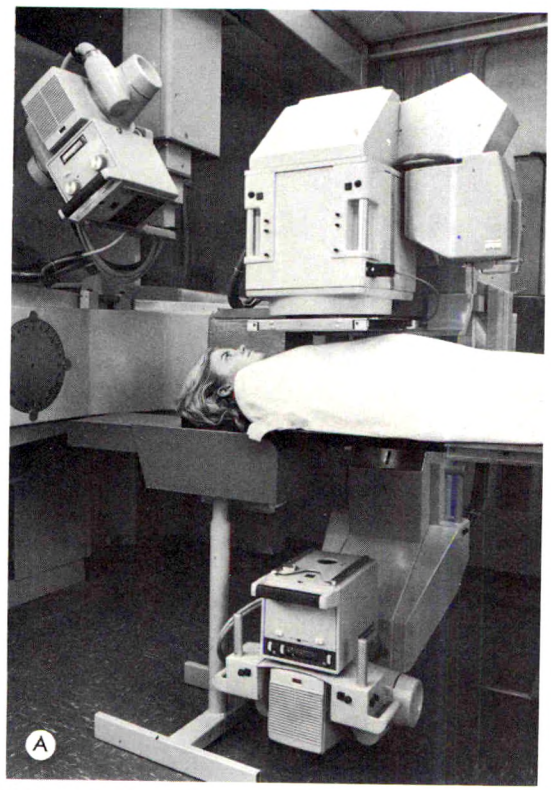


FIG. 5. Angiography.
(*A*) Patient on accessory table with film changers mounted adjacent to wall pedestal.

formed utilizing C-arm fluoroscopy.

Obviously, a second choice available with the system is the utilization of the pneumoencephalographic chair for air myelography. This is limited to the upper cervical region due to the patient support devices. This technique will, however, enable one to proceed with standard encephalography following evaluation of the cervical spine.

ANGIOGRAPHY

The accessory table is also utilized for angiographic procedures with the C-arm providing fluoroscopic versatility (Fig. 5*A*). The table provides for a motorized vertical height adjustment of 50 cm. for magnification studies. Linear movement of 125 cm. from the isocenter is available with both manual and a motor driven table top to provide fluoroscopy in the femoral region for catheter insertion. A foot switch is also available to convert the table top into a free floating top when necessary. Once the needle or catheter has been inserted into the desired vessel, filming may be obtained on the 105 mm. camera or with standard film changers (Fig. 5, *B–D*). The isocentric system facilitates filming with the 105 mm. camera, greatly reducing the time necessary for obtaining multiple views. Fluoroscopy may be used for positioning and centering, and the C-arm may be rotated for the desired projection. The frontal projection is usually obtained in a postero-anterior direction, allowing for the most mobility for angulation of the image intensifier and thus reducing the object film distance. Oblique views of the carotid bifurcation or intracranial vessels are easily obtained by the multiple possible compound angulations of the C-arm and L-arm.

One may also use standard serial film changers which are mounted above or caudal to the C-arm position. Changers mounted caudal to the C-arm would be most useful for abdominal filming. When the changers are mounted above the C-arm (Fig. 5*A*), a second tube may be positioned in the desired angle and minimize the horizontal movement of the patient necessary to obtain a film series. The single overhead hanging tube may then be used for the frontal and lateral projections, or if desired a second tube may be mounted on a floor pedestal for biplane filming. The limitation of space above the patient's head makes a smaller film changer more desirable in this location. The 24 cm. by 30 cm. changers provice the maximal versatility of patient movement and ease of operation in this position, although 36 cm. by 36 cm. film changers are possible if these are considered desirable.

An additional feature is the feasibility of angiotomography with this system (Fig. 5*D*). The C-arm maintains its potential of tomography wherever positioned and therefore frontal or lateral tomograms are instantly available when desired. The use of a multilayered cassette would seem to be the present choice for this examination.

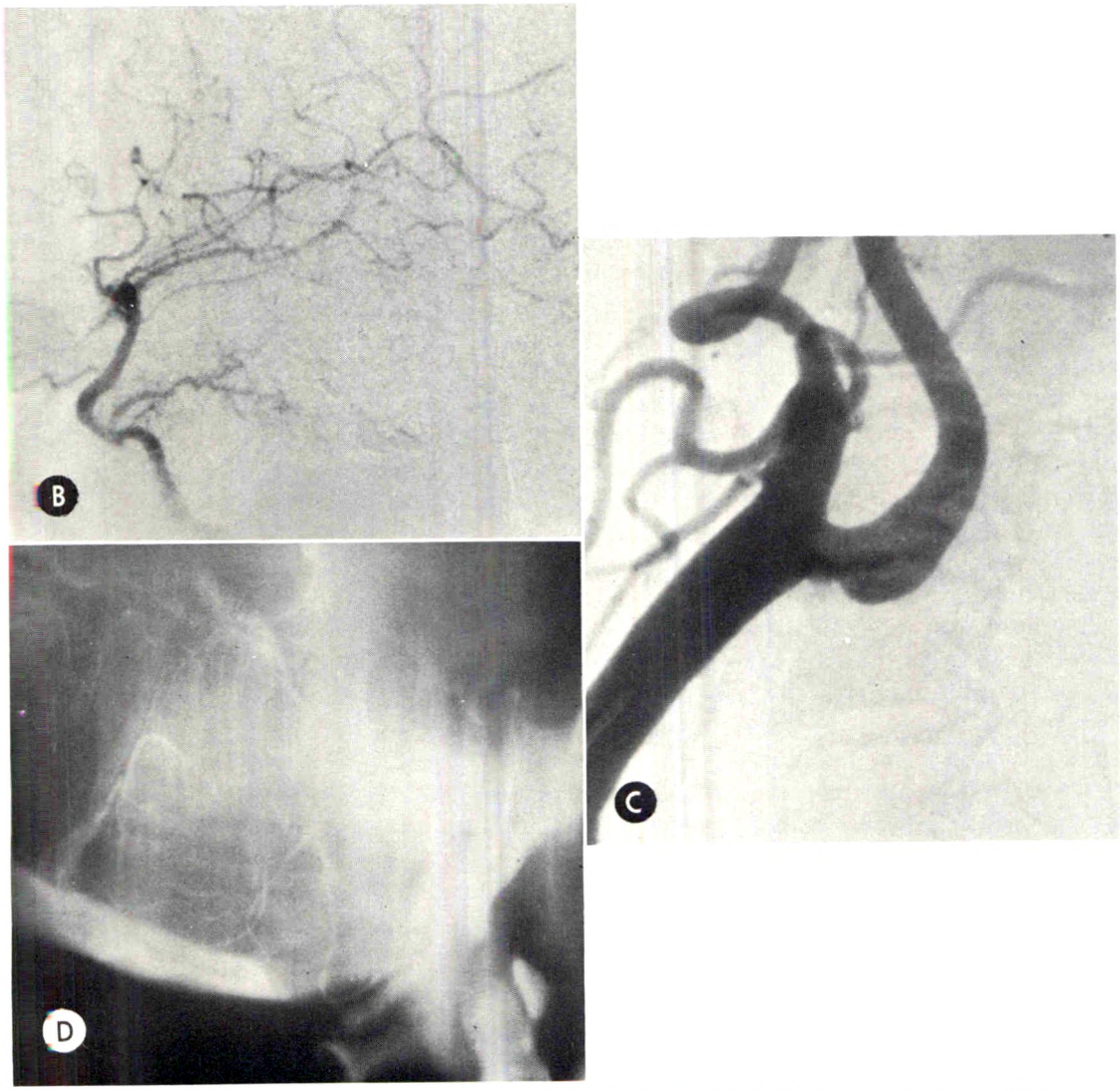


Fig. 5. (*B*) Petrous apex meningioma with abnormal meningeal vessels from the cavernous carotid (105 mm. film—subtraction). (*C*) Oblique carotid bifurcation with ulcerated plaque (105 mm. film—subtraction). (*D*) Lateral angiotomogram of a vertebral artery injection (30° arc) in a plane through the choroidal point just lateral to midline.

However, the most desirable technique has not been determined at present and will be the subject of a further report.

DISCUSSION

The outstanding feature of the Omnitome system for neuroradiologic diagnostic procedures is the ease and rapidity with which precise studies can be performed. The skull is easily maintained on the isocenter and filming may be obtained regardless of the patient positioning. The dual arm system, independent of the encephalographic chair, allows for multiple complex angulations for filming. Likewise, tomography is always available regardless of the position of the patient and may be obtained in a desired angle of incidence relative to anatomic structure. This is particularly useful for areas such as the posterior fossa, corpus callosum or temporal horns where the tomograms may be ob-

tained perpendicular to the locus of presumed pathology without altering the head and consequent position of the contrast agent. The anterior third ventricle may also be tomographed in different angles relative to the suprasellar cisterns.

Angiography procedures are also available with this system. The motorized movements of the chair arm allow it to be easily removed from the working area. Once this is accomplished, the unit becomes similar to any angiographic facility except for the preserved mobility of the fluoroscopic system. The table top movement may be converted into a free floating table by a foot switch and linear movement is adequate for femoral catheterization studies. Biplane instillation may be obtained if considered desirable.

A major additional benefit for both encephalography and angiography is the use of the 105 mm. camera. There is no question as to the significant benefit using the camera during encephalography. Standard views as well as multiple additional views can be obtained with significant reduction in the time required and preservation of diagnostic film quality. This is generally used for the film series during orbiting of the patient (supine, erect, prone and temporal horn views). The advantages and techniques of angiography with the 70 mm. and 105 mm. camera have been previously described during cerebral and coronary arteriography.[1,3] The film quality is excellent, although certainly one would not consider that it is equal to the quality of routine magnified series with standard film changers. Standard subtraction techniques are also easily applied to the 105 mm. film (Fig. 5, *B* and *C*). The question is whether the ease and facility with which one can obtain multiple additional projections and the ease of centering would balance some loss of film quality. At present, one would not consider the 105 mm. camera to be the optimal choice for clinical cases requiring a high degree of resolution of minute vessels. There are cases, however, which would be particularly well studied with this system. Patients with a spontaneous subarachnoid hemorrhage or those suspected of extracranial vascular disease can be very effectively studied with the 105 mm. system. Multiple additional views of the extracranial or intracranial vessels in complex projections may be obtained in a markedly reduced time. Additionally, one could, if necessary, obtain a standard film series or angiotomography in a predetermined position. The advantages for selective spinal cord arteriography are readily apparent. One may obtain multiple views with fluoroscopic control of the filming and catheter position.

Air myelography has been used primarily for the cervical region. The technique emphasized by Heinz and Goldman[4] has been employed on most occasions. This is most useful for cases of cervical spine trauma where maintenance of skeletal traction is necessary, but is also of primary benefit to evaluate cord compression and displacement due to cervical spondylosis.

A supplementary benefit of this system is standard views of the skull or cervical spine. The chair or table may be used as clinically indicated. Once the isocenter is established, the 2 arms permit free orbiting of the Omnitome about the skull or spine, allowing any desired projection. Fluoroscopy permits the visualization of the anatomic parts with simplified centering and lack of distortion during filming. Technique charts have been designed by others[2] to facilitate the precise degrees of rotation and angulation. One may also use the unit for any procedure requiring biplane fluoroscopy such as ventriculography, orbitography or cerebral biopsies.

CONCLUSION

The advantages of a new integrated isocentric system for diagnostic neuroradiology are presented.

The patient is easily positioned and remains on the isocenter throughout the

study without extra movements of the chair or filming system.

Angiography and air myelography can also be performed with this apparatus by using an accessory table.

The benefits of an integrated 105 mm. camera are emphasized.

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REFERENCES

1. Brinker, R. A., and Skucas, J. Small focus roentgen magnified 70 mm. films for study of extracranial arteries. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *115*, 137–142.
2. Cristi, G. Precision techniques for radiographic examination of skull. *Neuroradiology*, 1971, *2*, 55–61.
3. Grollman, J. J., Jr., Gray, R. K., Spiegler, P., Moler, C., MacAlpin, R., and Eber, L. 105 mm. serial photofluorography of coronary arteries. *Radiology*, 1973, *108*, 577–583.
4. Heinz, E. R. and Goldman, R. L. Role of gas myelography in neuroradiologic diagnoses. *Radiology*, 1972, *102*, 629–634.

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Fifty-seventh Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

EDITORIALS

OF *CAT* AND OTHER BEASTS*

CAT (Computerized Axial—or Assisted—Tomography)* is obviously here to stay. Agreement on this point is unanimous. A debate, however, seems to be developing on a corollary matter: how significant will the impact of this new system on established diagnostic methods be? This question is the fashionable guessing game among neuroradiologists as well as nuclear medicine experts. In general, the soothsayers are not optimistic about the well-being of the older techniques. According to these latter-day Cassandras, pneumoencephalography is in its last throes, and the fate of radionuclide brain scanning is not much brighter. In fact, some nuclear medicine specialists presage a last Armageddon between CAT and radioisotope cerebral scintigraphy, with the latter probably losing the battle and coming to an early death (the technique is less than 30 years old). This adversary approach is also hinted in some Requests for Proposals (RFP) for research grants and contracts. Cerebral angiography fares a little better, but only in reference to certain specific conditions, as berry aneurysms.

As an individual, who in former years[1–5] has devoted particular attention to the question of reliability and relative value of neuroradiological methods, I would like to voice a word of caution. Let us temporize and hold judgment. In the meantime, I suggest we carry out in our patients, whenever possible and practical, the well established diagnostic procedures, as well as CAT. Thus, we will avoid hasty, and perhaps regrettable decisions, and in a few years, with the facts in our grasp, we will have a clearer understanding.

Caution is particularly important because we do not really know what CAT will do. Two devices in operation, the EMI-Scanner[6,7] and the ACTA-Scanner,[8] although similar on the surface, are fundamentally different and we may well expect that other machines now being tested, built or designed, will perform in diverse fashion. Radionuclide sources are being considered and tested for transmission CAT, and a few investigators are experimenting with computerized image reconstruction from emission scanning (injected radionuclides).

The introduction in diagnostic radiology of the algorithms used for two-dimensional image reconstruction from sequences of one-dimensional X-ray profiles changes the entire basic outlook as far as equipment is concerned. The conventional commercial X-ray machines, although numerous, are essentially comparable products with dis-

* Other nomenclature: Computerized Transverse Axial—or Transaxial—Scanning; Computerized X-ray Scanning; Computed Scanning; Computed Tomometry.

[1] DI CHIRO, G. RISA encephalography and conventional neuroradiologic methods. *Acta radiol.*, 1961, Suppl. 201, 105–111.

[2] DI CHIRO, G. How reliable is neuroradiology? *Neurology*, 1962, *12*, 93–98.

[3] DI CHIRO, G. Comparison entre la scintigraphie et les méthodes conventionnelles de neuroradiologie pour la localisation des lésions intra-crâniennes. *Ann. radiol.*, 1962, *5*, 195–200.

[4] DI CHIRO, G. Reliability of neuroradiology. *Clin. Neurosurg.*, 1963, *10*, 151–168.

[5] DI CHIRO, G. Relative value of air studies, angiography and radioisotope scanning in diagnosis of glial intracranial tumors. *Progr. Neurol. Surg.*, 1968, *2*, 292–317.

[6] HOUNSFIELD, G. N. Computerized transverse axial scanning (tomography): Part 1. Description of system. *Brit. J. Radiol.*, 1973, *46*, 1016–1022.

[7] AMBROSE, J. Computerized transverse axial scanning (tomography): Part 2. Clinical application. *Brit. J. Radiol.*, 1973, *46*, 1023–1047.

[8] LEDLEY, R. S., DI CHIRO, G., and LUESSENHOP, A. J. Computerized transaxial X-ray tomography of entire human body. To be published.

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similarities limited, in the last analysis, to technical refinements. Even in the field of radionuclide brain scanning, equipment variations are not prominent: in reality a rectilinear scanner and a gamma camera—devices considered by many to be at opposite poles—are but variations on a single theme. The use of one large crystal and the clever employment of an array of photomultipliers in the gamma camera represent an elegant solution, but certainly not a breakthrough. In fact, we are still debating whether the resolution—the final test—of the gamma camera is equivalent to that of the rectilinear scanner. On the other hand, in computerized image reconstruction, a new algorithm may make a substantial, indeed basic, difference in what we will be able to see and distinguish. One device may be able to handle highly differential contiguous absorption-coefficient steps (ACTA), while another is possibly better suited to discriminate narrower variations in the absorption coefficients (EMI). These vastly diverse capabilities make it obvious that speaking of CAT as an homogeneous methodology is, at least, premature. It is most likely that the EMI and the ACTA represent only the dawning. The manipulation of a computer program offers an endless number of possibilities. In this context, it is interesting to note the increasing support for development and/or improvement of analog, digital or hybrid algorithms, and for a system designed to test the various algorithms to be used for CAT. In a sort of "Future Shock" script, we may envisage machines with a number of exchangeable programs, each program being suited to a particular diagnostic problem.

While I refuse to join the prognosticators of the impact that the new CAT methodology will have on older techniques, I would like to share some thoughts which have been crossing my mind. One of these is related to the long time lapse between availability of a technology and its implementation with development of a workable machine. Successful CAT images were first obtained in late 1971. Oldendorf,[9] however, had already, in 1961, patented the concept of passing an X-ray beam at successive angles through an object to obtain a measure of the absorption coefficient at the rotation center. Cormack,[10] in a remarkable paper of 1963, proposed making measurements of the X-ray transmission "along lines parallel to a large number of different directions" so as to obtain a sequence of X-ray transmission profiles. He actually carried out an experiment using a collimated gamma ray source, and measured the intensity of the transmitted beam by means of a Geiger-Müller counter at 5 mm. intervals over a 12.5 cm. scan-pass length. The absorption coefficients of an aluminum cylinder surrounded by a wooden annular were calculated. The experimental results agreed with the actual absorption coefficients of the materials to a high degree of accuracy. Cormack[10] noted that his results were applicable to the field of radiology for "the determination of a variable X-ray absorption coefficient in two dimensions." Thus, the essence of computerized axial tomography was already contained in Cormack's work, which represents a specific application of a more general methodology of image reconstruction. This methodology has been used in fields as diverse as astronomy and electron microscopy. I suspect, if radiologists had known and understood the implications of Cormack's contribution, CAT, its logical extension, would have been developed much sooner. As in many instances, we are again compelled to recognize the frightful gap between technical possibilities and actual accomplishments. It is obvious that inter-disciplinary cross-fertilization, to which lip service is paid at every scientific meeting, has a long way to go.

A second thought concerns the perennial

[9] OLDENDORF, W. H. Isolated flying spot detection of radiodensity discontinuities. *IRE Transac. on Bio-Med. Electronics*, 1961, BME-8, *1*, 68–72.

[10] CORMACK, A. M. Representation of function by its line integrals, with some radiological applications. *J. Appl. Physiol.*, 1963, *34*, 2722–2727.

tides of shifting emphasis on different basic aspects of neuroradiologic know-how. I am referring in particular to the reappearing importance of detailed anatomic knowledge of the CSF cavities. In the last decade, due to the increased application of the angiographic methodology, the young radiologists and neuroradiologists have been concentrating their attention on the fine anatomy of the cerebral vessels. In the same period, because of the decreased interest in pneumoencephalography, the detailed morphology of the ventricular system and subarachnoidal spaces has been neglected. It is clear that further CAT developments will be in the direction of improving the pictorial demonstration of the cerebrospinal fluid (CSF) spaces and the contours of the anatomic structures therein. We will be striving for pneumoencephalographic images without "pneumo." Actually, the current CAT pictures are quite comparable to those obtained by the technique of axial transverse encephalography described more than 10 years ago.[11,12]

The final point has perhaps to do with "guild" interests. A neurosurgeon gave us pneumoencephalography, a neurologist—cerebral angiography, and a general surgeon—radionuclide brain scanning. Now, at last, a neuroradiologist, Dr. James Ambrose, has been directly involved in the development of CAT. There are some indications that nonradiologists are inclined to consider the CAT device as the machine which will eliminate all other machines. In the same breath, unconfessed hopes are flourishing in some quarters of a swift obsolescence of that peculiarly hybrid creature, the neuroradiologist. It would indeed be an ironic twist of fate, if a methodology developed with a strong input by a neuroradiologist, should contribute to the demise of the neuroradiologic specialty. Only "innocent" practitioners, however, may delude themselves that the age of easy radiographic diagnosis has arrived. Sound neuroradiographic knowledge—with all that it entails in neuroanatomy, neuropathology, neurophysiology, neuropharmacology and X-ray technology—is still indispensable, and will be the essential ingredient for profitable application, reliable interpretation, and further development of CAT.

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[11] Di Chiro, G. Axial transverse encephalography. PHS Publication 1104, 1963, Bethesda, Maryland.

[12] Di Chiro, G. An Atlas of Detailed Normal Pneumoencephalographic Anatomy. Second edition. Charles C Thomas, Publisher, Springfield, Ill., 1971.

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WILLIS SEAMAN PECK, M.D.
1900-1974

ON July 8th, 1974, at the age of 74, the end of an outstandingly useful career in radiology came for Willis Seaman Peck. Having survived several massive myocardial infarctions over a period of 5 years, death came to him in his sleep. He is survived by his 2 daughters, Mrs. Joanne Dansard and Mrs. Mary Galloway, both of Toledo, Ohio.

Dr. Peck was born in Pulaski, New York and in 1924 was graduated from the medical school of Syracuse University. A year later he was married to Geraldine Benedict who died in 1969. He was appointed instructor in physical medicine, then a part of roentgenology, at the University of Michigan in 1928. He received training in radiology while serving in physical medicine and was promoted to Assistant Professor and then Associate Professor in Radiology. In 1937 he was placed in charge of radiation therapy activities.

Dr. Peck's training included a summer course in radiation physics at the Univer-

sity of Chicago and field trips to the Mayo Clinic, the old Memorial Hospital under James Ewing, Columbia University under Francis Carter Wood, Philadelphia General Hospital under Bernard P. Widmann and Weatherwax and the National Bureau of Standards in Bethesda.

In 1939 the Michigan training was carried over the state line into Toledo, Ohio, where Harry Dachtler and John Murphy had pioneered in roentgenology many years before. As an associate of Dr. Murray Goodrich at Toledo Hospital and the Toledo Clinic, he was instrumental in broadening partnership activities to cover additional hospitals in Toledo and neighboring towns. In those early years radiologic services were extended to communities from Sandusky on the east to Adrian, Michigan on the west with Fremont and Port Clinton in between, a span of 110 miles. In addition to major activities at Toledo Hospital and the Toledo Clinic, the growing partnership served Flower and Riverside Hospitals in Toledo and Dr. Peck participated in the radiation therapy work at the Maumee County Hospital. As professional radiologic services were developed in the satellite hospitals, time spent by partnership radiologists in road travel was reduced and transferred to the growing work load in Toledo. Peck will be long and gratefully remembered by physicians and patients in northern Ohio towns for the excellent radiologic services which he and his associates provided for them. The present group, The Toledo Radiological Associates, 13 strong, continues office and hospital practice in Toledo.

Willis Peck was a man of many interests. Over and beyond his vigorous and enthusiastic practice of radiology with chief interest in radiation therapy, he found time to devote to outdoor life which had had a strong hold on him from boyhood. He was an excellent wing shot both in skeet and in hunting ducks and Upland birds in Michigan, Ohio and South Dakota. An expert fly fisherman, he tied trout flies of professional quality and used them to fish throughout the mid-west and from Alaska to Labrador. His well equipped workshop was used for the production of authentic duck decoys and excellent cabinet making. He turned out a number of pleasing oil paintings and became an able photographer, using that talent to record his various travels which included South Africa, Yucatan, the Canadian wilds and Alaska.

Radiology has lost an able practitioner, administrator and teacher. The many persons who were privileged to know Willis Peck intimately have lost one of their best and truest friends.

Fred Jenner Hodges, M.D.

NEWS ITEMS

AMERICAN RADIUM SOCIETY

Resident/Fellow Essay Award—May 1975

The American Radium Society announces its Fifth Annual Resident Essay Award Contest.

A certificate of Award is presented, plus travel for resident and spouse (economy class) and $50 per diem for the duration of the Annual Meeting at the Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

For the rules of applying the procedure, please write to: Elliot W. Strong, M.D., Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York, New York 10021.

RADIOLOGY OF THE NEWBORN AND INFANT

The Department of Radiology of Emory University School of Medicine will hold a course on *Radiology of the Newborn and Infant*, November 8 and 9, 1974.

For further information, please contact: H. Stephen Weens, M.D., Department of Radiology, Emory University School of Medicine, Atlanta, Georgia 30322.

THIRD ANNUAL WENDELL G. SCOTT MEMORIAL LECTURE

The Mallinckrodt Institute of Radiology is pleased to announce the Third Annual Wendell G. Scott Memorial Lecture.

Mallinckrodt Institute of Radiology and Washington University School of Medicine have named John M. Dennis, M.D., as the Third Annual Wendell G. Scott Lecturer and Visiting Professor.

John M. Dennis, M.D., is Professor of Radiology at the University of Maryland School of Medicine and has been Head of the Department since 1953. He has recently been appointed as Dean of the Medical School. Dr. Dennis is noted for contributions to many areas of radiology and is currently a trustee on the American Board of Radiology and Vice Chairman of the Board of Chancellors, American College of Radiology.

For further information, please contact: Ronald G. Evens, M.D., Professor and Head, The Edward Mallinckrodt Institute of Radiology, 510 South Kingshighway, St. Louis, Missouri 63110.

RADIOLOGY CONFERENCE

The Department of Radiology of the University of Illinois College of Medicine, Abraham Lincoln School of Medicine is presenting the Eighth Annual Conference on Radiology in Otolaryngology and Ophthalmology at the Illinois Eye and Ear Infirmary, University of Illinois at the Medical Center, on Friday and Saturday, November 29 and 30, 1974.

This course, under the direction of Galdino E. Valvassori, M.D., will include lectures and visual representations by a distinguished faculty. Objectives of the program are to make radiologists more aware of the applications of radiology in otolaryngology and ophthalmology; also to present recent advances in otoradiology. One-and-one-half days will be spent on radiology and otolaryngology. Saturday afternoon will be devoted to radiology and ophthalmology.

For further information please write: Jane Whitener, Office of Continuing Education Services, University of Illinois College of Medicine, 1853 West Polk Street, Room 131, Chicago, Illinois 60612.

THE DELAYED CONSEQUENCES OF CANCER THERAPY: PROVEN AND POTENTIAL

A Conference sponsored by the Cancer Clinical Investigations Review Committee of the National Cancer Institute will be held in Orlando, Florida at the Sheraton Olympic Villa, 6700 Sand Lake Road, on January 9–11, 1975.

The topics include: Basic Mechanisms of Injury; Late Effects of Chemotherapy; Late Effects of Radiation Therapy; Late Effects of Combined Chemo- and Radiation Therapy on Organ Function; and Immune Competence, Oncogenesis, etc., as well as Epidemiology.

The participants are: G. Canellos, G. Casarett, W. Creasey, G. D'Angio, A. Evans, Z. Fuks, A. Gilladoga, E. Gehan, T. Hall, C. Harris, J. Harris, F. Hendrickson, G. Hutchison, N. Jaffe, R. Johnson, T. King, M. Lane, C. Lushbaugh, A. Meadows, M. Mendelshon, P. Morris-Jones, M. Nesbit, J. Nickson, R. Parker, I. Penn, T. Philips, G. Ross, U. Saffiotti, C. Shellabarger, H. Tagnon, M. Tefft, L. Wasserman, D. White, J. Wilson, and R. Withers.

For further information please write to: Mrs. Jeannette Steinbraker, Room 10A03 Westwood Building, National Institutes of Health, Bethesda, Maryland 20014.

POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

The Cornell University Medical College is sponsoring a 5 day postgraduate course in Diagnostic Radiology at the King's Inn and Golf Club in Freeport, Grand Bahamas Island, Bahamas on February 10–14, 1975.

Scientific sessions dealing with the GI and GU Tracts, Skeletal System, Chest and Pediatrics will be held from 8:30 A.M.–1:00 P.M. each day. Special emphasis will be placed on the anatomic and pathologic correlation.

The Guest Faculty includes Drs. David H. Baker, Elias G. Theros, John A. Evans, Harold A. Baltaxe, Robert H. Freiberger, Morton A. Meyers, Zuheir Majahed, Joseph P. Whalen, and Patricia Winchester.

For further information and registration forms please apply to: John A. Evans, M.D., Department of Radiology, Cornell University Medical College, 1300 York Avenue, New York, New York 10021.

DIAGNOSTIC RADIOLOGY OF THE URINARY TRACT

This course is designed for radiologists and urologists and will include discussions on diagnosis, radiologic/pathologic correlation, and management of neoplastic, inflammatory, and traumatic conditions of the kidney, ureters, bladder, urethra, prostate, and pelvic-genital organs.

The meeting will be held February 23–27, 1975, and will include informal talks, panel discussions, and round table discussions by an outstanding guest faculty and members of the staff of the Departments of Radiology and Urology of LSU School of Medicine.

For further information, please contact: Erich K. Lang, M.D., Department of Radiology, LSU School of Medicine, P.O. Box 3932, Shreveport, Louisiana 71130.

FIFTH ANNUAL ASPEN RADIOLOGY CONFERENCE

The Fifth Annual Aspen Radiology Conference will be held March 3–7, 1975, at the Aspen Institute for Humanistic Studies, Aspen, Colorado.

The Conference is designed for physicians and scientists interested in diagnostic radiology, nuclear medicine and radiation therapy, and will explore the impact of clinical and technologic advances on radiologic practice.

The topics for discussion will include advances in bone, vascular, gastrointestinal, and neuroradiology involving a tri-radiologic approach. Each morning course will survey the advances in a single radiology subdivision as a refresher course with independent diagnostic, nuclear medicine and therapy sessions. Previewed instructive cases, illustrating these topics, will be presented for open discussion in the afternoons.

The Faculty in Diagnostic Radiology will consist of: Stanley Baum, M.D., Harvard University; Marvin Daves, M.D., University of Colorado; Henry Goldberg, M.D., University of California; Fred Hodges, M.D., Johns Hopkins University; and Don Resnick, M.D., University of California.

The Faculty in Nuclear Medicine will

consist of: Fred Bonte, M.D., University of Texas; Gerald DeNardo, M.D., University of California; Alexander Gottschalk, M.D., University of Chicago; Robert O'Mara, M.D., University of Arizona; and Duncan Burdick, M.D., University of Colorado.

The Faculty in Radiation Therapy will consist of: Luther Brady, M.D., Hahnemann Medical College; Sam Hellman, M.D., Harvard University; Eric Mayer, M.D., University of Arizona; Theodore Phillips, M.D., University of California; and Philip Rubin, M.D., University of Rochester, New York.

Further information may be obtained from Maurice O'Connor, M.D., Conference Director, Division of Radiology, Denver General Hospital, Denver, Colorado 80204.

SEMINAR IN ANGIOGRAPHY: PROSPECTS FOR LATIN AMERICA 1975

The Departments of Radiology, Shadyside Hospital and University of Pittsburgh, will hold this Seminar in conjunction with the 11th Inter-American Congress of Radiology, at Tequendama Hotel, Bogota, Colombia, South America, February 14 and 15, 1975.

This Seminar is A.M.A. approved Category 1 (13 hr. credit).

For further information, please contact: Shadyside Hospital, Department of Radiology, 5230 Centre Avenue, Pittsburgh, Pennsylvania 15232.

POSTGRADUATE COURSE IN THE RADIOLOGY OF BONES AND JOINTS

A Postgraduate Course in the Radiology of Bones and Joints (Radiology PM 3) will be given at the College of Physicians and Surgeons of Columbia University under the direction of Dr. Frieda Feldman, Professor of Clinical Radiology and Director of Skeletal Radiology at the Columbia-Presbyterian Medical Center on March 17–19, 1975.

This course is designed for Radiologists, Orthopedists and Rheumatologists, and will cover basic and current concepts of skeletal radiology and topics relating to the pathology, physiology and treatment of skeletal disease.

The course is approved for 24 hours credit by the Commission on Education of the American Academy of General Practice.

For further information please write to Dr. Frieda Feldman, the Director of the Course, Department of Radiology, The Presbyterian Hospital, 622 West 168th Street, New York, New York 10032.

OPHTHALMOLOGIC RADIOLOGY: RADIOLOGY OF THE EYE, THE ORBIT AND VISION

Columbia University College of Physicians and Surgeons will sponsor a course on the Radiology of the Eye, the Orbit and Vision on Friday and Saturday, March 21 and 22, 1975.

The course will deal with the radiographic diagnosis of conditions affecting the eye and vision, including the orbit, the paranasal sinuses and the sella turcica.

The role of routine radiography and special procedures including tomography, ultrasonography, angiography, venography and dacryocystography will be presented.

This course is accredited by the A.M.A. Category I award for 14 hours.

The Faculty includes Drs. Coleman, Grove, Kieffer, Hui, Potter, Tenner, Tretter, Trokel, Valvassori, and Zimmer.

For further information please write to: Dr. Guy D. Potter, Professor of Radiology, Director of the Course, Department of Radiology, The Presbyterian Hospital, 622 West 168th Street, New York, New York 10032.

POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY OF THE CHEST

The Department of Radiology of the New York Medical College is sponsoring a 3 day Post-graduate Course in Diagnostic Radiology of the Chest on April 7–9, 1975.

The course will be held at the Plaza Hotel in New York City. Scientific sessions will be held from 8:30 A.M. through 5:00 P.M. daily.

For further information, please write to: Richard M. Friedenberg, M.D., Depart-

ment of Radiology, New York Medical College, 1249 Fifth Avenue, New York, New York 10029.

THE INTERNATIONAL SKELETAL SOCIETY

The International Skeletal Society announces that it will be holding a Second Refresher Course on Skeletal Disorders, an interdisciplinary course with emphasis on Radiology, at Royal College of Physicians, London, N.W.1, April 28–30, 1975.

The speakers will be members of The International Skeletal Society and will consist of eminent specialists in skeletal disorders encompassing radiology, pathology, orthopedic surgery, rheumatology, endocrinology, and epidemiology.

Numbers are strictly limited.

The Program Committee consists of Professor J. H. Middlemiss, Chairman; Dr. Philip Jacobs; Dr. Ronald O. Murray; Dr. Harold G. Jacobson; Dr. Jack Edeiken; and Dr. Hubert A. Sissons.

Applications should be returned to Dr. Jack Edeiken, Department of Radiology, Thomas Jefferson University Hospital, 11th and Walnut Streets, Philadelphia, Pennsylvania 19107.

INTERNATIONAL SYMPOSIUM ON CANCER THERAPY BY HYPERTHERMIA AND RADIATION

A 3 day international symposium on Cancer Therapy and Hyperthermia, under the co-sponsorship of the American College of Radiology and the National Cancer Institute, is being planned at the Key Bridge Marriott Hotel, Washington, D. C., U.S.A., for May 11–13, 1975.

This meeting will bring together physicists, radiation therapists, radiobiologists, physiologists, and engineers, and will focus on the status and potential of hyperthermia as an adjunct to cancer therapy.

The meeting will include a series of formal presentations and extended informal discussions. Among the topics to be considered are: Effects of Hyperthermia on Normal and Tumor Tissues; Mechanisms of the Combined Action of Hyperthermia and Radiation; Production and Measurement of Thermal Fields; Potential of the Combined Modality to Clinical Oncology; and Physiological Response to Hyperthermia. Noted researchers from around the world will present the formal papers and lead the discussions.

This symposium will be limited to 100 participants. Applicants will be considered on a first-come, first-served basis.

For further information and registration materials, please write: J. Eugene Robinson, Ph.D., or Morris J. Wizenberg, M.D., c/o The American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

BOOK REVIEW

THE FUNDAMENTALS OF RADIOLOGICAL SCIENCE. By John Hale, Ph.D., Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pa. Cloth. Pp. 343, with some illustrations. Price, $14.75. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1974.

This eminently readable book is a welcome addition to the literature despite the wealth of recent publications dealing with radiological physics. Like most textbooks on this subject, it is intended primarily for residents in radiology. The author's approach to teaching radiological physics is to divide the subject into two areas: (1) basic radiological science; and (2) clinical applications. The purpose of this book is to cover the first topic.

The first two chapters provide a systematic review of those aspects of general college physics and mathematics that are applicable to the study of radiological physics. Chapter 1 is a review of basic mathematics including an introduction to differential and integral calculus. The chapter is brief and to the point with a large number of practical examples. Of particular value is the outline at the end of Chapter 1 which condenses the review into two pages, providing a very useful reference. Chapter 2 is a review of basic physical concepts and includes a very good introduction to relativity and the uncertainty principle. The sections dealing with electricity, magnetism and electronics are particularly well written with most of the examples and circuits relevant to radiation producing machines. It is unfortunate that limits have to be set, so that a review of optics and sound is not included among the topics presented. These areas are pertinent to some of the new tools found in diagnostic radiology departments today, but are really outside the realm of basic radiological physics. Chapters 1 and 2 may very well be the most important chapters in the book, since they cover topics not reviewed in many of the other basic textbooks.

Chapters 3, 4, and 5 cover atomic and nuclear physics, machines for producing radiation and the interaction of radiation with matter. These chapters are well written and thorough without causing the typical resident to read information he will feel is beyond his needs.

The section on microwave linear accelerators in Chapter 4, and the distinctions which are made between the absorption of electrons and heavy particles and monoenergetic electrons and beta rays presented in Chapter 5 are particularly well written at the appropriate level.

The final Chapter deals with dose, kerma, and exposure. Instead of taking the historical approach of discussing exposure first, the author covers absorbed dose and kerma before introducing exposure. He then demonstrates the close relationship between kerma in air and exposure. I have used this approach in my lecture series for several years and was most pleased to see a basic textbook concurring with it. It is also delightful to see the author emphasizing the necessity of electron equilibrium for the practical measurement of exposure.

Although it is difficult to completely evaluate a textbook without receiving feedback from your students, I must nevertheless credit the author with producing a first-rate textbook on the fundamentals of radiological science.

WILLIAM G. VAN DE RIET, PH.D.

BOOKS RECEIVED

CONGENITAL DEFORMITIES OF THE HAND AND FOREARM. By H. Kelikian, M.D., Emeritus Professor of Orthopedic Surgery, Northwestern University Medical School; Senior Attending Orthopedic Surgeon, Northwestern Memorial Hospital, Chicago; and Consulting Orthopedic Surgeon, Bethany Methodist, Henrotin and Augustana Hospitals, Chicago, Ill. Cloth. Pp. 1,007, with 620 illustrations. Price, $35.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

THE VERTEBRAL COLUMN. By Bernard S. Epstein, M.D., Chairman, Department of Radiology, The Long Island Jewish-Hillside Medical Center, New Hyde Park, N. Y.; Professor of Radiology, School of Medicine, Health Science Center, State University of New York, Stony Brook, N. Y. An Atlas of Tumor Radiology, Philip J. Hodes, M.D., Editor-in-Chief. Cloth. Pp. 628, with many figures. Price, $40.00. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill. 60601, 1974.

THE RADIOLOGIC CLINICS OF NORTH AMERICA. Symposium on The Skull and Brain. Guest Editors, Norman E. Chase, M.D., and Irvin I. Kricheff, M.D. August, 1974, Volume XII, Number 2. Pp.

193, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1974.

Abdominal Echography: Ultrasound in the Diagnosis of Abdominal Conditions. By Ellis Barnett, F.R.C.P. (Glas.), D.M.R.D., F.F.R., Consultant Radiologist, Western Infirmary and Royal Maternity Hospital, Glasgow; and Patricia Morley, M.B., B.S., D.M.R.D., Medical Assistant, Radiodiagnostic Department, Western Infirmary, Glasgow. Paper. Pp. 138, with many figures. Price, £4.40. The Butterworth Group, 88 Kingsway, London WC2B 6AB, England, 1974.

Diagnosis and Treatment of Pituitary Tumors. Proceedings of a Conference Sponsored jointly by the National Institute of Child Health and Human Development and the National Cancer Institute in Bethesda, Md., Jan. 15–17, 1973. Edited by Peter O. Kohler, M.D., Professor of Medicine, Baylor College of Medicine, Houston, Texas; and Griff T. Ross, M.D., Ph.D., Clinical Director, National Institute of Child Health and Development, Bethesda, Md. Cloth. Pp. 343, with some figures. Price, $36.00. American Elsevier Publishing Company, Inc., 52 Vanderbilt Avenue, New York, N. Y. 10017, 1974.

Manpower Issues and Voluntary Regulation in Medical Specialty System. By Herbert J. Lerner. Paper. Pp. 200. Price, $5.95. Prodist: New York. Neale Watson Academic Publications Inc., 156 Fifth Avenue, New York, N. Y. 10010, 1974.

Traumatic Dislocation of the Patella: a Radiographic Investigation. By Sven Schelle and Lars Mårtenson, Departments of Diagnostic Radiology and Orthopaedic Surgery I, Sahlgrenska Sjukhuset, 413–45 Göteborg, Sweden. Acta Radiologica Supplementum 336. Paper. Pp. 160, with some figures. Price, Sw.Kr. 50:-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

Myocardial Scintigraphy and Estimation of Regional Blood Flow with Xenon-133. By Ossi Honhola, Institute of Diagnostic Radiology, Meilahti Hospital, University Central Hospital, Helsinki, Finland. Acta Radiologica Supplementum 337. Paper. Pp. 65, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

Optimised Tomography: Theoretical and Practical Analyses of the Elimination of Depiction Errors in Tomography. By Kurt Åstrand and Sven Reichmann, Department of Diagnostic Radiology III, Sahlgrenska Sjukhuset, S-411 32 Göteborg, Sweden. Acta Radiologica Supplementum 338. Paper. Pp. 76, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

Lymphography in Tuberculosis. By Ilkka Suramo, Department of Roentgenology, University of Oulu, Oulu, Finland. Acta Radiologica Supplementum 339. Paper. Pp. 58, with some figures. Price, Sw.Kr. 40:-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

^{75}Se-Sodium Selenite Scintigraphy in Diagnosis of Tumours. By Eeva Nordman, Department of Radiation Therapy, Central Hospital of Middle Finland, Jyväskylä, Finland. Acta Radiologica Supplementum 340. Paper. Pp. 78, with some figures. Price, Sw.Kr. 45-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

Pelvimetry with Image Intensifier Camera: a Low Radiation Dose Method. By Ilpo Lauteala, Department of Radiology, University Central Hospital, Turku, Finland. Acta Radiologica Supplementum 341. Paper. Pp. 132, with some figures. Price, Sw.Kr. 50-. Acta Radiologica, Box 2052, 103 12 Stockholm, Sweden, 1974.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston Salem, N. C. 27103. Annual Meeting: Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

American Radium Society
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Atlantic City, N. J., June 15–19, 1975.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following city during this year: Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1975 was September 30, 1974.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

American Association of Physicists in Medicine
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D.C. 20010. Annual Meeting.

American Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting: Sonesta Beach Hotel, Key Biscayne, Fla., Oct. 31–Nov. 3, 1974.

American Nuclear Society
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545. Winter Meeting: Washington, D.C., Oct. 27–Nov. 1, 1974.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

Fourteenth International Congress of Radiology
Rio de Janeiro, Brazil in 1977.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Univ. of South Florida, Tampa, Fla.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

Third Congress of the European Association of Radiology
Secretary General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland, Meeting: June 22–27, 1975.

Second Asian and Oceanian Congress of Radiology
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters 2161 Taft Ave., Manila, Philippines.
Meeting: Manila, Philippines, Nov. 10–14, 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society, Chapter ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

American Osteopathic College of Radiology
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

American Thermographic Society
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Mark M. Mishkin. The Graduate Hosp., Univ. of Pennsylvania, Philadelphia, Pa. 23rd Annual Meeting: San Diego, Calif., May 4–6, 1975.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Maj. Marco J. DiBiase, M.D., Radiology Service, 130th Station Hosp., APO N. Y. 09102. Next meeting Dec. 5–8 in Nürnberg, Germany.

Blockley Radiological Society
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Daniel E. Lewis, Grant Hosp., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Central Texas Radiology Society
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Robert L. Stein, 43 Dormidera Ave., Piedmont, Calif. 94611. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary. Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Kansas City Radiological Society
President-Secretary, Dr. Rodger W. Lambie, 701 E. 63rd St., Ste. 108, Kansas City, Mo. 64110.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Robert F. Conti, Holy Cross Hosp., Fort Lauderdale, Fla. 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert L. May, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. John A. Knote, 716 Carrolton Blvd., West Lafayette, Ind. 47906.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce Saxe, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Isaac Sanders, 1429 Valley View Rd. 13, Glendale, Calif. 91202. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MICHIGAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bldg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 16, 1975.

NEW ENGLAND SOCIETY FOR RADIATION ONCOLOGY
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

NORTH AMERICAN SOCIETY OF CARDIAC RADIOLOGY
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Pablo Ramos, Box 1618, Bismarck, N. D. 58501. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 5301 F Street, Sacramento, Calif. 95819. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 175 S. Merkle Rd., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1975.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Stanley G. Seat, 476 Prospect St., LaJolla, Calif. 92037. Meets the first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Chairman, Dr. Roy G. Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting: St. Francis Hotel, San Francisco, Calif., May 28–31, 1975.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. James O. Richardson, Jr., 280 Mamaronek Ave., White Plains, N. Y. 10605. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wis. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociacon de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Meijia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1, Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St.

Catherine St. West, Montreal 107, Que., Canada. Annual Meeting: Toronto, Ont., May 11–16, 1975.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professiona Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, Lincolns Inn Fields, London WC2A 3PN, England.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

Société Canadienne-Française de Radiologie
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

The Royal Australasian College of Radiologists
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

Sociedad Argentina de Radiología
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiología
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiología
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Cordoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting: Madrid, Apr. 24–26, 1975.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Électrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, du Massif Central, d'Outre-Mer et d'Étranger. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dept., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14-1151, Tehran, Iran. The Society meets on the second Saturday of each month. 5th Iranian Congress of Radiology, April 12–17, 1975. Deadline for acceptance of papers, Jan. 20, 1975.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel. Nineteenth National Radiology Congress: Tel-Aviv, Israel, Nov. 6–8, 1974.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Briggs, Michael. Traumatic pneumocephalus. *Brit. J. Surg.*, April, 1974, *61*, 307–312. (Address: Regional Neurosurgical Unit, Brook General Hospital, London SE18, England.)

The presentation and management of 33 patients with traumatic pneumocephalus have been analyzed and 2 main groups recognized. The first consisted of 12 patients who developed an intracerebral aerocele, the onset of which was delayed for up to 52 days from the time of injury. These patients presented with signs of raised intracranial pressure which required urgent surgical treatment.

The intracranial air in the second group was situated in the subdural or subarachnoid spaces and was usually visible on the first roentgenogram taken after injury. Adequate early prophylactic antibiotic treatment against meningitis was of great importance to these patients, who did not all require surgical intervention.

The association between traumatic pneumocephalus and fractures of the frontal and facial bones should lead to the careful scrutiny of the roentgenograms of patients with these injuries for evidence of air in the subarachnoid or subdural spaces.—*John H. Harris, M.D.*

Bernstein, Joel, and Hoffer, Paul B. Use of the delayed brain scan in differentiating calvarial from cerebral lesions. *J. Nuclear Med.*, Aug., 1974, *15*, 681–684. (Address: Paul B. Hoffer, M.D., University of Chicago, Department of Radiology, 950 E. 59th Street, Chicago, Ill. 60637.)

Ninety-two sequential positive brain scans on 69 patients were reviewed; 12 mc Tc^{99m} pertechnetate was given and scans were obtained immediately and repeated in 3 to 4 hours. The relative activity in the lesion was compared with the radioactivity in the rim or periphery which represents primarily the extracellular fluid space of the skull and scalp with a minor vascular component.

A total of 100 lesions were found in the 92 scans: 34 lesions were calvarial, and 66 were intracranial; 22 of the calvarial lesions showed a decreased activity on the delayed scan, 4 showed increased activity and 8 were unchanged. The 4 lesions showing increased activity on the delayed scan included 2 cases of metastases, a surgical bone flap and Paget's disease of the skull.

Fifty-five intracranial lesions showed an increased activity on the delayed scan, whereas 5 had decreased activity and 6 were unchanged. Three of the 5 lesions which showed decreased activity on the delayed scan were arteriovenous malformations. The other 2 cases were 1 acoustic neuroma and 1 craniopharyngioma.

If the lesion showed decreased activity on the delayed scan, it was calvarial in 82 per cent of the cases. If the lesion showed increased activity on the delayed scan, it was intracranial in 93 per cent of the cases. Lesions showing no change in radioactivity were almost equally divided between the calvarial and intracranial sites.

The combination of early and delayed scans is of value in the determination of the location of a lesion in the brain scan.—*Charles W. Cooley, M.D.*

Potter, Guy D. Radiologic assessment of the facial nerve. *Otolaryng. Clin. North America*, 1974, *7*, 343–355. (From: Columbia-Presbyterian Medical Center, 622 West 168th Street, New York, N. Y. 10032.)

The area through which the facial nerve runs can be seen in the conventional radiographic views—the posteroanterior, Towne, Stenvers, and Schüller projections. However, the actual course of the facial nerve can be demonstrated only on tomograms. Therefore, tomographic examination of the ears should be performed in patients with facial palsy.

The coronal and lateral tomographic projections should be used for adequate visualization of the internal auditory canal.

Twenty degree oblique tomograms should be obtained for visualization of the first knee of the facial nerve canal and the horizontal portion of the facial nerve canal.

Lateral tomograms should be employed for visualization of the vertical portion of the facial nerve canal.—*E. Frederick Lang, M.D.*

Neck and Chest

Rosen, Irving B., Walfish, Paul G., and Miskin, Murray. The use of B mode ultrasonography in changing indications for thyroid operations. *Surg., Gynec. & Obst.*, Aug., 1974, *139*, 193–197. (From: Departments of Surgery, Medicine, and Radiological Sciences, Mount Sinai Hospital and The University of Toronto Medical School, Toronto, Ontario, Canada.)

The solitary thyroid nodule, which does not concentrate radioactive iodine or which is hypofunctioning, is a major concern to the clinician because this type of nodule presents the greatest risk of malignancy. Since 20 per cent of such nodules are cysts, and are rarely associated with malignancy, their excision does not appear to be indicated. Conservative management of this group includes needle aspiration and careful follow-up study. In the other 80 per cent more aggressive treatment is proper.

In an attempt to differentiate patients into 2 groups, only 1 of which should be treated aggressively, 215 patients with enlarged thyroid glands were evaluated during an 18 month period. Of these, 112 were found to have dominant or solitary nodules

and clinically and biochemically proved euthyroidism. A nonfunctioning or hypofunctioning nodule was present in 100 of these as shown by radioscintiscanning, and of these, the 77 in whom there was laboratory confirmation of histologic type form the basis of this report.

Six errors were made in predicting the type of lesion found by pathologic examination, and in none of these 6 did the sonographic prediction result in adverse influence on patient management. The reliability rate by ultrasonography is thus 92 per cent. The difficulties which were encountered arose in the identification of a small cyst or a mixed solid and cystic lesion. The transducer used in this study did not resolve echoes in volumes less than 1 cm. in diameter, and, furthermore, loculated cysts appeared solid. Thus, the errors are all false positives, and no neoplasm went undiscovered. The ability of ultrasound to discriminate cystic and solid lesions permits intelligent, conservative treatment of the cysts thus found.

The examination is carried out without patient discomfort and can be repeated without side effects. This noninvasive method requires approximately 15 minutes and provides information not otherwise accessible by clinical or isotope methods. Since there is no radiation hazard, it is available to pregnant and pediatric patients.—*E. Frederick Lang, M.D.*

Strutynsky, Natalie, Balthazar, Emil J., and Klein, Robert M. Inflammatory pseudotumours of the lung. *Brit. J. Radiol.*, Feb., 1974, *47*, 94–96. (From: Department of Radiology, New York Medical College, and Flower and Fifth Avenue Hospital, New York, N.Y.)

The designation inflammatory pseudotumor of the lung indicates a benign lesion which may have a varied histologic appearance.

Morphologically it simulates a true tumor. Histologically it contains inflammatory cells with mature fibroblastic components and remnants of pre-existing parenchyma in a mass with a striking lack of mitoses.

Clinically, it often suggests a diagnosis of neoplasm or tuberculosis.

It is predominantly composed of fibroblasts, histiocytes, and plasma cells. In various lesions all phases of vascular proliferation may be found. These tumors have been called sclerosing hemangiomas of lung, xanthomas, xanthogranulomas, histiocytomas, plasma cell tumors, and postinflammatory tumors. All of these terms probably represent the same lesion, the difference being in the dominant cellular component.

They may be solitary or multiple, vary between 1 and 8 cm. in diameter, and are sharply delineated, without associated pleural changes. Fifty per cent of them are detected in asymptomatic patients.

Two patients are reported in detail. One patient was a 32 year old male seen 12 years after open lung biopsy at which time a diagnosis of Wegener's granulomatosis was made. This diagnosis, made elsewhere, was not substantiated. Examination of his chest showed multiple bilateral pulmonary nodules with confluent nodules on the left and sparing of the basal segments. The differential diagnosis included pulmonary metastases, sarcoid, Hodgkin's disease, tuberculosis, fungal infection and pneumoconiosis. An open lung biopsy disclosed a well encapsulated pulmonary tumor, which contained a predominance of plasma cells, with mature fibroblasts, histiocytes and vascular elements. This was classified as a hyalinized plasma cell granuloma, an inflammatory pseudotumor.

A second patient, a 30 year old male, also asymptomatic, was investigated because of an abnormal chest roentgenogram. The original study suggested right upper lobe pneumonia, but tomography showed 2 well defined masses which subsequently, under antibiotic treatment, became larger and coalesced. A thoracotomy and lung biopsy were carried out. The lesion was composed of alveolar spaces completely obliterated by an infiltrate of polymorphonuclear leukocytes, lymphoid cells, plasma cells, epithelioid cells, and histiocytes. The pathologic diagnosis was lymphoid granuloma of lung, an inflammatory pseudotumor.

In patients with this lesion, the prognosis is excellent and excision results in permanent cure.—*E. Frederick Lang, M.D.*

Rossi, Nicholas P., and Goplerud, Clifford P. Recurrent catamenial pneumothorax. *A.M.A. Arch. Surg.*, Aug., 1974, *109*, 173–176. (Address: Nicholas P. Rossi, M.D., Division of Thoracic and Cardiovascular Surgery, Department of Surgery, University Hospitals and Clinics, Iowa City, Iowa 52242.)

The authors report 6 cases of catamenial pneumothorax and review the literature.

Catamenial pneumothorax involves the association of pneumothorax with menstruation. The patient complains of chest pain which occurs only with or just before the onset of the menses. Typically, the patient is in the same age group in which endometriosis is frequently diagnosed and the symptoms are almost always on the right side of the chest.

The etiology is unclear and many theories have been proposed including endometriosis, diaphragmatic rents, and hormonal factors.

The authors suggest that this condition represents a normal or an exaggerated pulmonary response to menstruation and may relate to increased prostaglandin (prostaglandin F_2 tromethamine) levels in the serum and the menstrual fluid. They postulate an altered bronchial epithelium which is the source for the air. The fact that anovulatory agents reduce the prostagladin levels and also prevent the recurrent

pneumothorax in many cases support the authors' theory.—*Daniel P. Link, M.D.*

Holman, B. Leonard, Lesch, Michael, Zweiman, Franklin G., Temte, John, Lown, Bernard, and Gorlin, Richard. Detection and sizing of acute myocardial infarcts with ^{99m}Tc (Sn) tetracycline. *New England J. Med.*, July, 1974, *291*, 159–163. (Address: B. Leonard Holman, M.D., Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Mass. 02115.)

Twenty-one patients suspected of myocardial infarct were studied in the coronary care unit. Eight patients without evidence of ischemic heart disease served as a control population.

Twenty millicuries of technetium (Sn) tetracycline was administered intravenously and imaging performed using a scintillation camera 24 hours after injection. Patients in the coronary care unit were examined 24 to 72 hours after the onset of symptoms.

To differentiate cardiac activity from that in the liver, spleen and stomach, selected patients had technetium pertechnetate and/or technetium sulfur colloid administered intravenously after the tetracycline images were obtained. Fourteen patients had evidence of acute myocardial infarction by serial enzyme determination, electrocardiography, etc., (10 transmural, 4 intramural). Five additional patients had equivocal evidence of infarction, 1 had recurrent arrythmias without infarction and the remaining 8 patients had no evidence of coronary artery disease. Focal myocardial concentration of technetium Sn tetracycline was present in all 14 patients with acute infarction. Two of the 5 patients with equivocal evidence of infarction had focal myocardial concentration of tracer. Scintigraphy was normal in all 8 patients without evidence of coronary disease and in the patient with recurrent arrythmia.

Images obtained earlier than 24 hours following tracer administration were unsatisfactory due to the high blood level, which did not permit accurate separation of cardiac blood pool from the area of damaged myocardium.

In the 14 patients with documented acute infarction, there was a good correlation between the electrocardiographic site of myocardial damage and the location of increased tracer concentration on scintigrams. A comparison in the size of infarct as judged by maximum serum CPK and the size of the infarct by scintigraphy showed a linear relationship with a correlation coefficient of $r=0.85$. The intensity of infarct activity was evaluated as a function of time from onset of initial symptoms, and appeared maximum between the first and fourth day following onset of symptoms.

This study demonstrates the feasibility of detecting acutely infarcted myocardium with a radiotracer that is selectively sequestered by microbic tissue.—*H. William Strauss, M.D.*

Abdomen

Spanos, Panayiotis K., Simmons, Richard L., Rattazzi, Luca C., Kjellstrand, Carl M., Buselmeier, Theodore J., and Najarian, John S. Peptic ulcer disease in the transplant recipient. *A.M.A. Arch. Surg.*, Aug., 1974, *109*, 193–197. (Address: Richard L. Simmons, M.D., University of Minnesota Hospitals, Box 185, Minneapolis, Minn. 55455.)

Management of peptic ulcer disease in renal transplant patients was reviewed in 377 patients at the University of Minnesota hospitals. Bleeding in the post-transplant period is a serious and many times lethal complication which occurs in about 10 per cent of transplant patients. Uremia patients show an increase in acid production and an increase in gastrin secretion. The coagulopathy of uremia, the intermittent anticoagulation required for dialysis, and hyperparathyroidism all contribute to the bleeding problems in the post-transplant period.

The authors strongly recommend aggressive prophylaxis for ulcer disease in the pre-transplant period. Prophylactic operations include: truncal vagotomy and pyloroplasty (V and P); vagotomy and antrectomy (V and A); vagotomy and pyloroplasty with hiatal hernia repair; and selective vagotomy and pyloroplasty. Twenty patients had ulcer history and V and P operations and only 5 of these patients bled in the postoperation period. Only a few patients in this series received a V and A procedure, but none of these patients had bleeding in the post-transplant period.

The authors realize that the information on this subject is incomplete and that the correct approach is unclear. However, they feel that planned pre-transplant prophylaxis can significantly reduce the incidence of post-transplant bleeding. They recommend antrectomy and vagotomy for patients with evidence of peptic disease on upper gastrointestinal examination or gastroscopy, and a V and P operation is recommended for patients with historical evidence only.—*Daniel P. Link, M.D.*

Monnier, J. P., Bigot, J. M., Chermet, J., and Fragoas, M. Les hématomes intrapariétaux du grêle. (Intraparietal hematomas of the small intestine.) *J. de radiol., d'électrol. et de méd. nucléaire*, March, 1974, *55*, 217–222. (Address: Dr. J. P. Monnier, Service du Pr. Chirurgie, C.H.U. Saint-Antoine, 184, rue du Faubourg-Saint-Antoine, F 75012, Paris, France.)

Intraparietal small intestinal hematoma may be due to lowered coagulability of the blood. This may be spontaneous or the result of anticoagulant coumarin medication; less often, it may be due to hemophilia.

The clinical history may be ill defined and not revealing, while the radiologic findings are striking and can be readily demonstrated by barium motor meal studies.

The authors report 4 cases, 3 following coumarin anticoagulant treatment and 1 due to hemophilia.

The clinical manifestations are not characteristic and suggest progressive intestinal obstruction: nausea, vomiting, colicky abdominal pain, and distention.

The differential diagnosis includes acute pancreatitis, mesenteric infarction and, less likely, abdominal manifestations and myocardial infarction.

Signs and symptoms related to the use of anticoagulants are due to the associated hemorrhage—ecchymosis, epistaxis, gingival bleeding, hematuria, and gastrointestinal hemorrhage.

Submucous hemorrhage, infiltrating into the muscularis assumes a circular pattern with retraction and narrowing of the intestinal lumen, while the mucosa remains intact. The hematomas may be 10–30 cm. long. The jejunum is involved in 75 per cent of the cases, and the clinical picture becomes that of progressive jejunal obstruction.

A barium meal examination is indispensable for localizing the involved area as well as the site of hemorrhage. Barium studies reveal considerable narrowing of the involved segment of the small intestines with sharply demarcated proximal dilatation and gaseous distention.

Patients with pre-existing gastric or duodenal lesions are likely to bleed during a course of anticoagulant therapy.

The mucosal folds are thickened, the valvulae conniventes are irregular and enlarged. Although the mucosa remains intact, and does not ulcerate, the mucosal folds flatten and disappear.

The radiologically demonstrated changes disappear in 3–4 weeks after discontinuation of the anticoagulant therapy.—*William H. Shehadi, M.D.*

Bates, T., and Kaminsky, V. Diverticulitis and ulcerative colitis. *Brit. J. Surg.*, April, 1974, *61*, 293–296. (Address: Dr. T. Bates, Department of Surgery, Guy's Hospital, London SE1, England.)

Four patients are described who had long-standing sigmoid diverticulitis in association with fulminating ulcerative colitis. Three of the 4 patients died following surgical therapy.

It is suggested by the authors that sigmoidoscopy should be performed prior to the emergency surgery for the sigmoid diverticulitis. This would alert the physician to the presence of ulcerative colitis in association with the sigmoid diverticulitis, thus allowing more appropriate management.—*John H. Harris, M.D.*

Martinez, Luis O., and Raskin, Michael M. Fat under diaphragm simulating pneumoperitoneum. *Brit. J. Radiol.*, June, 1974, *47*, 308–309. (From: Department of Radiology, Mount Sinai Medical Center, Miami Beach, Florida, and the University of Miami School of Medicine, Miami, Fla.)

Free air in the peritoneal space can usually be demonstrated as a lucent defect below either the right or left diaphragm leaf, or both, when the patient is upright. A layer of fat between the peritoneum and the diaphragm can simulate a small amount of free air when the patient is upright. The lucency due to the fat, when shown by a tangential beam, will persist with change in position of the patient, and is visible when the patient is supine. This will differentiate fat from free air. Observations in cadavers disclose that in some there is an accumulation of fat between the diaphragm and peritoneum which probably explains this phenomenon, particularly since extraperitoneal fat is almost always visible in the flanks.

A group of 7 patients is presented, none of whom was obese or receiving steroids. Two were followed for a period of 1 year because they had presented with symptoms suggesting acute abdominal disease. The lucency which was subsequently proved not to be air was almost always visible under the left diaphragm, under either the highest portion of the dome or slightly medial to it. One of the 7 had bilateral lucent defects and in another it was only on the right side.

When the crescentic lucent defect is seen under the diaphragm in both upright and recumbent roentgenograms, the diagnosis of pneumoperitoneum is unlikely, as the defect is probably due to fat.—*E. Frederick Lang, M.D.*

Coutsoftides, Theodore, and Hermann, Robert E. Nonparasitic cysts of the liver. *Surg., Gynec. & Obst.*, June, 1974, *138*, 906–910. (From: Department of General Surgery, The Cleveland Clinic Foundation, and The Cleveland Clinic Educational Foundation, Cleveland, Ohio.)

Ten patients ranging in age from 5 days to 74 years with nonparasitic cysts of the liver were evaluated with physical examination, complete blood cell count, electrolyte, liver function tests, chest roentgenography, intravenous pylography and liver scanning. Eight of the 10 patients presented with increased abdominal girth. Hepatomegaly was invariably found on physical examination. Routine laboratory studies were normal with the exception of 1 patient, who was anemic due to bleeding.

The chest roentgenogram revealed elevation of the right hemidiaphragm without pleural effusion. Only 1 patient had evidence of calcification in the right upper quadrant. Liver scans were abnormal in 7 of 8 patients showing single or multiple lesions. A single "false negative" scan was found in the 5 day infant. Selective angiographic procedures were performed in 7 patients. All were abnormal.

Treatment included total and partial excision of the cyst, unroofing of the cyst and drainage into the peritoneal cavity as well as drainage into a defunctionalized jejunal segment.

The long term prognosis in regard to liver function in patients with cyst of the liver is good and repeated efforts to drain or decompress the cystic liver are worthwhile.—*H. William Strauss, M.D.*

Liguory, C., Gouerou, H., Chavy, A., Coffin, J. C., and Huguier, M. Endoscopic retrograde cholangiopancreatography. *Brit. J. Surg.*, May, 1974, *61*, 359–362. (Address: Professeur-Agrégé M. Huguier, Hôpital Rothschild, 43, Boulevard de Picpus, 75012 Paris, France.)

Direct nonoperative injection of radiopaque material through a cannula introduced at the duodenal papilla by means of a fiberoptic duodenoscope achieved opacification of the bile ducts, the pancreatic duct or the entire biliary pancreatic ductal system in 392 (89 per cent) of 439 patients in whom it was attempted. The desired ducts were opacified in 75 per cent of cases; the bile ducts in 206 out of 278; and the pancreatic duct in 120 out of 161 cases.

The anatomic conditions found were: long common biliary pancreatic channel, 30 per cent; short common biliary pancreatic channel, 28 per cent; 2 orifices in the hepatopancreatic ampulla, 24 per cent; 2 orifices on the duodenal papilla, 18 per cent; 2 orifices on the duodenum, there being no papilla, less than 1 per cent. Morbidity resulting from the procedure consisted of infectious reactions in 20 out of 295 patients in whom the bile ducts were opacified and hyperamylasemia and abdominal pain in 28 out of 300 patients in whom the pancreatic duct was opacified.

The method is of value in diagnosing the cause of cholestatic jaundice. In nonjaundiced subjects it may complement or clarify inconclusive cholangiograms or cholecystograms obtained by other techniques. If it shows dilated bile ducts, operation should not be delayed, otherwise there is a risk of cholangitis.

Pancreatic carcinoma may be revealed by stenosis, partial or total, of the pancreatic duct or by filling defects in the pancreatic parenchyma. Both chronic pancreatitis and pancreatic carcinoma may cause partial stenosis of the pancreatic duct. A filiform appearance of the pancreatic duct suggests chronic pancreatitis.—*John H. Harris, M.D.*

Sugawa, Choichi, Raouf, Razia, Bradley, Volker, Westreich, Melvyn, Lucas, Charles E., and Walt, Alexander J. Peroral endoscopic cholangiography and pancreatography: the surgeon's helper. *A.M.A. Arch. Surg.*, Aug., 1974, *109*, 231–237. (Address: Choichi Sugawa, M.D., Department of Surgery, Wayne State University, 540 Canfield Avenue, Detroit, Mich. 48201.)

Peroral cannulation of the ampulla of Vater with retrograde cholangiography and pancreatography (ERCP) provides an additional method of visualizing the biliary and pancreatic ducts. ERCP is especially useful in the jaundiced patient of undetermined cause, the patient with unexplained biliary colic, the patient with postcholecystectomy syndrome, and in patients with suspected pancreatic disease. In the authors' experience the success rate for the examination was 76 per cent (65 of 85 patients) and the average time for the procedure was 45 minutes. The authors arbitrarily halted the procedure after being unsuccessful for 90 minutes.

In the jaundiced patient the success rate for visualization was 69 per cent. The cholangiograms revealed choledocholithiasis in 8, common bile duct stenosis in 3, periampullary carcinoma in 2, and 8 normals. In patients with suspected pancreatic disease the success rate was 63 per cent. The studies showed pseudocyst in 3, pancreatitis in 12, neoplasms in 2, and 12 normals.

An increase in the serum amylase frequently followed the examination, but resolved in 48 hours. None of the patients went on to develop pancreatitis. ERCP is contraindicated in patients with acute pancreatitis and the authors suggest antibiotic coverage when a pseudocyst is suspected.—*Daniel P. Link, M.D.*

Blood and Lymph System

Bergan, John J., Yao, James S. T., Henkin, Robert E., and Quinn, James L., III. Radionuclide aortography in detection of arterial aneurysms. *A.M.A. Arch. Surg.*, July, 1974, *109*, 80–83. (Address: John J. Bergan, M.D., Northwestern University Medical School, 303 E. Chicago Avenue, Chicago, Ill. 60611.)

This study was done to determine the potential for radionuclide aortography in the differential diagnosis of large vessel aneurysms and to compare it to plain film roentgenography and careful clinical examination in the detection of aneurysm disease. Forty-three patients were studied: 6 cases had previous aortofemoral reconstruction, and 2 had femorofemoral grafts; 28 cases had an abdominal aortic aneurysm; 1 had a thoracic aortic aneurysm and the others were without aortic aneurysm.

A dose of 20 mc Tc^{99m} albumin was injected intravenously below a constricting tourniquet. Recordings were made at 2 second intervals following release of the tourniquet, employing a scintillation camera equipped with digital videotape and Polaroid and 70 mm. cameras. Recordings were also made 2 hours after the injection. The albumin may become fixed to the intramural clot allowing visualization of the aneurysm for many hours. If Tc^{99m} pertechnetate is

used instead of Tc^{99m} albumin, the most useful images are made during the bolus phase. Delayed imaging is of no value.

Twenty-six of the 28 cases with aortic aneurysms and 3 of the 5 anastomotic aneurysms were demonstrated by the radionuclide technique; 3 previously unsuspected aneurysms were also found by this method. A negative study excluded the presence of aortic aneurysms in 7 cases (3 thoracic and 4 abdominal). Two femorofemoral grafts were well shown without aneurysm. There was 1 false positive study in which an aneurysm was not shown by contrast arteriography.

Physical examination identified 26 of the 28 aneurysms.

Plain film roentgenograms identified 23 of 28 aortic aneurysms and none of the anastomotic aneurysms.

Radionuclide aortography is of value in the determination of aortic and anastomotic aneurysms.—*Charles W. Cooley, M.D.*

Romanowski, B., and Wawrzynek, Z. Radiodiagnostic de rupture de l'aorte, suite d'accident de la route. (Radiodiagnosis of ruptured aorta following a traffic accident.) *J. de radiol., d'électrol. et de méd. nucléaire*, March, 1974, *55*, 205–209. (Address: Pr. B. Romanowski, Institut de Radiologie de l'Académie Médicale Silésienne, Katowice, Poland.)

An accident due to a frontal automobile collision may involve the driver and/or his front-seat neighbor resulting in injury to the lungs, the trachea, the bronchi, the diaphragm, or the heart. In 6 per cent of cases, rupture of the aorta occurs.

During the collision, the bony thorax is held by the dashboard, against which it strikes, or by the safety belt. The heart and arch of the aorta are violently thrown forward, while the descending aorta is held back by the periaortic tissues and the parietal pleura.

In 95 per cent of cases, the aorta ruptures at the level of the isthmus and in 5 per cent the rupture is at the level of the aortic bulb which, at this level, is covered by the pericardium.

The diagnosis of rupture of the aorta is based on radiologic examination rather than on clinical findings. The plain film roentgenogram of the chest is often of adequate and immeasurable value. However, aortography provides the significant information needed by the surgeon before surgical intervention.

In 40 per cent of the cases, there is a complete tear of the aorta with complete separation and/or displacement at the site of the rupture.

Clinical symptoms experienced by the patient are severe chest pain, which gradually diminishes during the succeeding days. As mediastinal hemorrhage increases, dyspnea increases and dysphagia progressively worsens until total esophageal obstruction occurs.

Intrathoracic hemorrhage is manifest by a fall in arterial tension. Dissecting aneurysm is accompanied by a blowing murmur to the left of the sternum.

Three case reports are presented on patients 34, 30, and 25 years old, respectively. Two patients died despite surgical intervention and 1 made an uneventful recovery after operation.—*William H. Shehadi, M.D.*

Skeletal System

Serjeant, G. R., Ennis, J. T., and Middlemiss, Howard. Haemoglobin SC disease in Jamaica. *Brit. J. Radiol.*, Nov., 1973, *46*, 935–942. (From: Department of Biochemistry, M.R.C. Abnormal Haemoglobin Unit, Tennis Court Road, Cambridge, England.)

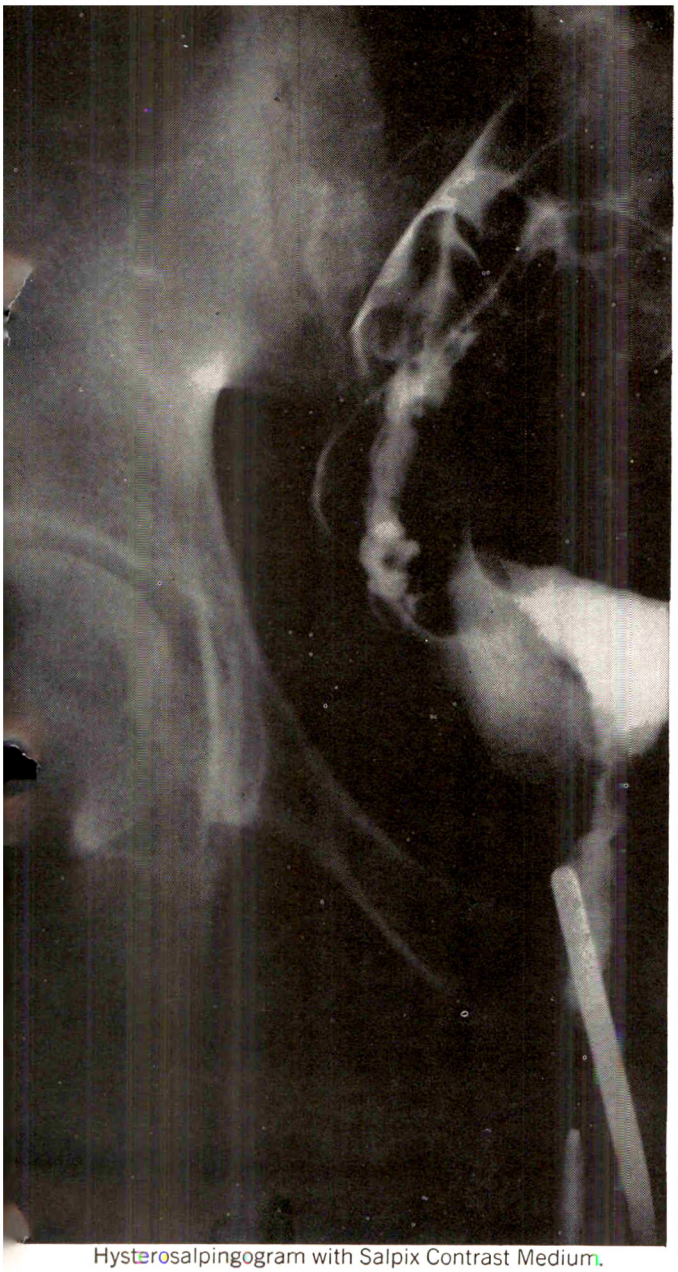
A radiographic survey, including examinations of the skull, legs, thoracolumbar spine, chest, pelvis, femora, shoulders, and hands and wrists, was made of a series of 84 patients, 44 male and 40 female, who were clinically stable and without acute symptoms. All had hemoglobin S and C demonstrated on starch gel electrophoresis, and in each hemoglobin O was excluded.

The usual changes due to osteoporosis and marrow expansion were common. Osteoporosis was most commonly shown in the metacarpals, phalanges, tibia, and skull, and was observed in 58 per cent. In 35 per cent medullary expansion was shown as increase in the thickness of the diploic layer of the skull, expansion of the lower third of the femur, "squaring" of the metacarpals, and expansion of the tibia or ribs.

The most characteristic bone lesions, however, are those attributed to avascular necrosis, and such changes were found in 39 per cent. The commonest sites were the distal femora, the proximal tibia, and the humerus. The usual medullary calcification occurred, but in addition cortical infarctions with extensive periosteal reaction were shown. In some patients these were strikingly bilaterally symmetric. Cortical thickening with narrowing of the medullary cavity was encountered in 57 per cent of the patients.

In addition to the usual exaggeration of concavity of the vertebral end plates, typical step signs with intravertebral protrusion of a segment of the cortex from the upper or lower surface were also encountered frequently.

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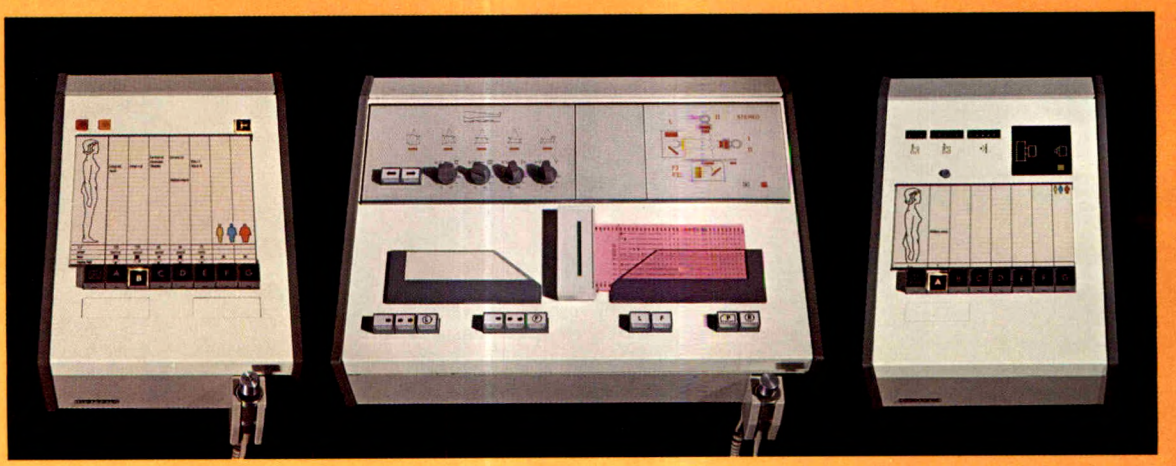
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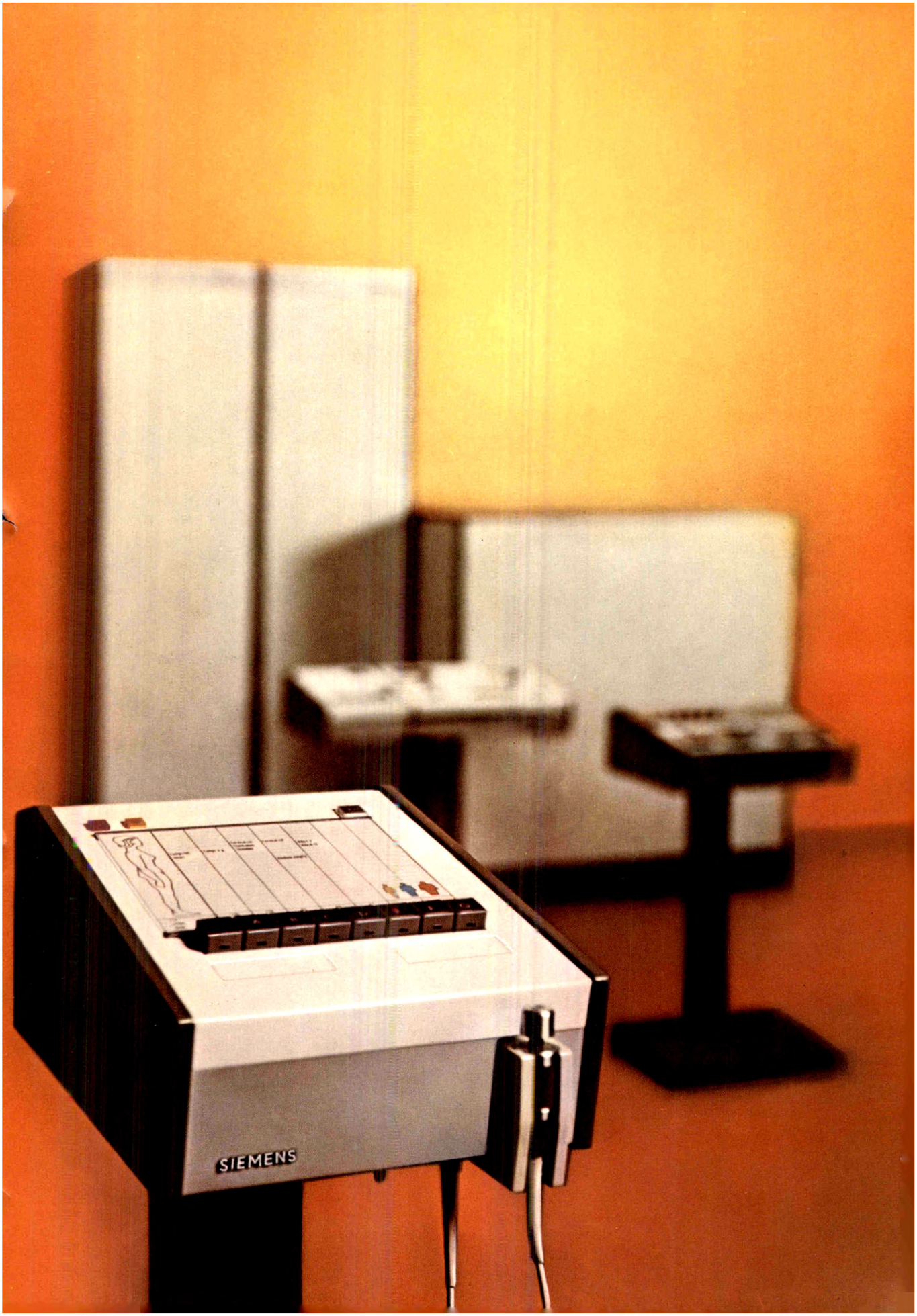
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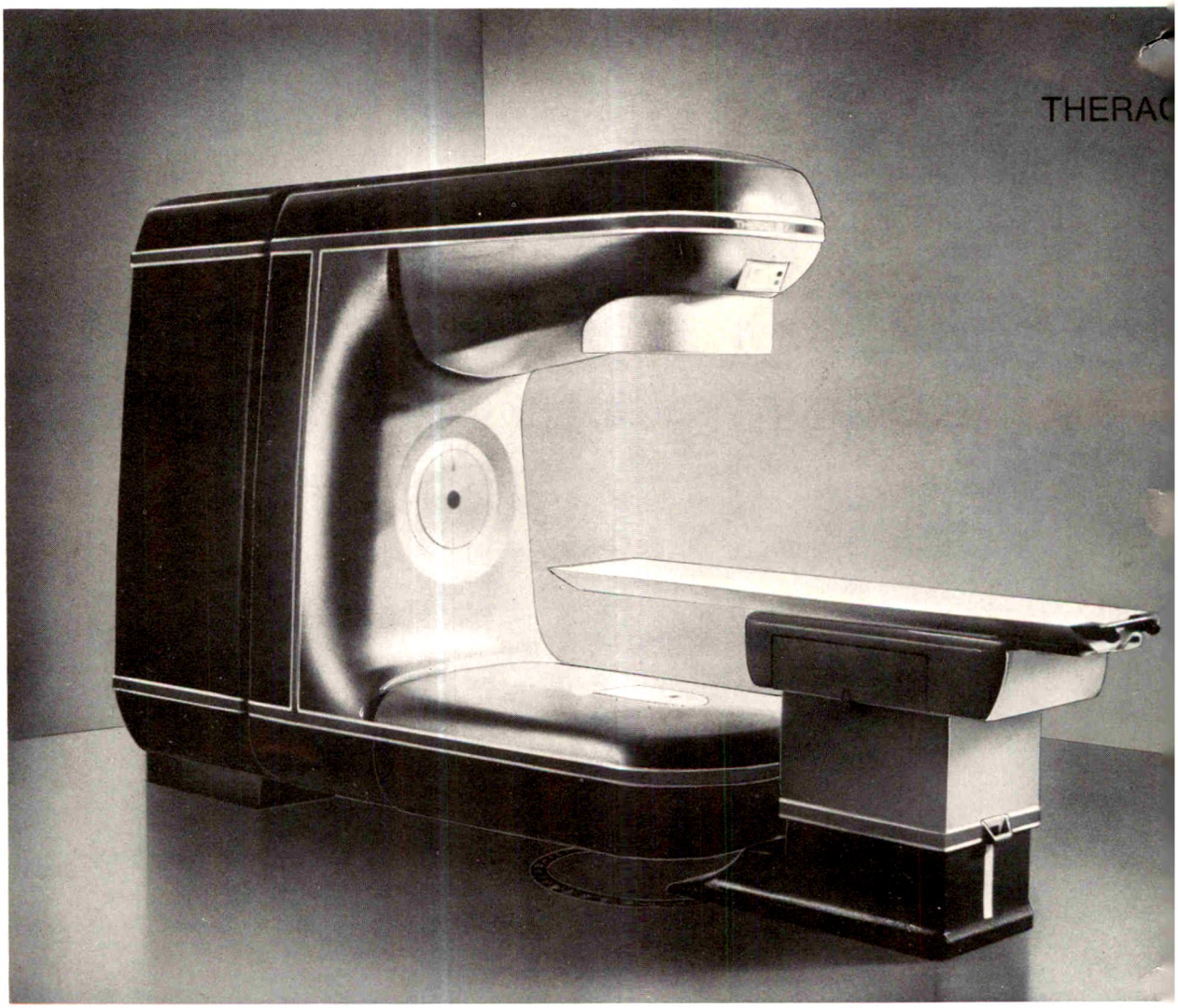

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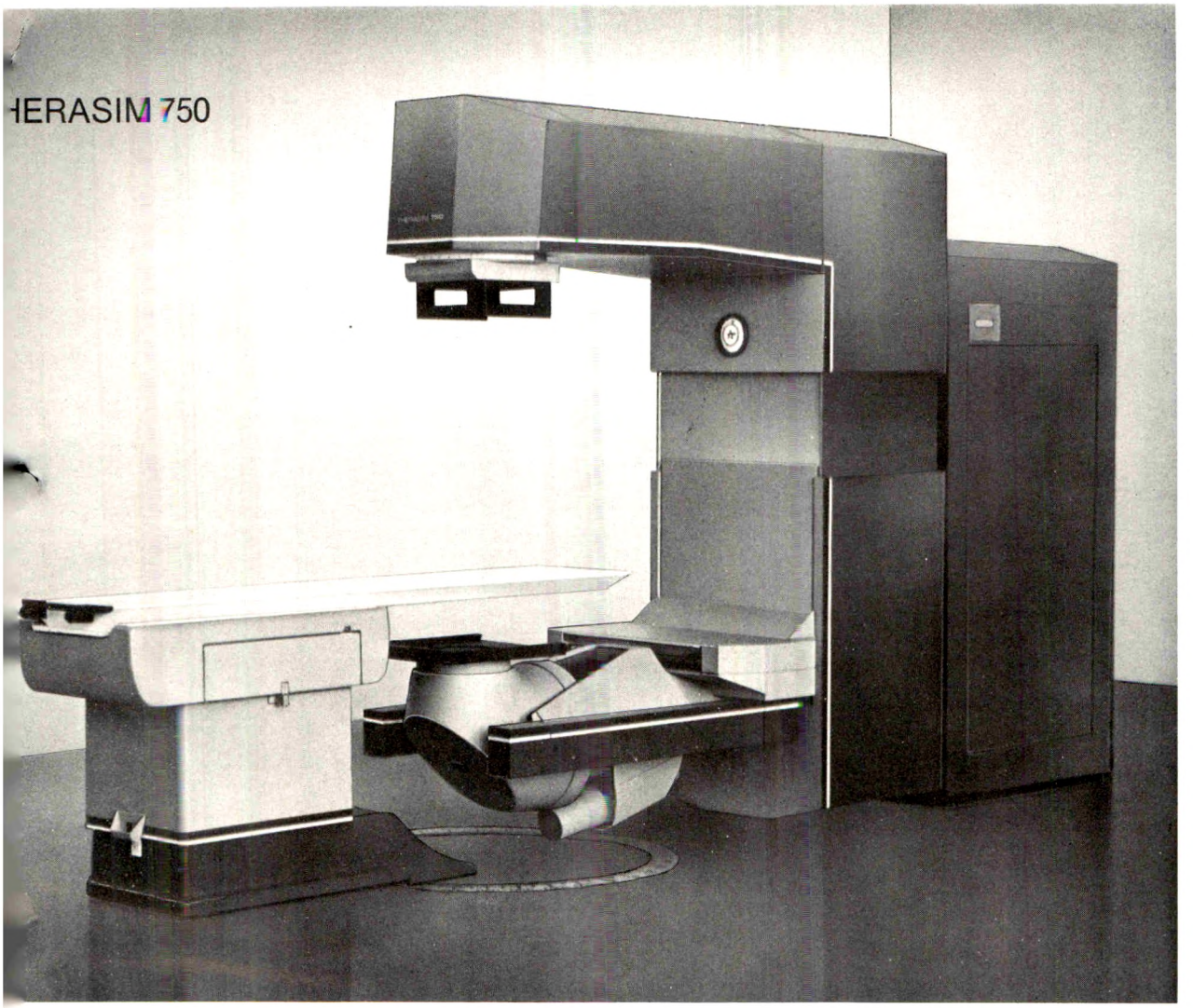
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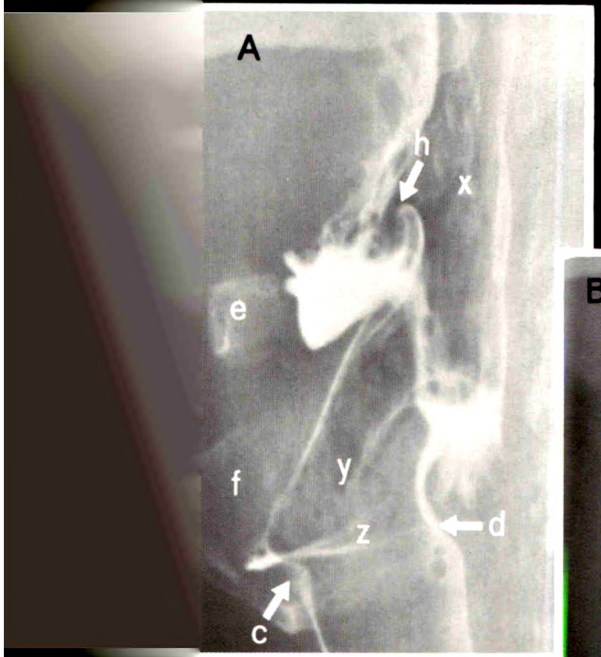
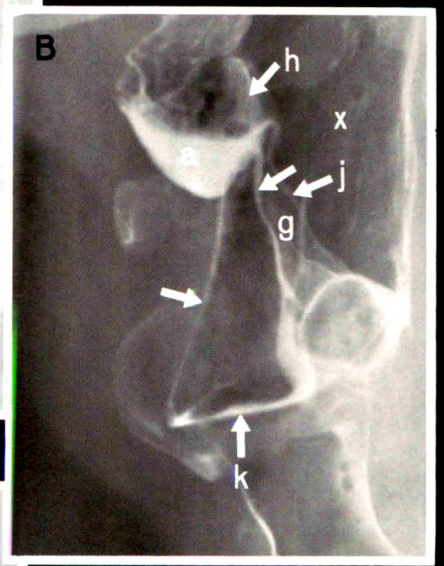
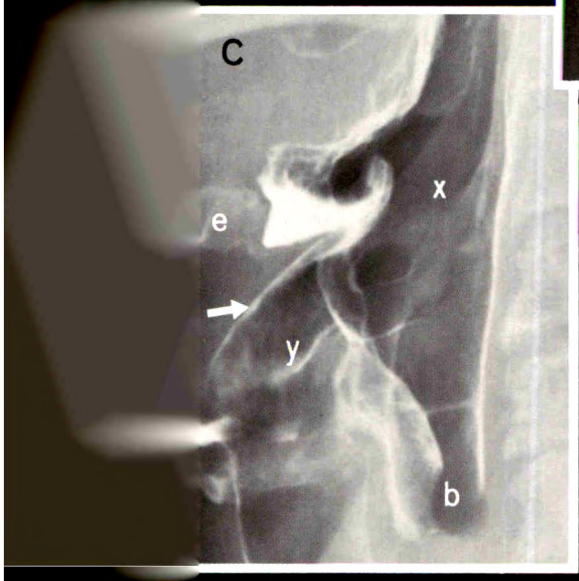


Figure I—Normal Laryngogram.

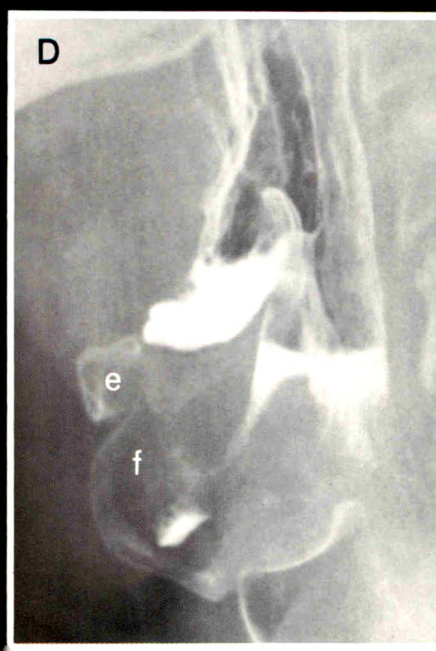
A. Quiet Breathing: Oropharynx (x); laryngeal vestibule in hypopharynx (y); laryngeal ventricles in hypopharynx (z); valleculae (a) pyriform sinuses (b); anterior commissure (c); posterior commissure (d); hyoid bone (e); calcified thyroid cartilage (f); epiglottis (h).



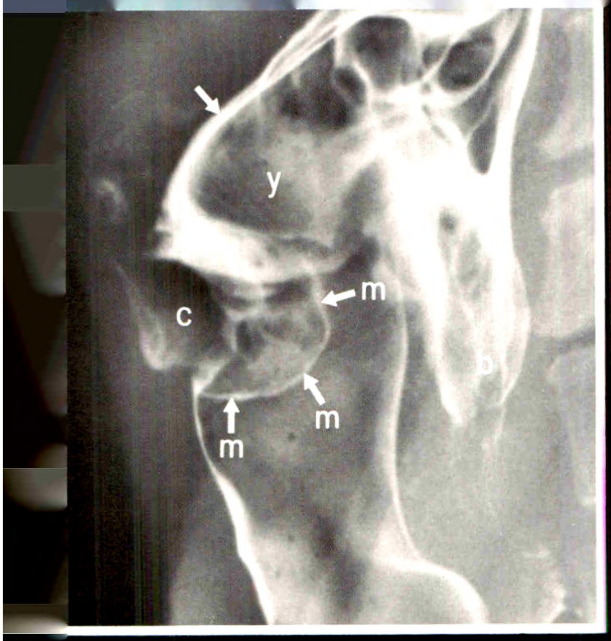
B. Phonation: When th vowel "eee" is sounded at high pitch, the oropharyn (x) becomes distended wit air. The epiglottis (h) a sumes a more vertical pos tion billowing out the arye iglottic fold (g) revealin its free margins (j). The sli like laryngeal ventricle (k may now be seen. Note th change in the configuratio of the valleculae (a) and a terior wall of the laryngea vestibule (arrow); the latte is now delicately curved t ward the cervical vertebra



C. Modified Valsalva: This is likened to blowing the trumpet with lips closed and cheeks fully expanded. Even greater distention of the oral pharynx (x) and laryngeal vestibule (y) develop with reversal of the gentle curve of the anterior wall of the latter (arrow). Note how well the pyriform sinuses (b) are now visualized. The hyoid bone (e) lies higher in the neck.



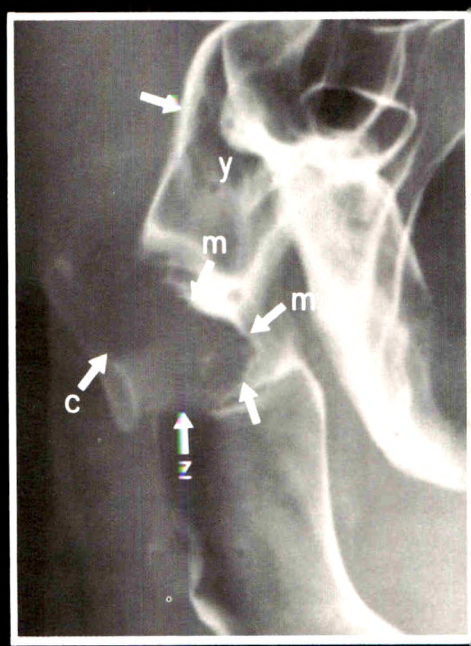
D. True Valsalva: As one strains against the closed glottis, the previously elevated hyoid bone (e) (seen in Fig. C) now descends to rest upon the thyroid cartilage (f). This is cogent evidence of normal tissue resilience in the pre-epiglottic space.



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Figure II—Subglottic Tumor at Anterio Commissure.

Modified Valsalva: The laryngeal vestibule (y) is well distended and its anterior wal (arrow) bulges anteriorly as should be during this maneuver. Note too, the distended pyr form sinuses (b). A large polypoid mass (m involves and extends below the anterior po tion of the laryngeal ventricle. Note total di ruption of the anterior commissure (c).



igure III—True Vocal Cord Tumor with Marked An- erior Commissure Involvement.

Modified Valsalva: The opacified irregularly lobu- ated laryngeal mucosa (m) lies well behind the site of the normal anterior commissure (c). The anterior portion of the laryngeal ventricle (z) too has been destroyed. Most of the anterior wall (arrow) of the laryngeal vestibule (y) appears unaffected.

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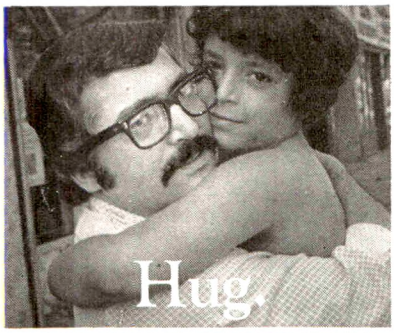
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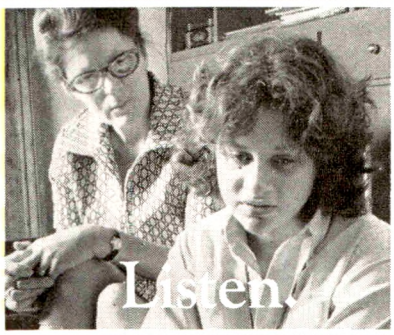
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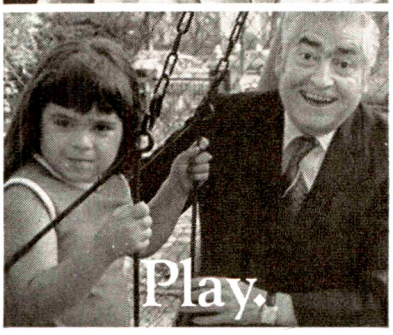
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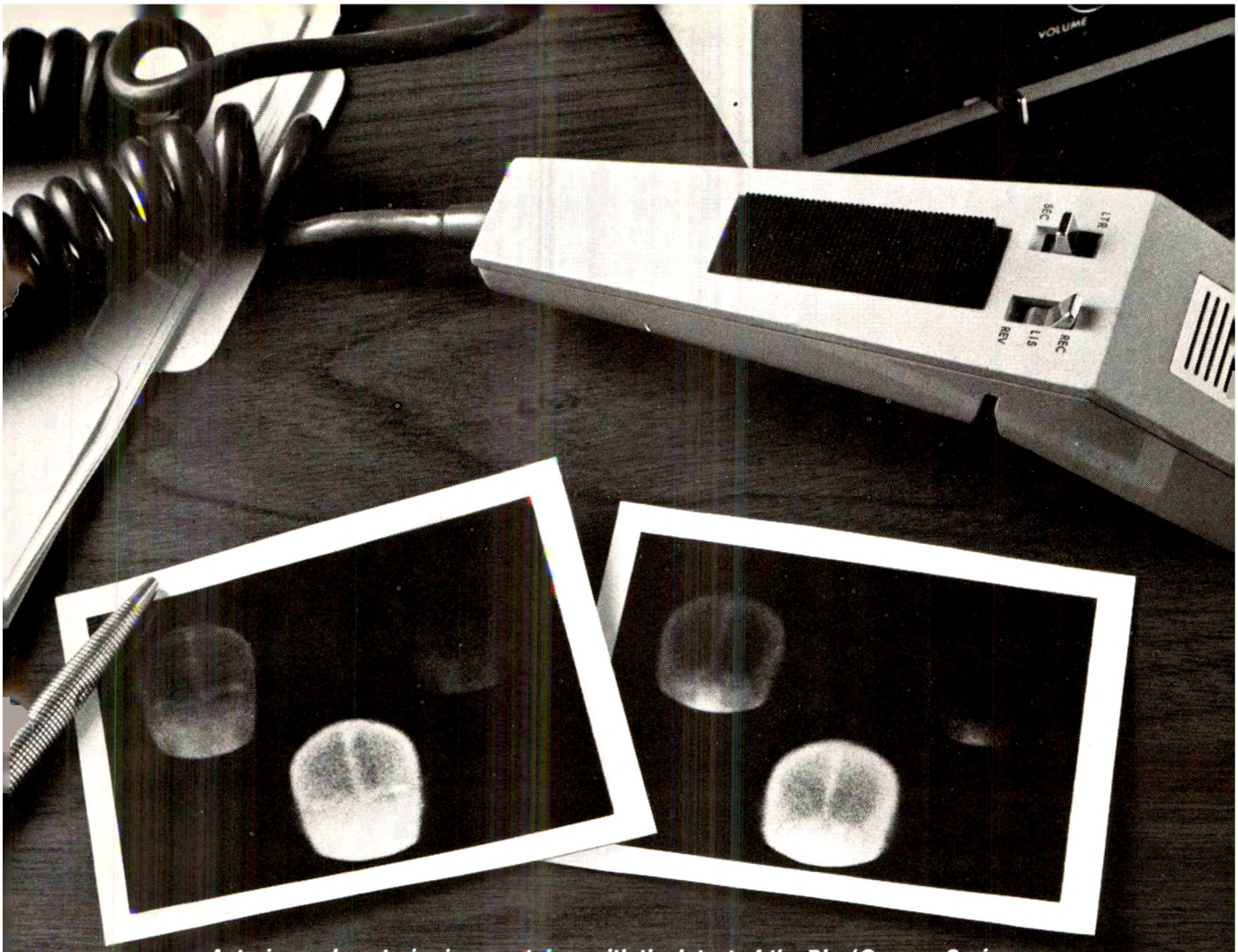
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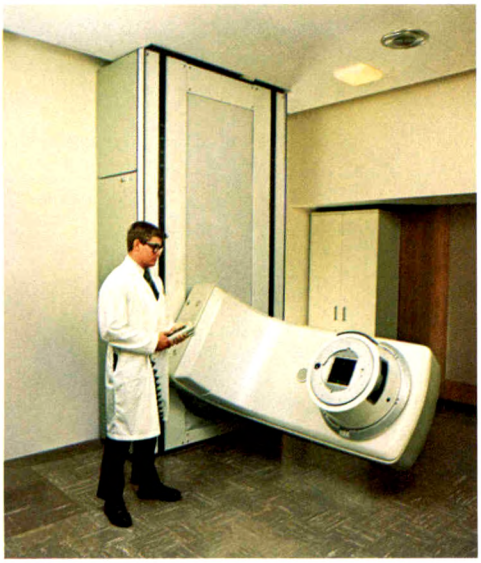
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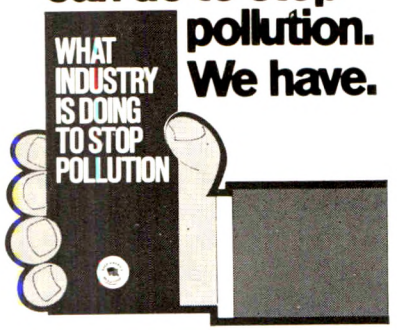


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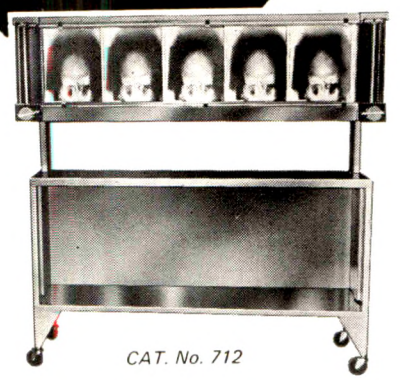
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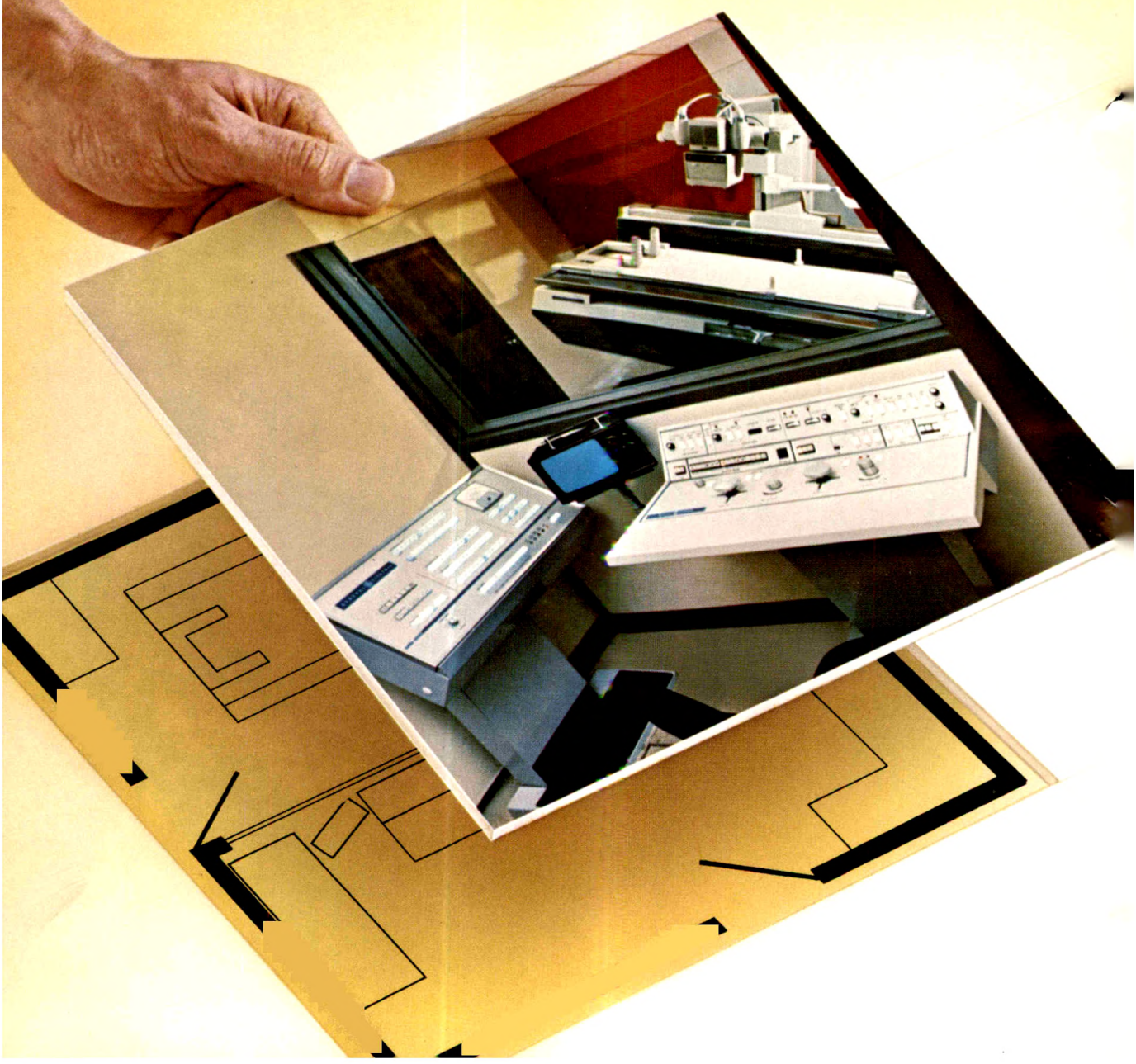
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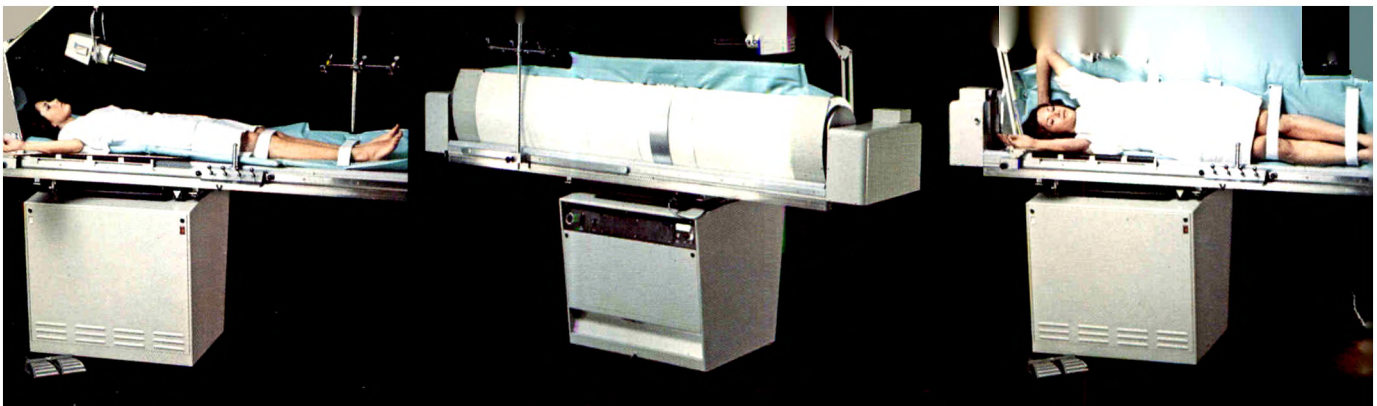
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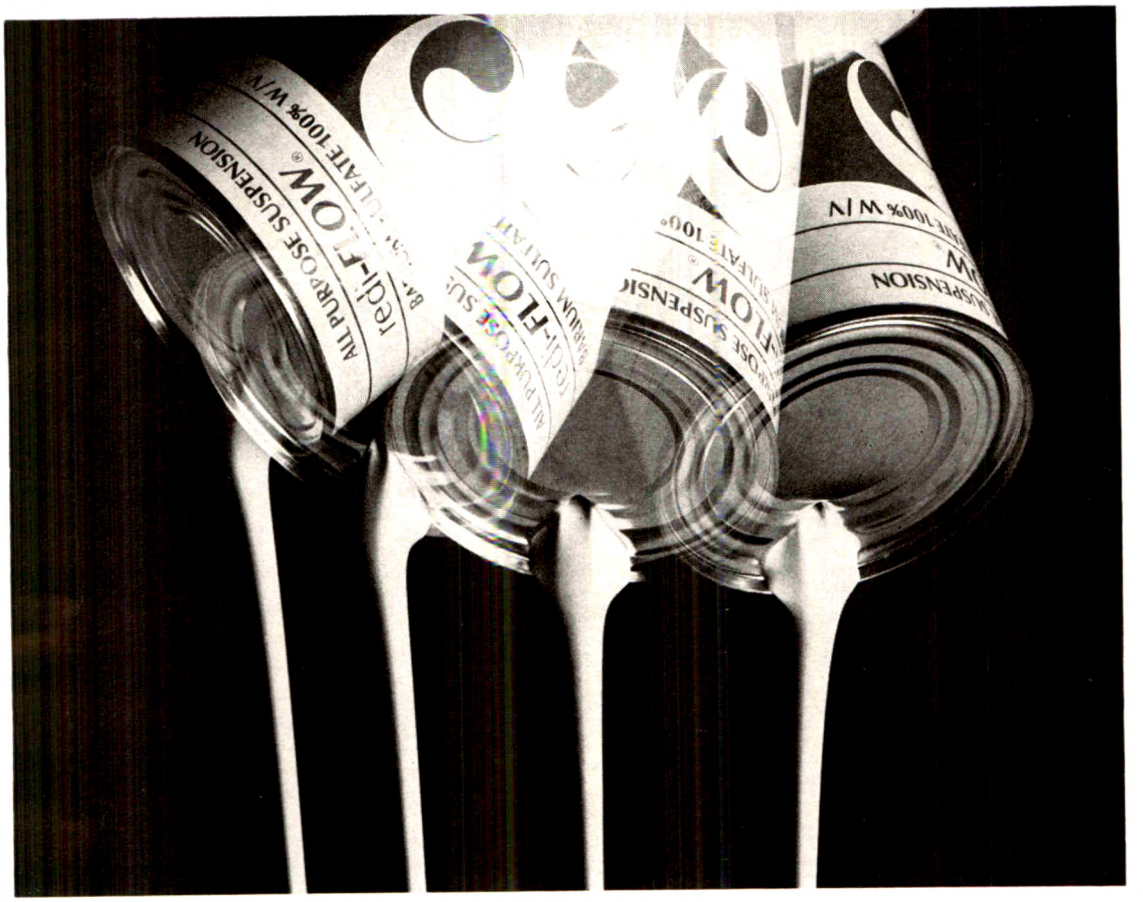
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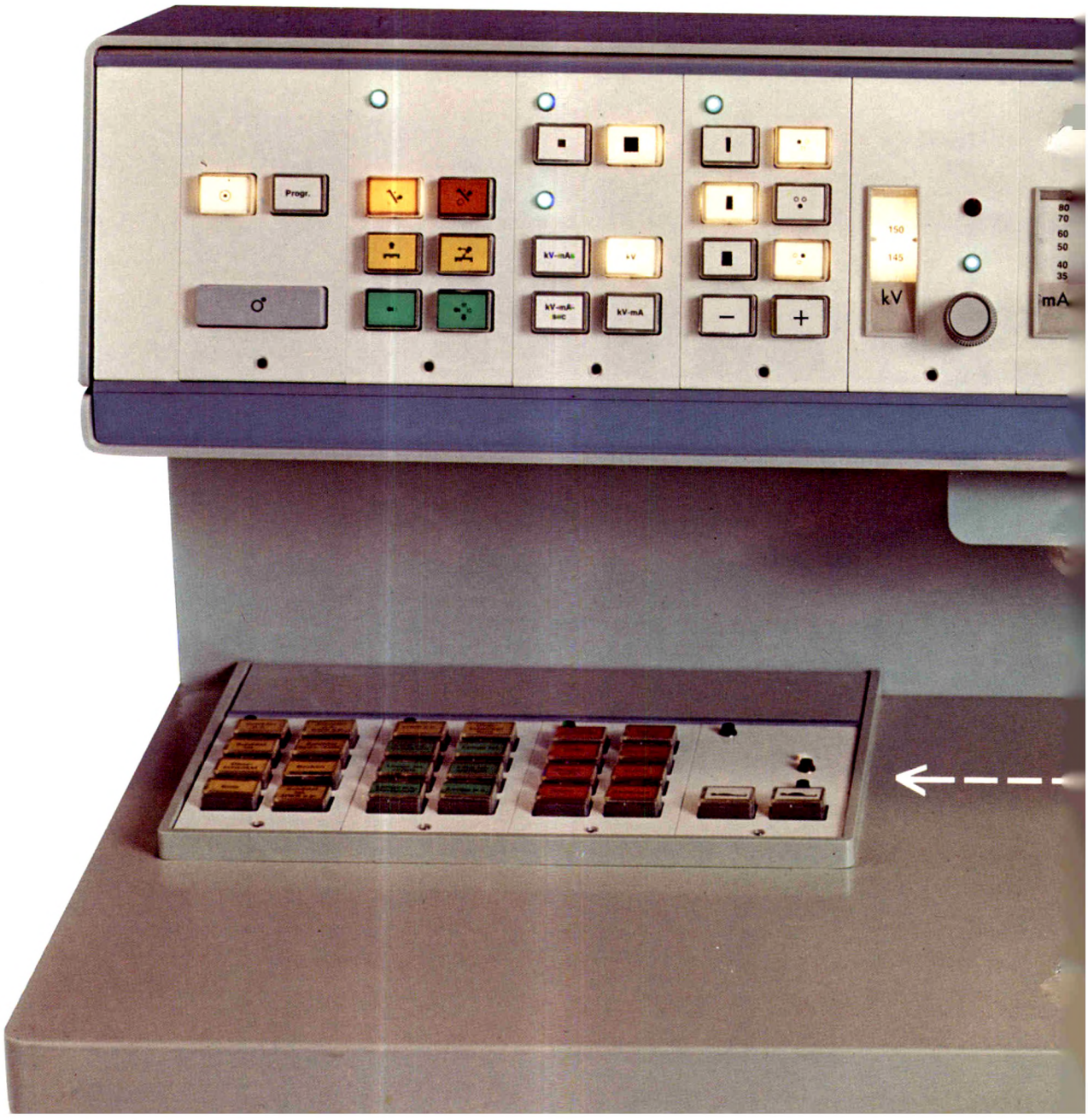
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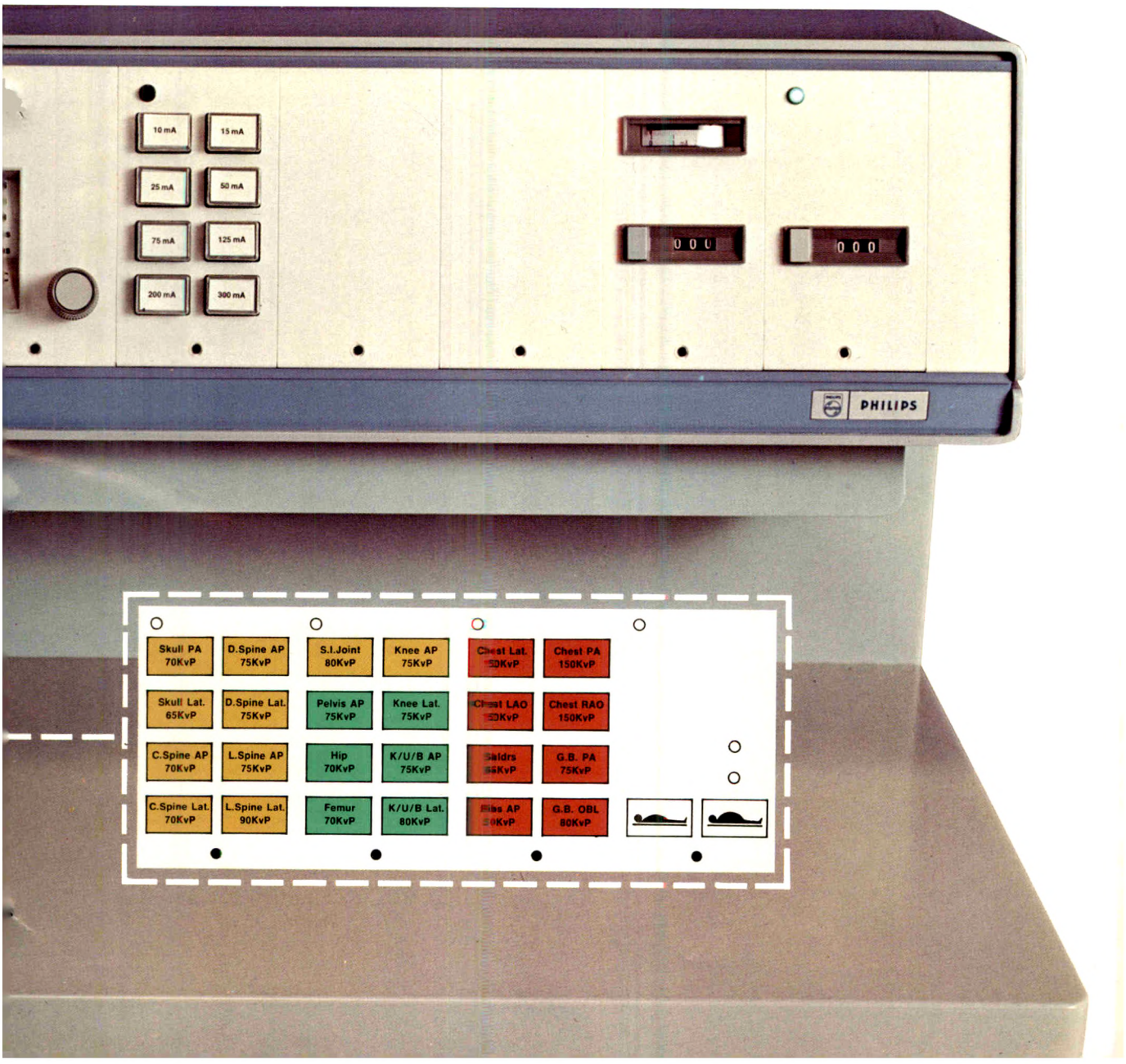
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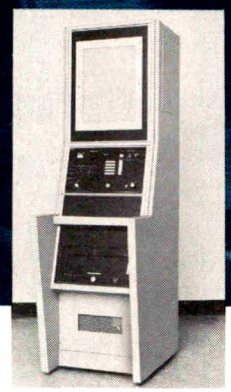
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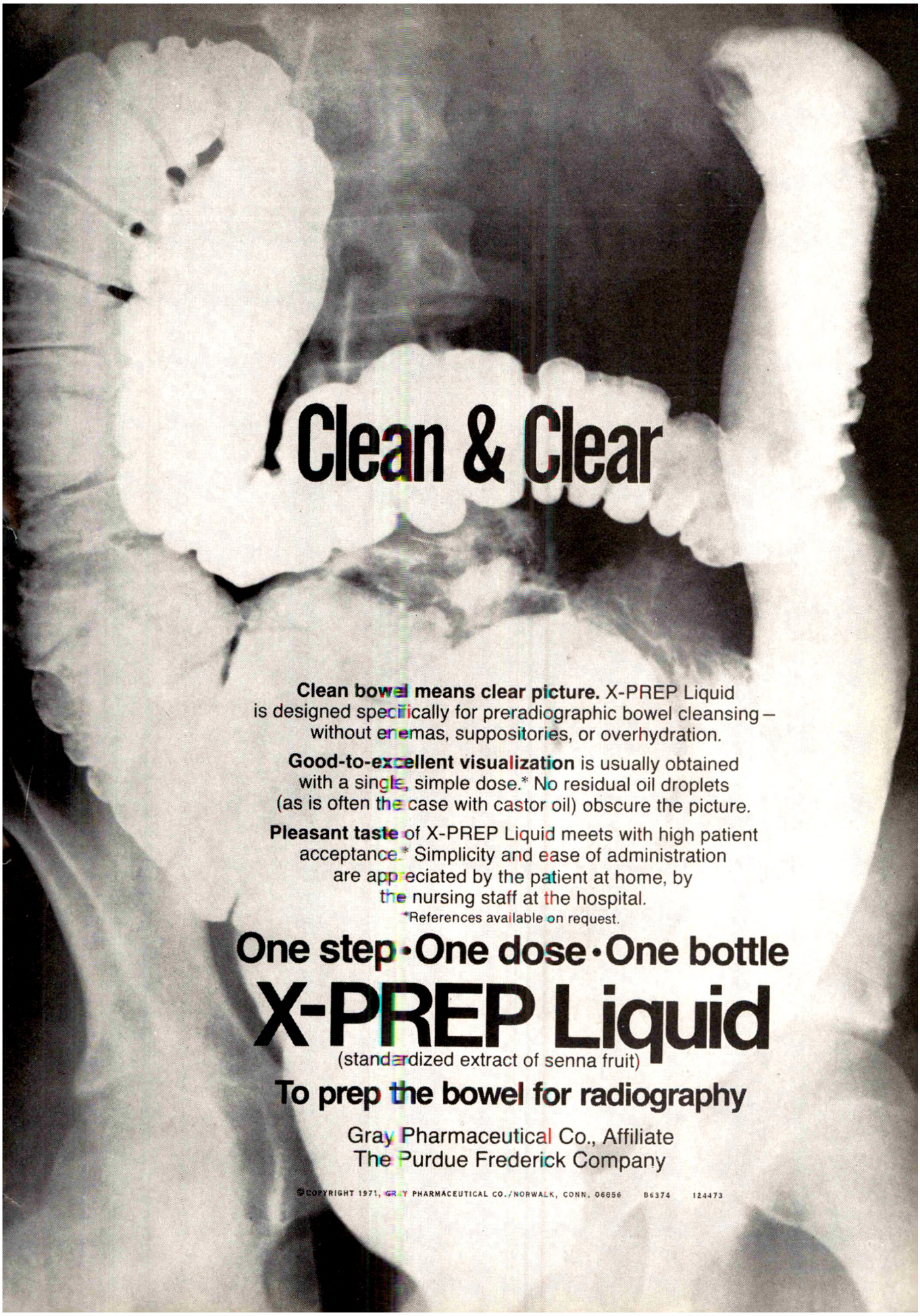
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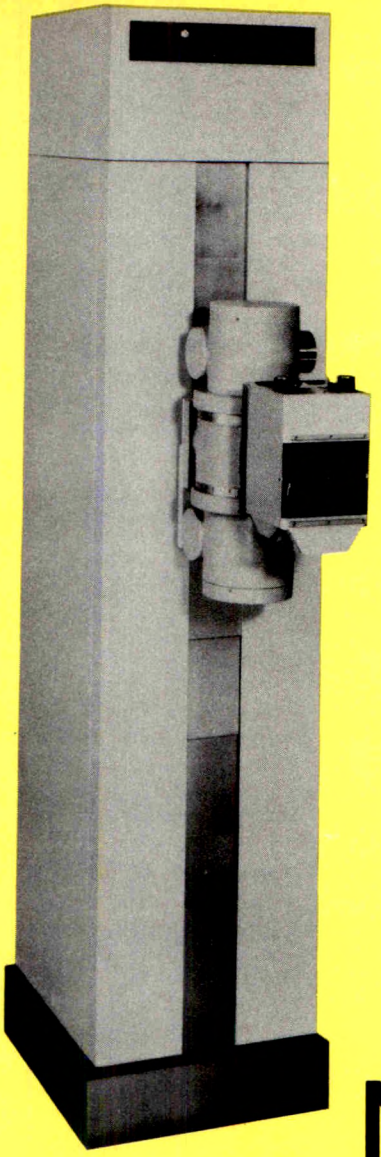
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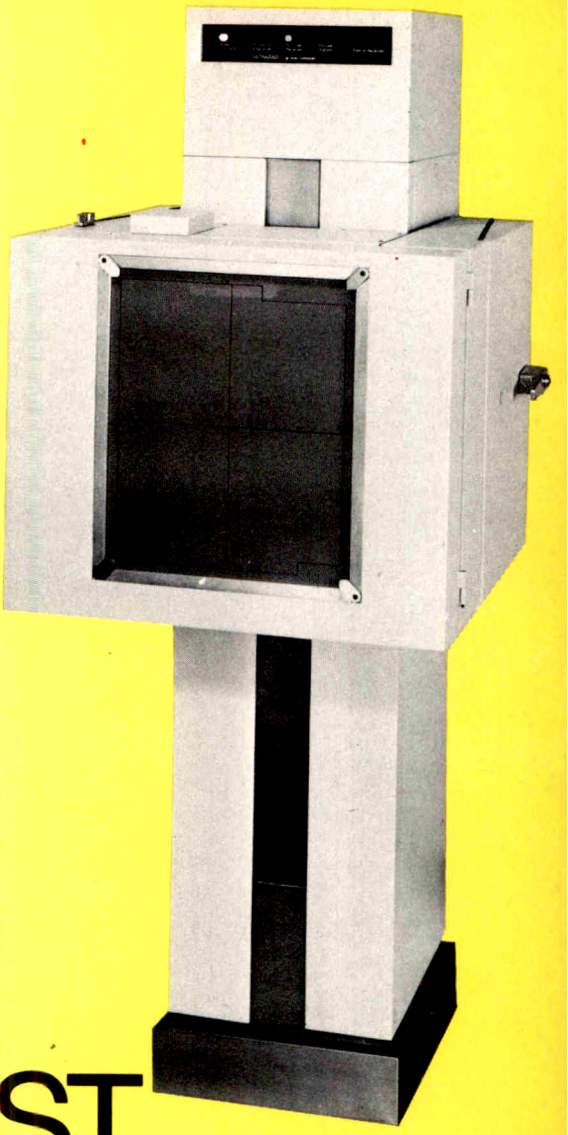
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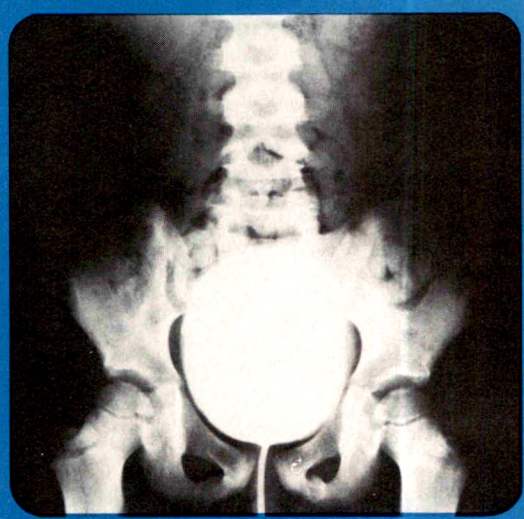
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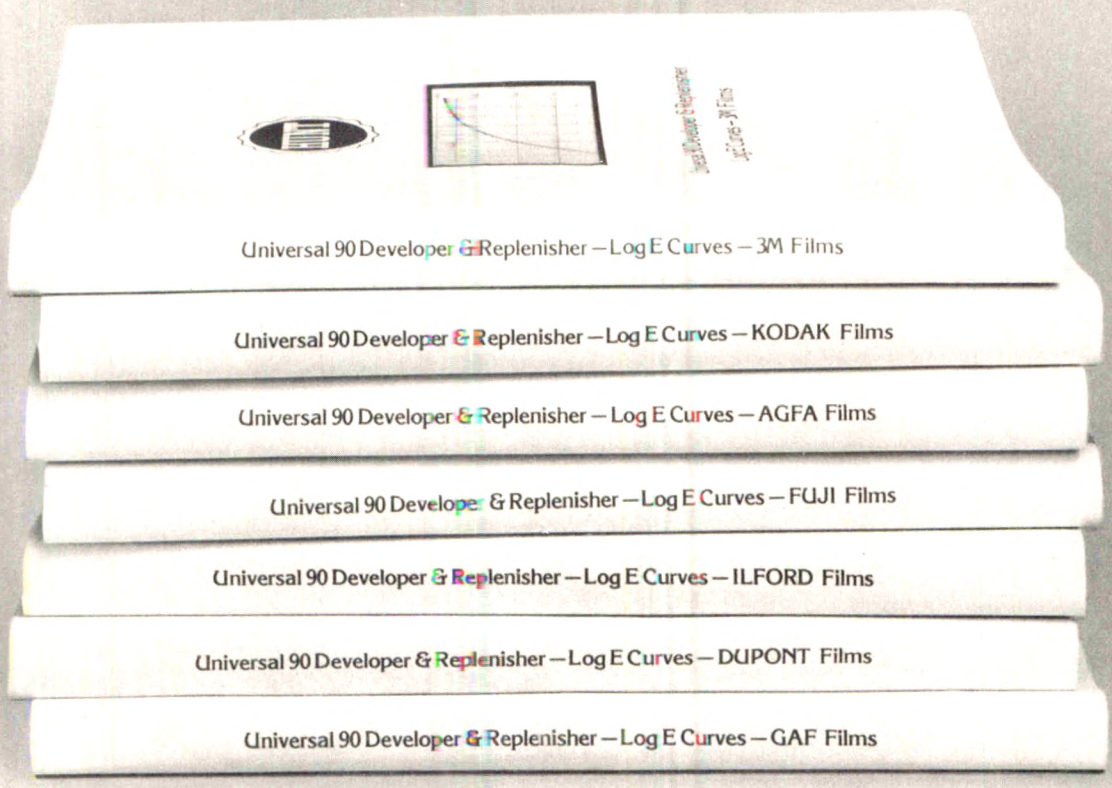
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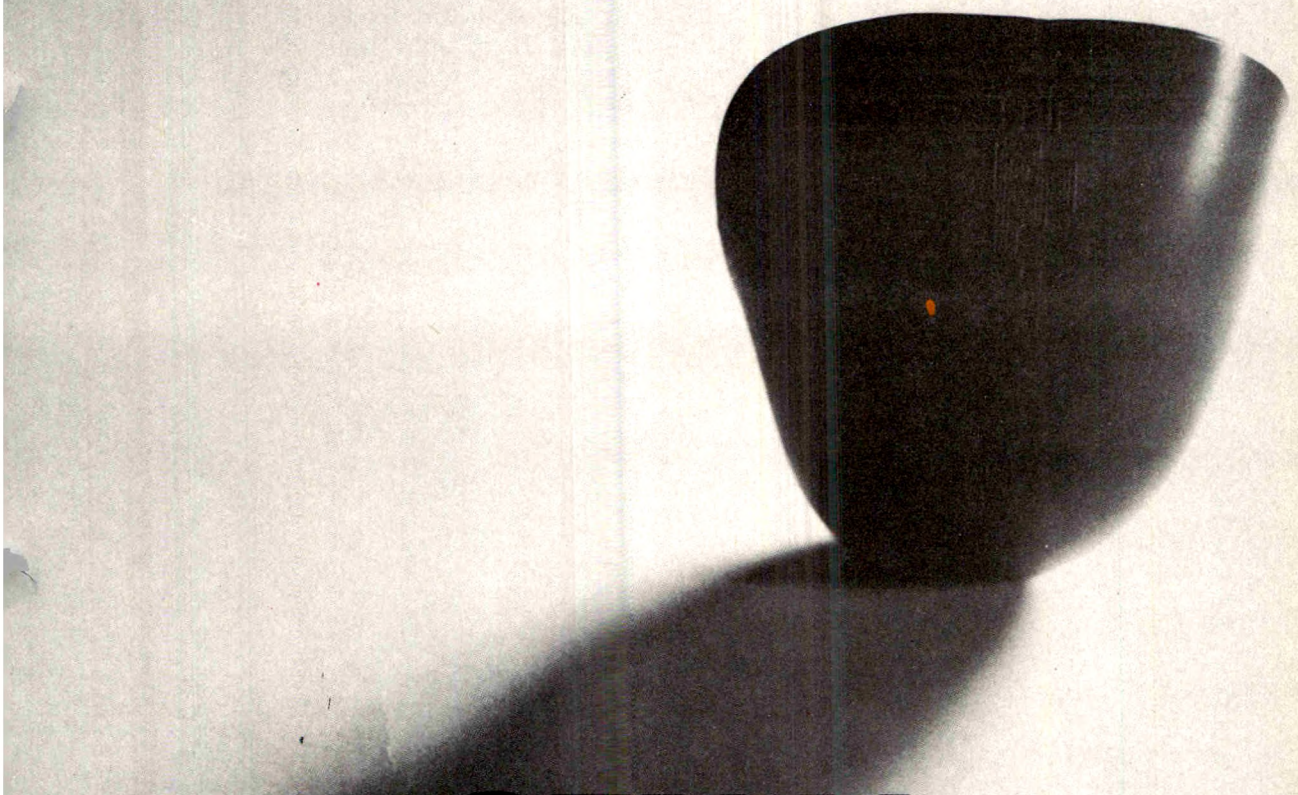
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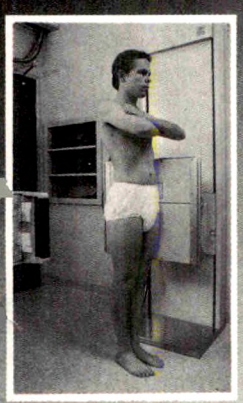
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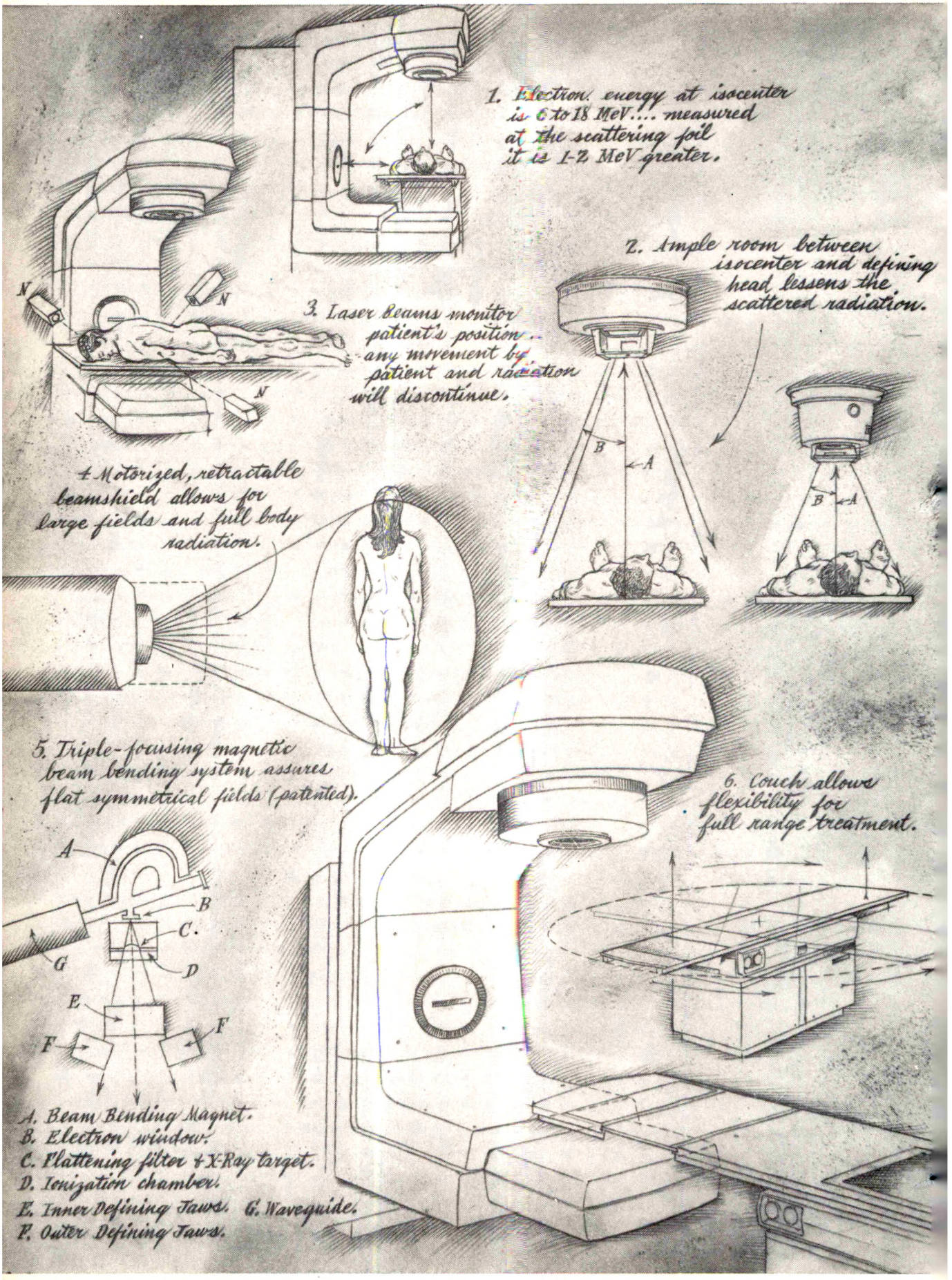


References: "Gonadal Shielding in Diagnostic Radiology"
DHEW Publication (FDA) 74-8028, June 1974
Bureau of Radiological Health, Rockville, Maryland 20852
"An Acceptable Gonadal Shield," R. F. Brown, B. M. Burnett, V. Benary
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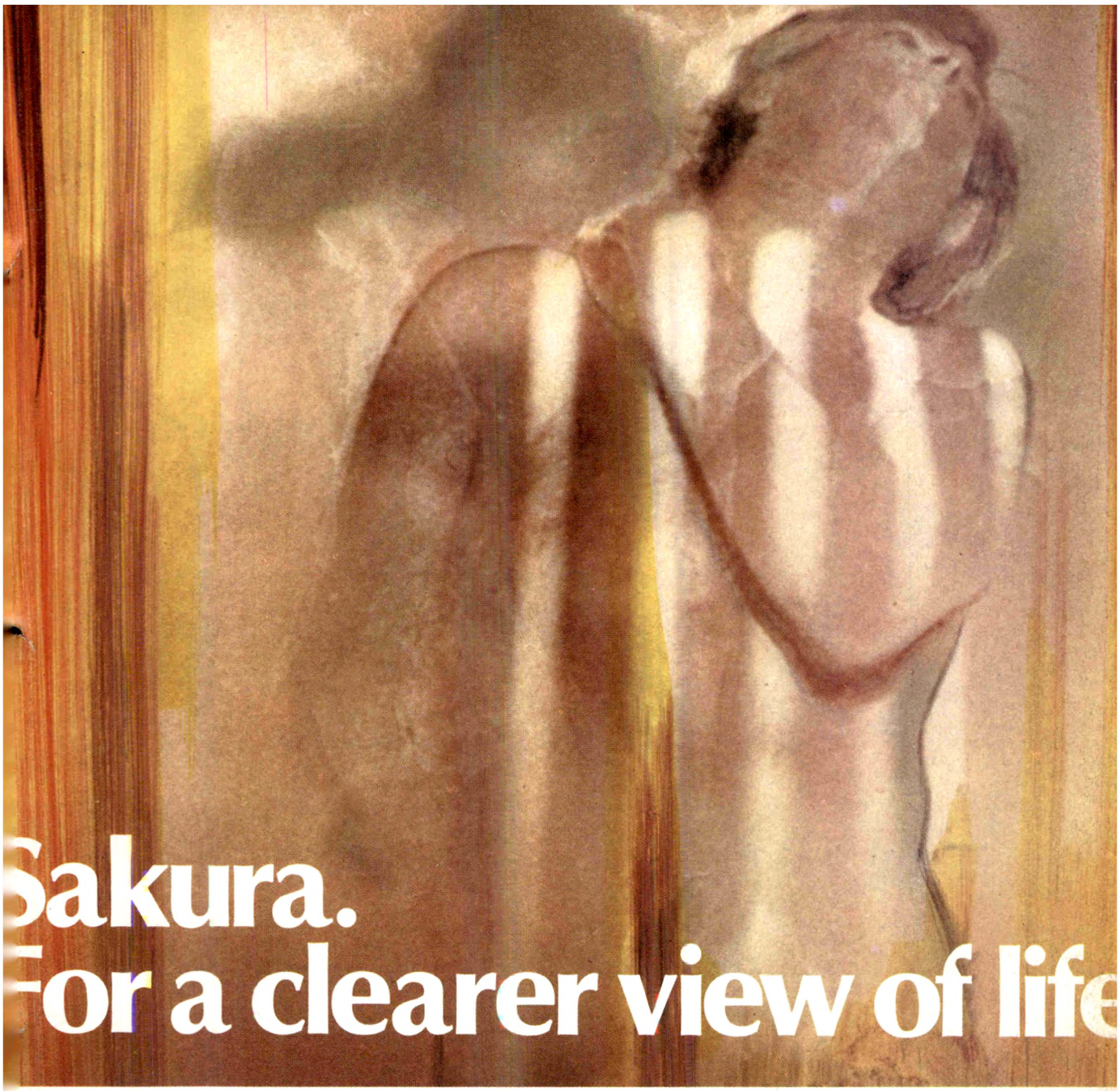
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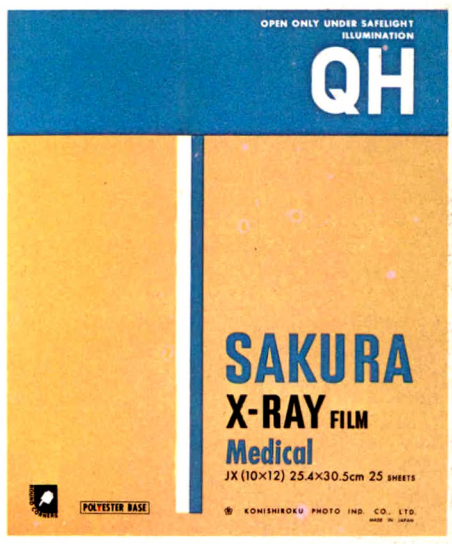
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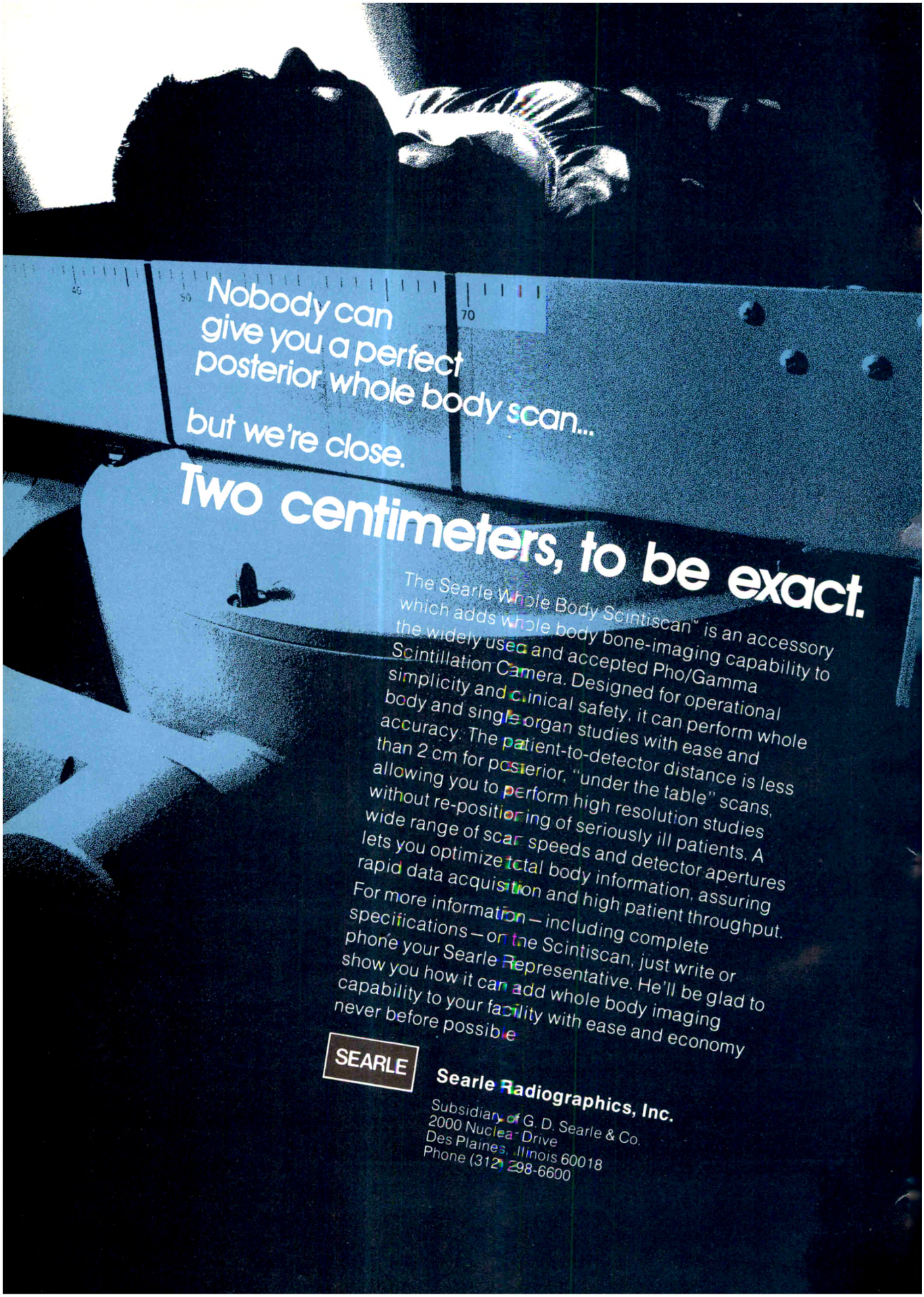
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THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 122 DECEMBER, 1974 No. 4

CHANGING IMAGES*

THE PRESIDENT'S ADDRESS, 1974

By COLIN B. HOLMAN, M.D.
ROCHESTER, MINNESOTA

"NOTHING endures but change." You have heard this piece of wisdom, which is attributed to Heraclitus, quoted many times. This truism implies that at least one of the qualifications for continuing success should be the ability to change. Throughout its first 75 years, the American Roentgen Ray Society has changed in an orderly and evolutionary manner to best serve the needs of patient care and scientific advancement through organized radiology. It should continue to alter its functions and composition in ways that are appropriate to its various educational, technical, and administrative responsibilities. This is one aspect of *changing images* in radiology.

The second interpretation of the title of this address concerns the concept of images in the technical sense, and it also relates to the need to consider changes in the composition of our membership. Changes in image form will alter to some degree the practice, but not the responsibility, of the radiologist. It is not too early to consider new technologies of imaging carefully and to incorporate them where appropriate into the armamentarium of the radiologist.

CHANGES IN THE ANNUAL MEETING

First, let us consider the changes relating to our meeting this year. Needed alterations in the format of the annual meeting have been introduced by the Executive Council after consideration of the thoughtful and helpful report of an *ad hoc* committee, chaired by Dr. James Martin.

Perhaps the most radical departure from our traditional policies has been the introduction of a registration fee. This was instituted with the greatest reluctance, but the financial stability of the Society is threatened by two major factors. The first is familiar to you all: the creeping and now the galloping inflation running roughshod over a shocked world, with little evidence of control. Except for the costs related to the publication of our superior journal and those previously allowed for education and research activities, the income of our Society is essentially devoted in one way or another to providing an annual scientific

* Presidential Address at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24, 1974.

From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

meeting that will continue to be worth the while of all participants; that is, members and guests, including instructors and exhibitors.

The expenses of our meetings have increased about 80 per cent in the past 10 years. This is commensurate with expenses in general but, because no registration charge has been made heretofore, the entire burden has been assumed by the Society.

The second important factor related to this change is the interpretation of the federal tax laws by the Internal Revenue Service regarding the income of scientific societies that is derived from related advertising within their journals. This has resulted in an absolute reduction of income to the Society.

It is apparent that registration fees, while not popular, are common to most national society meetings. The American Roentgen Ray Society has held off as long as possible, but the inevitable has become a reality. The Society has always had the highest regard for the registrants at its national annual meeting and now expresses again its appreciation for such continued interest and support.

Some minor changes in the structure of the meeting are being tried in an effort to conform to the changing priorities of the registrants.

This year, as was done last year, the Caldwell Lecture is to be held at 11:30 A.M. on Wednesday, the second day of the meeting. This change accommodates a great many who are unable to be present in the evening.

The formal banquet has been replaced by a Presidential reception, to which all registrants and their wives who have registered are cordially invited. This reception will be held from 6:30 until 8:00 P.M. on Thursday evening, and it is hoped that it will provide all of you a pleasant opportunity to meet with your old and new friends and acquaintances.

Another change involves the schedule of scientific presentations on Friday, the last day of the meeting. Because some have found it difficult or impossible to attend the instruction courses held at the end of the meeting, these courses will now be held in the latter part of the morning. Therefore, in order to accommodate a full schedule of scientific presentations, two sections of diagnostic papers will be presented during the first half of the morning. The sessions will be in adjacent facilities so that one may pass from one to the other between presentations. If any of these changes do not meet with general approval, they probably will not be continued. We welcome your comments and suggestions.

CHANGES IN SOCIETY MEMBERSHIP

Another change contemplated by the Executive Council concerns the composition of the membership of our Society. To study this and to make recommendations, an *ad hoc* committee under the chairmanship of Dr. Eugene Klatte was formed. For most of the past 75 years, all active members have been certificants of the American or Canadian Board of Radiology and have been exclusively devoted professionally to radiology. This was appropriate because, when ionizing radiations were first used for medical purposes, there were few if any constraints on these activities, for possible untoward effects were little known and the medical uses were not well defined or developed.

The changing composition of the membership of the Society since its organization as the Roentgen Ray Society of the United States is of some historical interest. The first annual meeting was held at the Grand Central Palace in New York City on Dec. 13 and 14, 1900. At that time, Dr. Heber Robarts of St. Louis, who was elected President, stated that "we need the assistance of physicists in our meetings;" the Society then was not limited to physicians. In 1911, admission standards were altered and applicants were required to have a medical degree and 2 years of x-ray work following graduation together with three letters of recommendation, two from members in good standing of the American Roentgen Ray Society. The category of

associate membership was developed in 1914 at the meeting in Cleveland, Ohio.

Throughout the intervening years, however, much of diagnostic and therapeutic value—and, indeed, most of the scientific and technical developments—has been attributable to physicists, engineers, physicians, and other professionals who have devoted most or all of their efforts to these activities. It seems, therefore, most appropriate that such individuals be welcomed into our Society as active members, and their acceptance of such an invitation would enhance the image of our Society.

The expanding fields of radiology require the expertise of such scientists because our imaging methods now involve a number of new techniques, including those relying on nonionizing and on ionizing radiations. This has been reflected by the fact that many state and federal government agencies which are concerned with the safety of use of ionizing radiations have extended their regulations to cover the use of nonionizing radiations. Indeed, we are now concerned with the whole range of the energy spectrum with regard to its use in acquiring data that can be expressed so as to evoke a visual image capable of interpretation, in terms familiar to radiologists. We may only refer, for example, to ultrasound, laser acoustic holography, computerized axial tomography, liquid-crystal thermography, videodensitometry, and three-dimensional reconstruction, to mention a few. The changing of a photographic emulsion and the activation of the crystals on a screen are not the only useful diagnostic end products of the application of radiant energy to our patients. It is fortunate that our patron "saint," Professor of Physics William K. Roentgen—in whose honor our specialty and, therefore, our Society is named—exposed a piece of cardboard coated with barium platinocyanide to his unknown ray and obtained an image instead of activating a crystal linked to a computer. Surely he must have had the scientific credentials for membership.

The impact of new imaging techniques is felt not only in the abstract scientific sphere or in the fields of clinical usefulness but also in the mundane, commercial aspects of our occupations. You are all becoming more and more aware of the frightening escalation of costs associated with the acquisition of ever more sophisticated apparatus. We all want the most useful, effective, safe, and reliable equipment that is available, but few of us who limit our expertise to clinical diagnostic or therapeutic radiology are able to understand all of the technical considerations involved in choosing, installing, and using much of this exciting new apparatus. We need and welcome the advice of our associated physicists to help us evaluate, choose, and use these super-sophisticated and expensive devices. This cooperation will necessarily involve mutual educational efforts by clinical radiologists and physicists or engineers. The professional scientists must be familiar with the clinical problems and needs.

Closer professional ties between radiologists and other scientists who devote their professional lives largely to problems related to radiology might encourage and support efforts by societies such as ours to develop advisory technical groups or committees. These might provide information and technical facts and so be able to make sound objective recommendations as to the true or potential value of available equipment or that in various stages of development. That such cooperation might be practical is apparent when one realizes that the American Association of Physicists in Medicine already has several subcommittees that have acquired a wealth of information regarding the technical aspects of diagnostic radiologic techniques.

Our association with these interested scientists is most evident in their teaching of our residents and their extensive cooperation throughout the years in our Society's instructional courses. We will depend even more on them in the future if we are to understand and use the many new imaging techniques for the benefit of our patients. Our expanding educational efforts already include some of the newer technical ad-

vances. It is known that ultrasound will be included as a subject in the American Board of Radiology examinations in 1980. Who knows how many other new developments will be included soon thereafter?

In the press of the everyday practice of radiology, it is difficult to appreciate the enormous practical impact of the new developments in the physical sciences. For such an understanding, I strongly recommend to each of you a most interesting paper entitled "New Directions in Physics" by Dr. D. Allan Bromley, the Henry Ford II Professor of Physics and Chairman of the Physics Department at Yale University.[1] My time does not permit a review, but he calls attention, in terms that we can understand, to a host of new directions: newer developments in nuclear physics; atomic, molecular, and electronic physics; condensed-matter physics that concerns the solid-state devices that are so important for our storage and retrieval information; optics that concern our signal-to-noise ratios; the unfolding field of holography; acoustical developments, including holographic applications; and other exciting new ideas that are likely to become clinically important in medical imaging technology.

CHANGING REQUIREMENTS FOR RADIOLOGISTS

Another suggestion for change relates to the interval presently required before a young radiologist may be invited to join with us. Again, referring to the past, during the time when there were no certifying boards, no academic status, and little formal instruction in radiology, it was reasonable to require evidence of serious commitment to the young specialty before one could participate as a member in this type of a scientific society—hence the requirement of 8 years devoted to training and practice and the submission of a suitable thesis.

[1] Bromley, D. A. New directions in physics. *Am. Sc.* 1974, *62*, 293–303.

It is now apparent that, by the time a young radiologist has been certified by the American or Canadian Board of Radiology, he has demonstrated commitment to and competence in his chosen specialty and that his association with us will be mutually beneficial. The 8 year requirement, therefore, is an anachronism, and this has been recognized by the Executive Council and those who have framed the new bylaws for our constitution.

The requirements for the thesis have gradually been relaxed in recent years because it is well recognized that most scientific projects today represent the combined efforts, ideas, and skills of more than one author. Therefore, it is the merit of the project that must be recognized. The candidate's participation in the research documents his dedication to radiology and indicates his promise of excellence in scientific, clinical, or teaching work. This great Society could well benefit by welcoming more young, well-qualified, industrious, and intelligent radiologists as members. Many of the authors of the scientific papers in our journal are dedicated radiologists who are finishing or have just recently finished training programs. I hope not only that many would become active members for mutual scientific benefits but also that after affiliation they would interest themselves in, and contribute to, the many organizational aspects of our Society.

CHANGES ON THE NATIONAL LEVEL

The changing image of radiology with respect to research efforts involving the older, as well as the newer, technologies presents our Society with an opportunity and, I believe, an obligation to support and assist efforts of other prestigious radiologic organizations in attempting to secure for radiology categorical support on a national level. It has been pointed out that clinical radiology is rather unique in not being specifically represented in programs of the National Institutes of Health (NIH) but is designated as a part of biomedical engineering. This is a curious situation

and obviously not in the public interest as far as the development through research of probably the largest and most important field of medical diagnosis is concerned. Surely, if our national leaders are serious in their commitment of public funds to the future health problems of our people—and I am sure they are—it is only reasonable and necessary to recognize that future research in radiology must have institutional input and support, similar to that provided for cancer, psychiatry, nervous diseases, pediatrics, and so forth. A National Institute of Radiological Sciences included within the NIH would be a most appropriate organization and could provide the needed advocacy and part of the sustenance for vigorous, coordinated research efforts in diagnostic radiology, nuclear medicine, ultrasound, holography, thermography, radiation physics, radiation protection, radiation biology, and other related technologies involving ionizing and nonionizing radiations for medical purposes.

The philosophy of categorical financial support has recently been examined to some extent in a thoughtful editorial by Dr. Irvine H. Page.[2] He countered a suggestion that NIH discontinue the categorical approach in favor of more centralized control by the director of NIH, by noting that "Strong institutional leadership of NIH, with the aid of the director of the categorical institutes is a first step" in the public interest to promote worthwhile objectives without " . . . undue and often unwise pressure from lay groups and professionals with too strong biases." He said further that " . . . securing of ties with the important scientific societies is a second step." It is here that our Society can aid in the professional support of a much needed National Institute of Radiologic Sciences, standing on its own to provide our population with the benefits of carefully planned research in radiology.

[2] Page, I. H. Financing research by disease categories (commentary). *J.A.M.A.*, 1974, *228*, 995–996.

Conclusions

It therefore seems clear that, just as imaging techniques are improving and changing, so too the images of the specialty of medical radiology and those of scientific societies such as ours also are changing to keep in step with their scientific and technical responsibilities as well as with their clinical and educational responsibilities. Although we do not know at this time whether the suggested changes in our Constitution and Bylaws concerning membership requirements will meet with the approval of the majority of the membership, the opportunity to consider, debate, and evaluate these proposals, the changes that have already been implemented in the format of this annual meeting, and, most significantly, the continued superiority of our official journal through a dedicated editorial staff serve to verify the vitality of this venerable organization on its diamond anniversary.

Mayo Clinic and Mayo Foundation
Rochester, Minnesota 55901

HYDATID DISEASE OF THE LUNGS*

A ROENTGENOLOGIC STUDY OF 50 CASES

By JIRAYR P. BALIKIAN, M.D.,† *and* FAYSAL F. MUDARRIS, M.D.‡
BEIRUT, LEBANON

PULMONARY hydatid disease presents a wide range of roentgen manifestations. Some of the appearances of the hydatid cysts are characteristic of the disease, while others present a challenging roentgen diagnostic problem.

Hydatid disease is a parasitic infection caused by the larval stage of the tapeworm genus *Echinococcus*. There are 2 main forms of the disease: the cystic unilocular form; and the alveolar multilocular form. The former has 2 varieties: the common, classic, European or pastoral variety, which is caused by *E. granulosus*; and the less common Canadian-Alaskan or sylvanic variety, which is said to be more benign and is probably caused by a different strain or variety of *E. granulosus*.[31] The alveolar hydatid disease is caused by *E. multilocularis* and is characterized by the formation of multilocular vesicles which infiltrate the surrounding structures and has a graver prognosis.[4,11,31]

The disease is widespread in many temperate and subtropical areas of the world and is not infrequently encountered in colder climates such as Iceland, U.S.A., Canada and Great Britain.[4,5,8,10,14,15,20,30] Reports of the various roentgen findings of pulmonary hydatidosis have been scanty and fragmentary in the English language literature.[8,18,23,25,26,30] The preoperative diagnosis depends mainly on the findings of the chest roentgenograms and serologic results.

The purpose of this report is to present a detailed analysis of the various roentgen manifestations of 50 cases of the classic form of hydatid disease of the lungs encountered in the last 10 years at the American University Medical Center in Beirut. All the cases reported herein were proven at operation and at histologic study.

GENERAL CONSIDERATIONS

A knowledge of the life cycle of the parasite and the morphology of the hydatid cyst is essential for the understanding of the protean roentgen manifestations of hydatid disease of the lungs.

LIFE CYCLE OF *E. granulosus*

The minute adult tapeworm, *Echinococcus granulosus*, lives in the intestine of the canine animals, specially dogs; millions of ova are passed on to pastures where sheep and cattle may be grazing. When these animals, and occasionally men, ingest the ova, the digestive juices dissolve the egg's external coat, the free hexacanth embryo migrates through the duodenal mucosa and enters the venules and lymphatics. Most of it is filtered by the liver, some by the lungs and a few may reach distant organs. If the embryo is not destroyed by the lungs, it is transformed into a tiny cyst.[11,25] When the fertile cyst or its contents are ingested by the dog, the life cycle is renewed. Infection could also possibly be acquired by inhalation of ova.[30]

MORPHOLOGY OF THE HYDATID CYST

As the hydatid cyst grows in size, the lung reacts by forming a connective tissue layer, the *pericyst*, a layer embracing the parasite which is called the *endocyst*.[23] The wall of the parasite itself consists of a thicker laminated chitinous outer layer, the *ectocyst*, and a delicate inner layer, the germinal membrane also called the *endo-*

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cyst. The germinal membrane gives rise to the hydatid fluid, brood capsules, scolices and daughter cysts.[11,18,23]

AGE AND SEX DISTRIBUTION

Hydatid disease affecting all organs has been seen between the ages of 2 and 93 years and more commonly in males.[5,9,18,28] The youngest patient in our series was 5 years old and the oldest 62. The incidence was 10 per cent in the pediatric age group, 10 per cent above the age of 50 and approximately 20 per cent in each of the second, third, fourth and fifth decades. Sixty per cent of the patients were males.

SYMPTOMS AND OTHER LABORATORY RESULTS

The majority of the intact pulmonary cysts are known to produce no symptoms or are occasionally responsible for a nonproductive cough or minimal hemoptysis. However, when the cyst ruptures, there is an abrupt onset of cough, expectoration and fever and an acute hypersensitivity reaction may develop. The sputum may become purulent and may contain fragments of hydatid membrane; rarely, microscopic examination of the sputum reveals hooklets from scolices. Other symptoms may arise such as Horner's syndrome from superior sulcus cysts, bone pain from posterior mediastinal cysts, dyspnea from tracheal compression, hemorrhage due to erosion of great vessels and tension pneumothorax.[1,11]

The serologic studies in hydatid disease include Weinberg complement fixation test, the indirect hemagglutination test, and the Casoni intradermal test.[12,23]

The analysis of our series showed the following results: 60 per cent gave a history of hemoptysis; 20 per cent a history of coughing out membranes and salty fluid; and 10 per cent showed scolices or hooklets at sputum microscopy. Eighty per cent had positive serology to 1, 2 or all 3 tests (the 20 per cent false negatives were commoner in the unruptured lung cysts with a ratio of 4:1). Eosinophilia above 5 per cent was present in 40 per cent of patients.

Positive serology to any or all 3 tests is said to be lower in children,[27] and as high as 90 per cent in adults (to all organs in body).[12] The Weinberg test becomes negative within 6 months and the indirect hemagglutination test may take more than 2 years to become negative after removal of the cyst if no residual cysts are present.[17]

ROENTGEN CONSIDERATIONS

NUMBER OF HYDATID CYSTS: SOLITARY AND MULTIPLE

Hydatid cysts may be solitary or multiple, the number depending mainly on the amount of ova ingested and the number of embryos filtered through the liver and lungs.[11,23] Multiple hydatids are reported up to 30 per cent.[18] Our series showed 60 per cent solitary (Fig. 1, *A–D*), and 40 per cent multiple hydatids, of which 24 per cent were with 2–5 hydatids (Fig. 2; and 5), 4 per cent with 6–10 hydatids (Fig. 5*B*) and 8 per cent with 11–30 hydatids (Fig. 5*E*).

SIZE AND RATE OF GROWTH: COIN-SIZED LESIONS, AND GIANT HYDATID CYSTS

It is known that a hydatid cyst usually reaches 1–2 cm. in diameter at the end of 6 months,[5] and up to 6 cm. in diameter within a year.[2,5] The doubling time ranges from 16 to 20 weeks.[3] Some cysts in the same patient can grow faster, while others remain inactive.[3] The sizes in our series varied between 1 to 20 cm. in diameter. Four patients showed numerous hydatids, 1 to 4 cm. in diameter, totaling 70 cysts (Fig. 5*E*). The remaining 46 patients showed a total of 80 cysts with the following distribution: 1–5 cm. (45 per cent); 6–10 cm. (41 per cent); 11–15 cm. (10 per cent); 16–20 cm. (4 per cent) (Fig. 1). We have observed different rates of growth of 2 cysts in the same patient, both still active. One of the cysts grew from 1 cm. to 8 cm. in diameter and the other from 2.5 cm. to 5 cm. in diameter during a 7 month period,

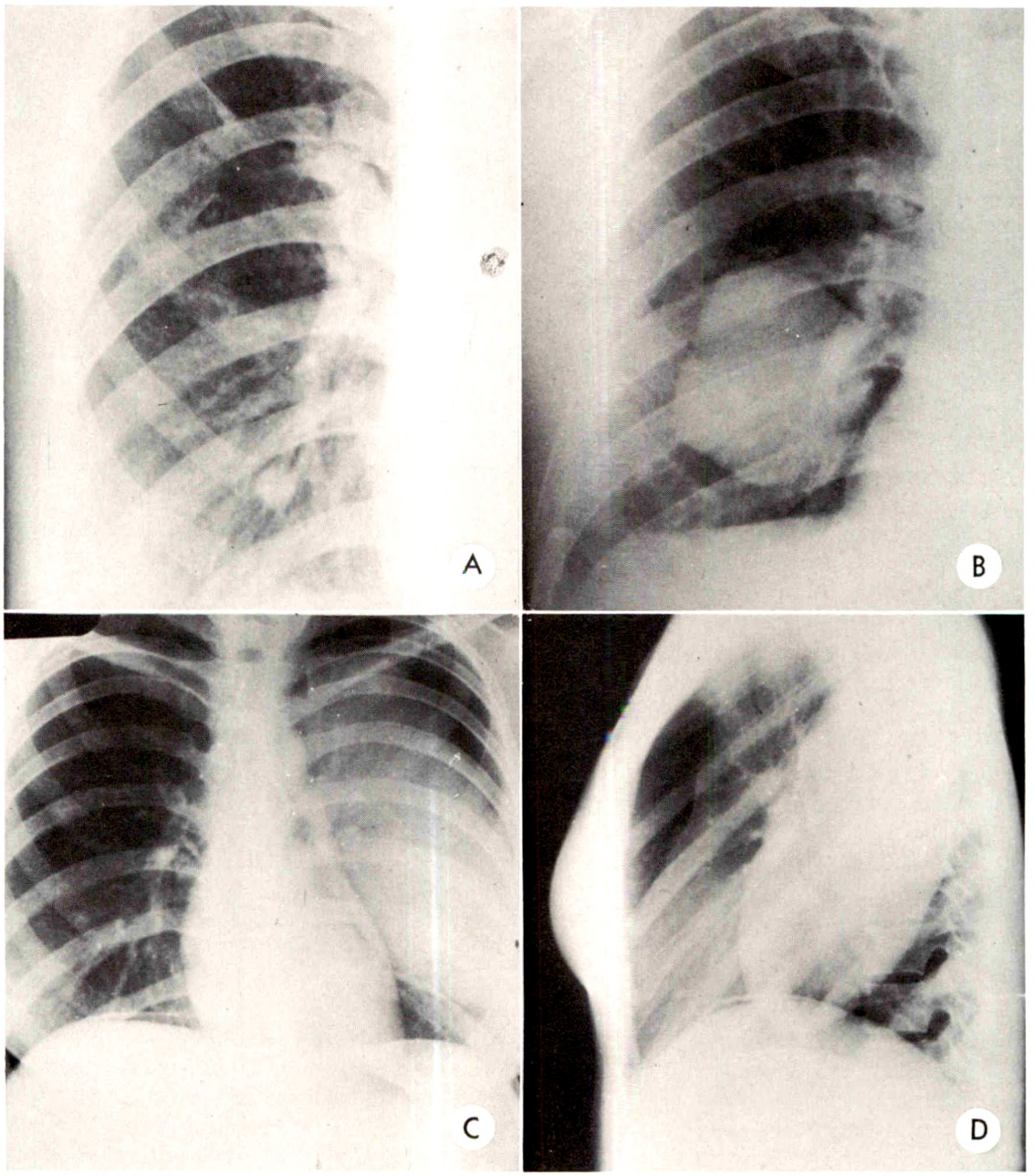


FIG. 1. *Hydatid Coin Lesion, Average-sized and Giant Hydatid Cysts.* (*A*) Roentgenogram showing a small hydatid "coin lesion" which is round and sharply circumscribed. (*B*) Common average-sized simple solitary well circumscribed polycyclic hydatid cyst. (*C*) Simple (uninfected, unruptured) giant solitary oval-shaped interlobar hydatid cyst with some loss of cystic image and (*D*) the same in lateral projection simulating loculated interlobar effusion. (*C* and *D*) Note air under the diaphragm following operation for acute appendicitis.

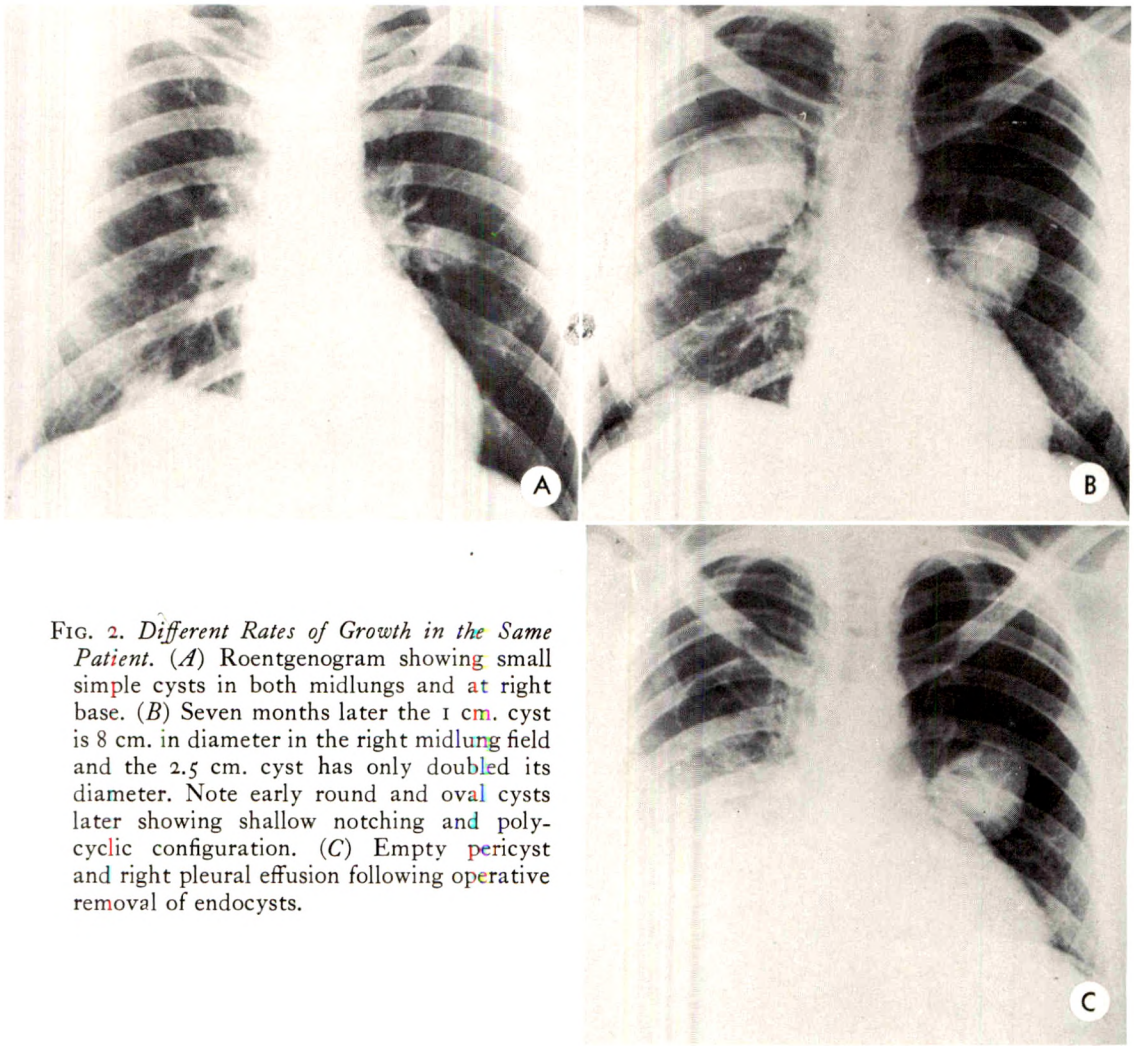


Fig. 2. *Different Rates of Growth in the Same Patient.* (*A*) Roentgenogram showing small simple cysts in both midlungs and at right base. (*B*) Seven months later the 1 cm. cyst is 8 cm. in diameter in the right midlung field and the 2.5 cm. cyst has only doubled its diameter. Note early round and oval cysts later showing shallow notching and polycyclic configuration. (*C*) Empty pericyst and right pleural effusion following operative removal of endocysts.

the fastest rate of growth that we encountered (Fig. 2).

SHAPE OF HYDATID CYSTS

A centrally located cyst is said to be usually round, but may become oval or polycyclic to conform to pressures from bronchus, mediastinum, pleura and adjacent cysts.[3,5,18] The cysts may change shape on maximum inspiration and expiration, which is known as the Escudero-Nimerov sign, but which is true also of any thin-walled water filled cyst.[18,24,26,29]

The commonest shape noted in our series was that of a bizarre polycyclic form accounting for 40 per cent of cysts (Fig. 1*B*) and including those showing shallow notching (5 cysts) (Fig. 2*B*), deep notching that gave a bilocular appearance simulating 2 cysts (7 cysts) (Fig. 3, *A* and *B*), and bud-like projections (4 cysts) (Fig. 3*C*). Thirty per cent were roughly oval (Fig. 1*D*; and Fig. 3*D*), 20 per cent round (Fig. 1*A*) and 10 per cent presented as ill-defined nonresolving pneumonia. (Fig. 7, *B* and *C*)

LOCATION OF THE CYSTS

The commonest location of the 80 cysts was the right lower lobe, accounting for 35 per cent. The remainder were distributed as follows: 10 per cent were in the right upper lung, 15 per cent in the right mid lung, 22 per cent in the left lower lung and 18 per cent in the left upper lung. About 65 per

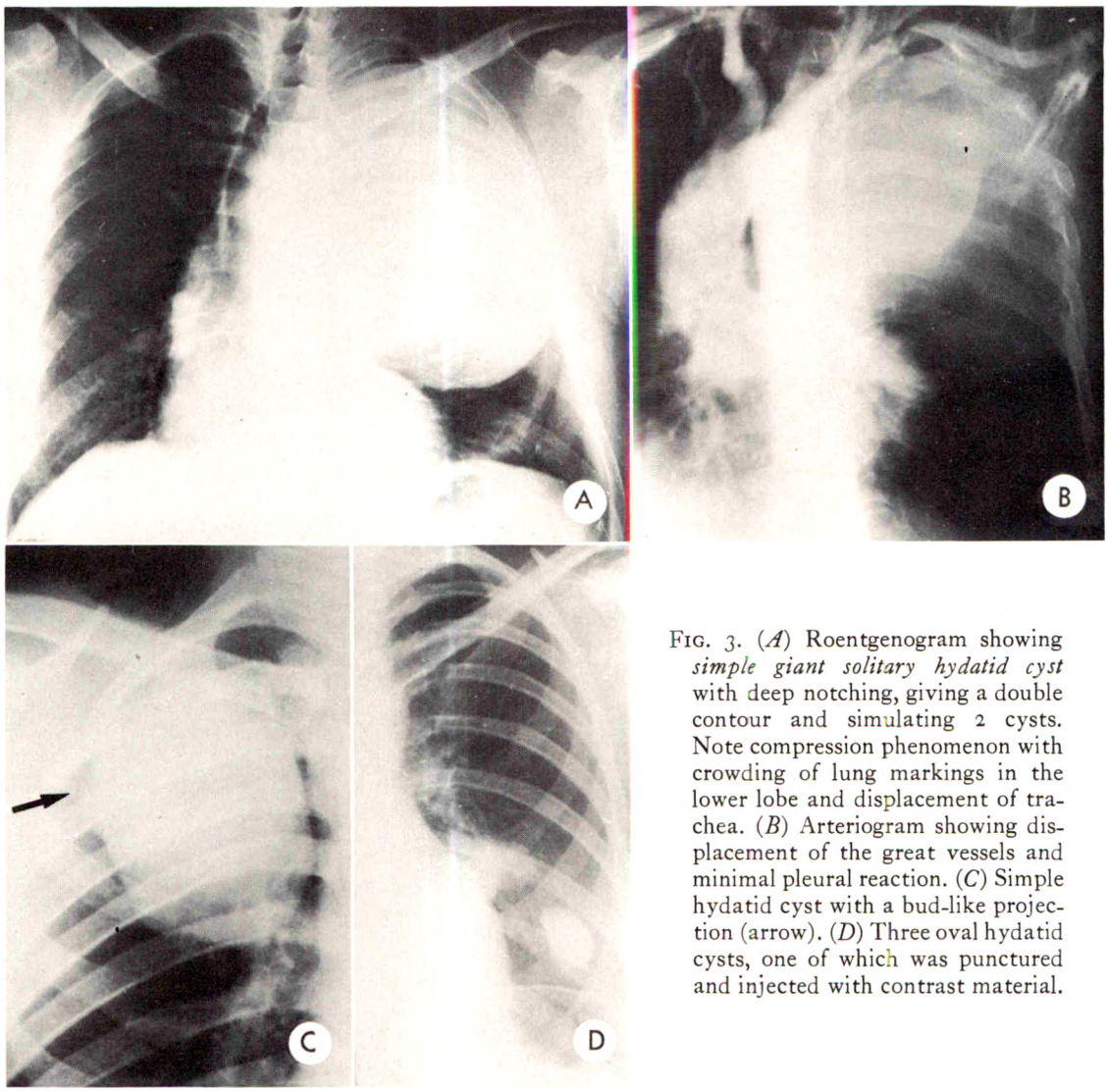


FIG. 3. (*A*) Roentgenogram showing *simple giant solitary hydatid cyst* with deep notching, giving a double contour and simulating 2 cysts. Note compression phenomenon with crowding of lung markings in the lower lobe and displacement of trachea. (*B*) Arteriogram showing displacement of the great vessels and minimal pleural reaction. (*C*) Simple hydatid cyst with a bud-like projection (arrow). (*D*) Three oval hydatid cysts, one of which was punctured and injected with contrast material.

cent were in the posterior segments. Bilateral involvement occurred in 20 per cent of patients. These findings are in agreement with most of those reported in the literature.[5,6,18,25]

Shifts of surrounding structures are said to be rare.[3] We had 5 patients with large hydatids that depressed the diaphragm (Fig. 4), and 4 that shifted the mediastinal structures (Fig. 3, *A* and *B*). Three cysts were interlobar in location (Fig. 1, *C* and *D*), and 5 subpleural in location (Fig. 13*C*).

"SIMPLE" HYDATID CYST

The viable, intact or closed cysts are termed "simple" cysts and the ruptured or infected cysts are considered "complicated" cysts. The simple cysts are commonly sharply demarcated, homogeneously dense and simulate primary sarcomas of the lungs, benign tumors, granulomas, solid lung cysts and metastases (Fig. 1; and 2), whereas the larger and older simple cysts, because of further localized atelectatic and reactive changes in the surrounding parenchyma, lose their sharp demarcation and simulate pneumonia and carcinoma[23,26] (Fig. 3*B*). Infarction, hemorrhage and more commonly infection can also cause loss of cystic image.[2,24] At

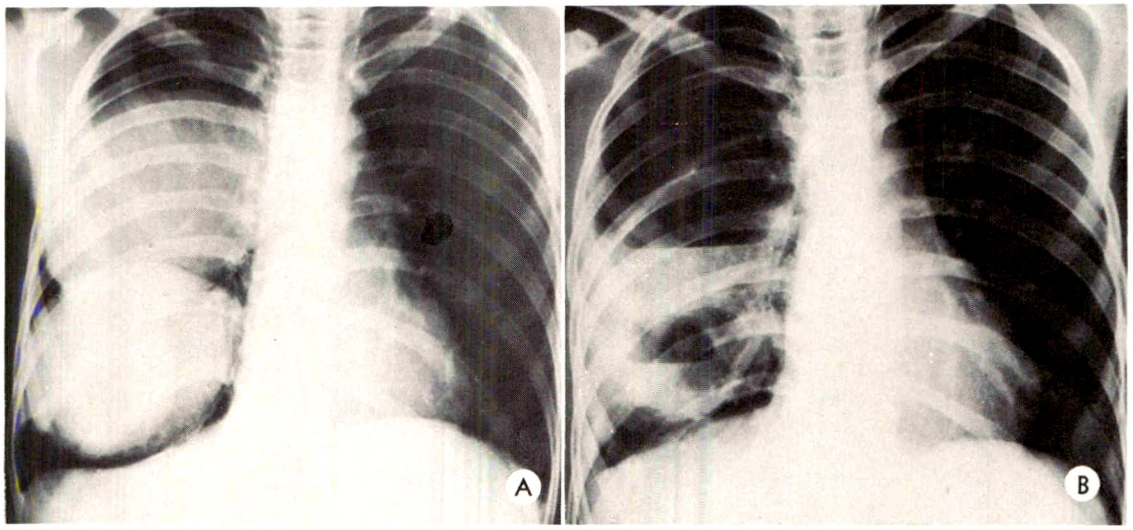


FIG. 4. (*A*) Roentgenogram showing *multiple large cysts* depressing and slightly inverting the diaphragm. (*B*) Pericyst effusion following operative removal of endocysts.

this stage, other roentgen criteria for diagnosis include the presence of multiple large masses which are virtually pathognomonic of hydatid cysts[26] (Fig. 5*A*); other hazy or ruptured cysts in the same patient (Fig. 5, *B–D*); characteristic dense rim shadows (Fig. 6) and calcified cysts in the liver, spleen or kidneys; multiple large defects on the liver scan or one large defect with little, or more commonly, no diaphragmatic pleural reaction (*vs.* amebic liver abscess). Needle puncture should be condemned because of the dangers of allergic reaction and implants, although in our experience with one case (only case), no untoward reactions resulted (Fig. 3*D*).

The simple closed cysts comprised 70 per cent of our cases of which 10 per cent were viable closed cysts with some loss of cystic image (Fig. 3, *A* and *B*).

ALLERGIC AND BACTERIAL PERI-PERICYST PNEUMONITIS AND ASPIRATION BRONCHOPNEUMONIA

Early in the process of rupture of the endocyst, as hydatid fluid pours out into the bronchus, it may spread into the area surrounding the pericyst simulating consolidation.[18] An allergic pneumonitis with eosinophilic reaction may develop which is reversible in up to 10 days.[25] At this stage, sometimes the patient may go into anaphylactic shock. The allergic reaction is explained on the basis of sensitization by small leaks of hydatid fluid into the surrounding tissues. After 10–14 days, secondary bacterial pneumonitis may develop[25] and be perpetuated by the adjacent infected cyst. There may also be transbronchial spread of the fluid producing aspiration bronchopneumonia.

We have noted 4 patients with acute transient peripneumonitis (Fig. 7*A*), 3 patients with distant aspiration bronchopneumonia (Fig. 7*A*), and 25 patients with chronic bacterial peripneumonitis giving a fuzzy and irregular outline to the cyst (Fig. 7, *B–D*).

CRESCENT SIGN OR PULMONARY MENISCUS SIGN

This sign is produced when an enlarging cyst eventually erodes the bronchioles and with coughing and straining air may be introduced between the pericyst and the endocyst producing a radiolucent air shadow in the form of a crescent or miniscus.[26] This is a sign of detachment and signifies impending rupture and death of the parasite. Clinically the individual may have a cough and slight hemoptysis at this stage. The sign is highly reliable, but not patho-

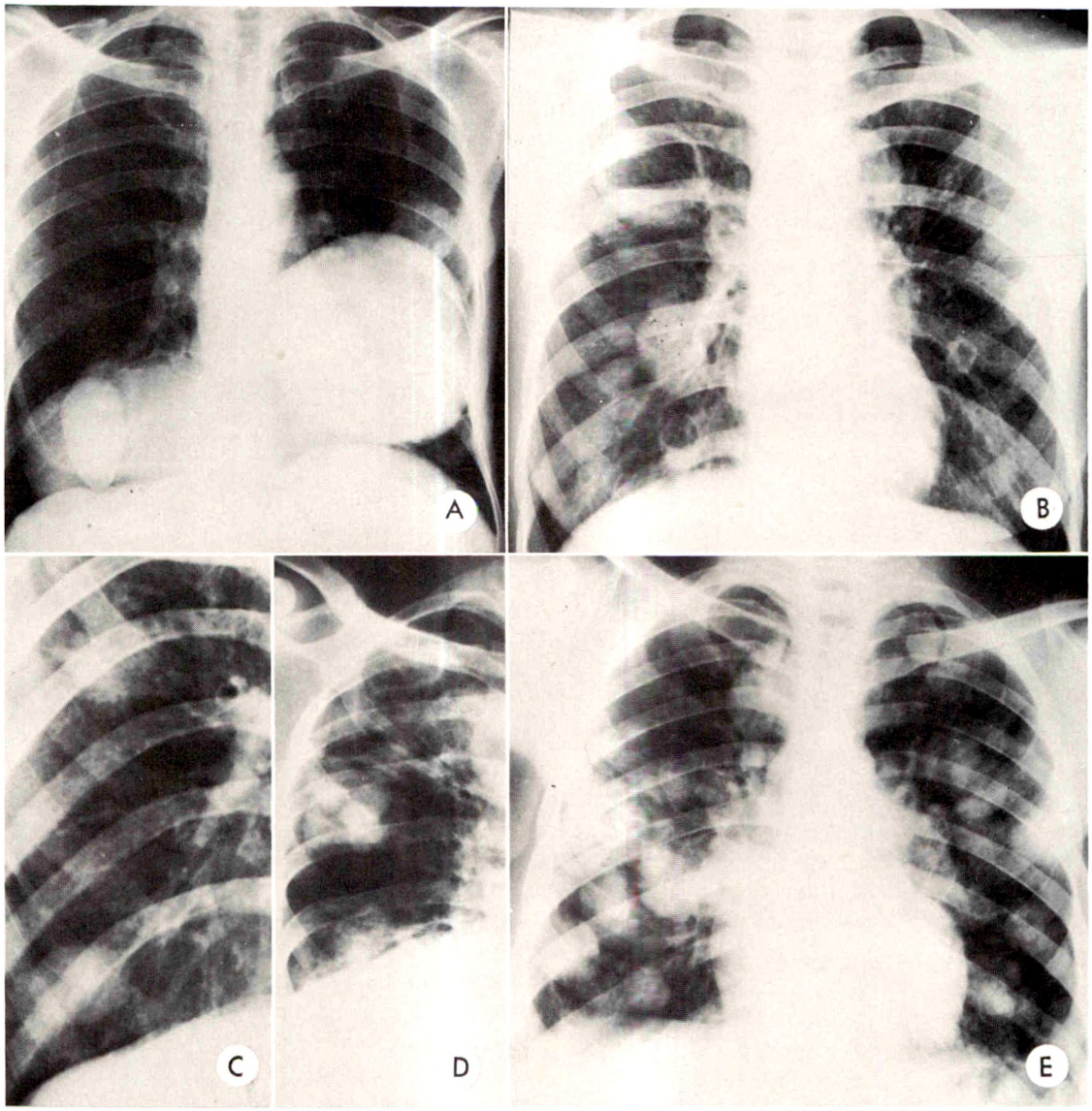


FIG. 5. *Multiple Hydatid Cysts.* (*A*) Roentgenogram showing 3 large unruptured cysts virtually pathognomonic of hydatid cysts. Note a fourth small ruptured cyst in the left midlung for further evidence of hydatid disease and sunburst changes at lower border of the left lower lobe cyst. (*B*) Multiple cysts, some intact, others showing air-fluid levels and demilune fluid collection. (*C*) One of the infected cysts with surrounding pneumonitis. (*D*) One of the cysts showing empty pericyst in right subclavicular area. (*E*) Multiple cysts simulating metastases. On tomography, few of the cysts showed surrounding pneumonitis.

gnomonic of hydatid cyst.[23] In nonendemic areas, cavities with fungus balls (mycetoma) are the most common cause of the meniscus sign, but blood clots, carcinoma, pulmonary gangrene, tuberculosis and sarcomas may also present with a pulmonary meniscus sign.[7]

We have noted this sign in 8 patients (16 per cent), some with very sharp crescent shadows (Fig. 8, *A* and *B*). It has been less commonly noted in other series.[18,31]

DOUBLE ARCH OR CUMBO'S SIGN

As more air enters between the pericyst and the endocyst, the shrinking cyst ruptures and some of the salty fluid pours

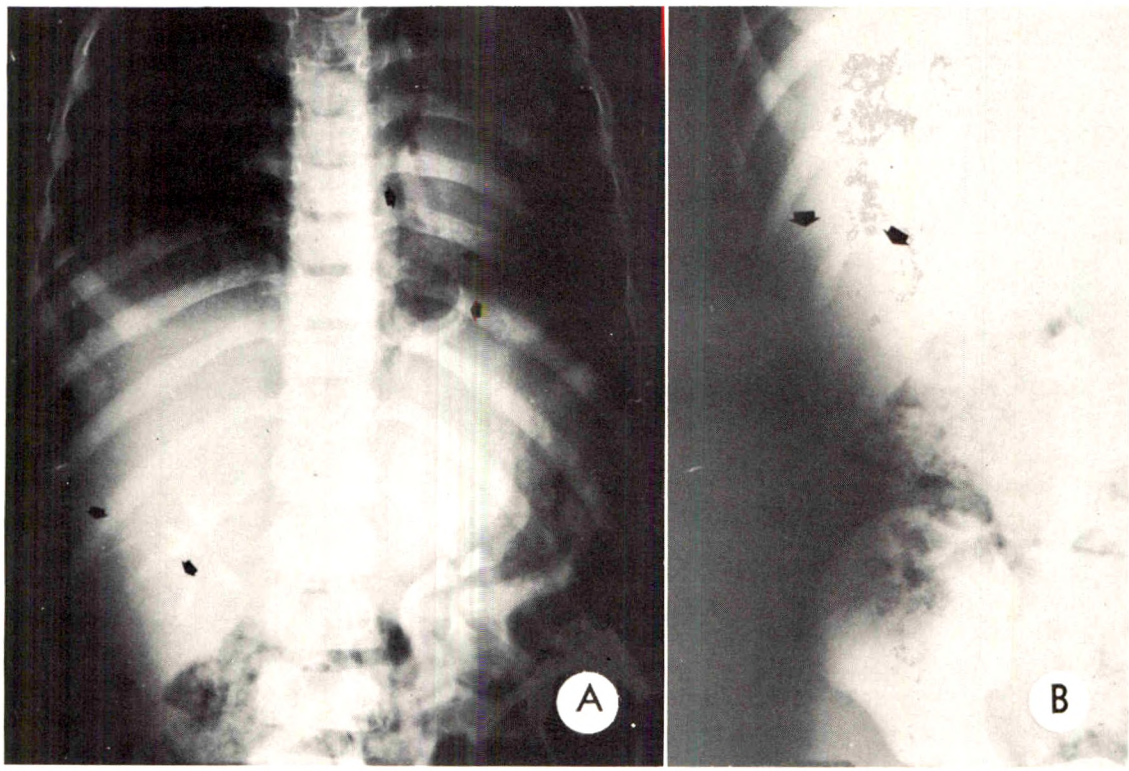


Fig. 6. (*A*) Roentgenogram showing multiple lung cysts, 1 of which shows collapsed pingpong ball type of ruptured endocyst and a liver cyst showing characteristic dense rim shadow (arrows). (*B*) Close-up view of the rim sign of the liver cyst (arrows).

out and air enters the endocyst, giving an air-fluid level within the endocyst which is capped with a crescent of air between the pericyst and the endocyst.[16,18,23,26] Spontaneous rupture occurs in 50 per cent of cases.[6]

We have seen this sign in 4 patients, with pericyst arch and endocyst arch showing fluid levels (Fig. 9*A*); with 2 floating endocyst arches (Fig. 9*B*) and with 2 large endocyst arches (Fig. 9*C*). We consider this sign pathognomonic of hydatid cyst.

WATER LILY SIGN OR CAMALOTE SIGN

This sign occurs when with further collapse of the endocyst and evacuation of the fluid, a wavy endocyst membrane floats on top of the remaining fluid.[18,23,26] The pericyst wall is usually thinner than is seen with other cavities.[18,26] We have seen this sign in 5 lung cysts (10 per cent) (Fig. 10, *A* and *B*).

"MEMBRANE" SIGN

Since the pulmonary meniscus sign and the water lily sign can be produced by diseases other than hydatid cysts,[7] we have chosen to introduce contrast material into these cavities in an attempt to show the incarcerated or the ruptured membranes. At bronchography, the contrast agent enters between the layers of the ruptured hydatid membrane giving a radiolucent undulating line which we like to refer to as the lucent membrane sign. When this sign was present, it was pathognomonic of hydatid cyst (Fig. 11*C*). At tomography, these cysts may show an onion-peel appearance with opaque membranes (Fig. 11*B*). We have noted this sign in 6 cases. In 2 patients, the contrast material failed to enter into the infected cysts which were completely filled with pus and incarcerated membranes (Fig. 7, *C* and *D*).

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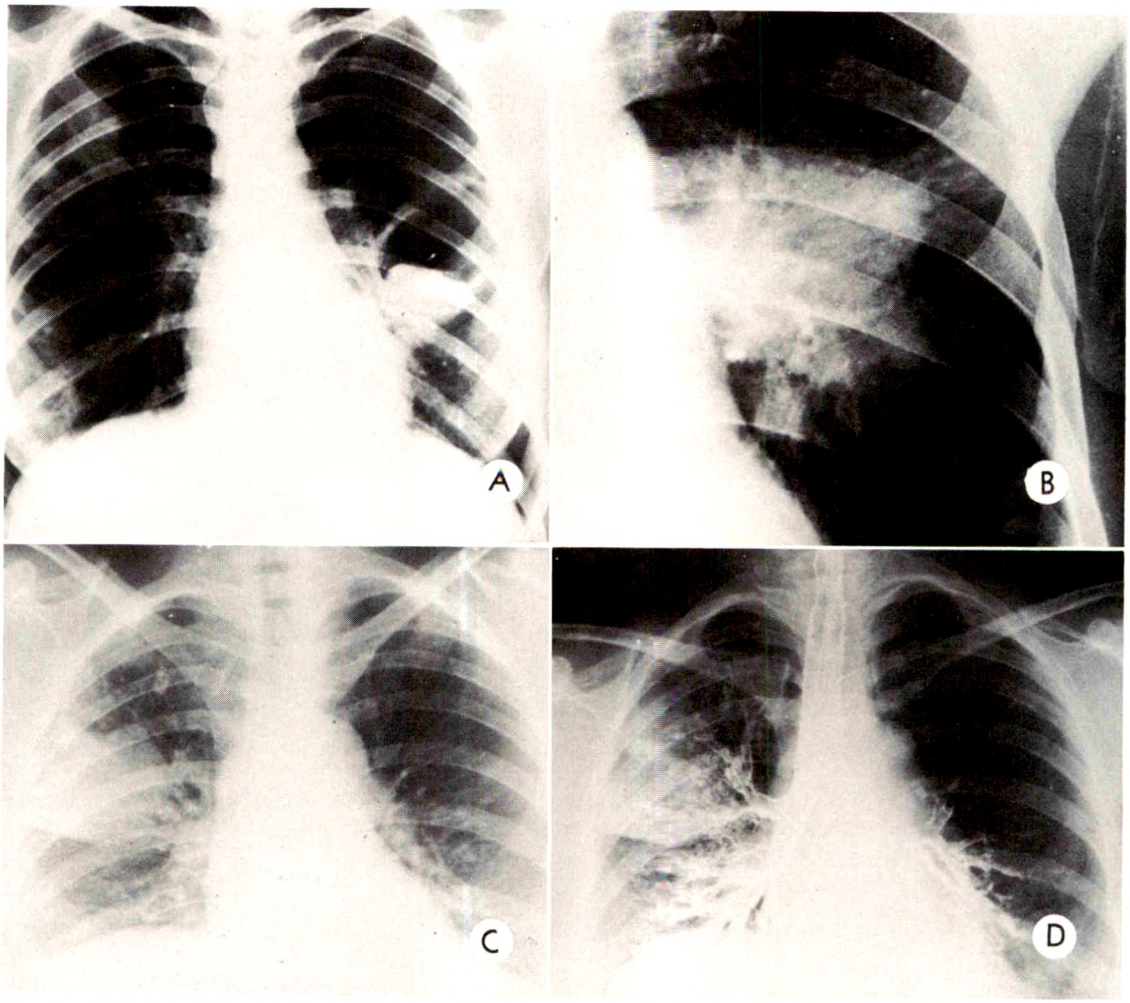


FIG. 7. *Allergic and Bacterial Peri-Pericyst Pneumonitis and Aspiration Bronchopneumonia.* (*A*) Roentgenogram showing ruptured cyst with an allergic peri-pericyst pneumonitis that disappeared within 10 days and transient transbronchial aspiration bronchopneumonia in the right upper and lower lobes. (*B*) Early infected cyst with bacterial peri-pericyst pneumonia showing sunburst appearance simulating carcinoma of the lung. (*C* and *D*) Infected cyst on bronchography showing spreading of bronchi and minimal bronchiectasis.

DAUGHTER CYST SIGN

With the disintegration of the endocyst membrane, the daughter cysts may make their appearance as circular shadows at the bottom of the cyst and among the disintegrated membrane, giving a picture similar to the "rising sun." The daughter cysts develop usually as a result of mechanical, infectious, and toxic disturbances. They are rarely seen in the lungs,[18] but are common with the liver cysts.[23]

We had 9 patients (18 per cent) with pulmonary cysts that showed daughter cysts at operation, but could demonstrate the sign of a daughter cyst in only 2 instances (Fig. 12), and consider it a characteristic sign. Foley catheters passed into the pericyst cavity following operation should not be mistaken for daughter cysts. We could not find an illustration of a daughter cyst of the lung in the literature.

HYDATID LUNG ABSCESS

With uncontrolled bacterial infection and further disintegration of the membrane, the purulent material may produce

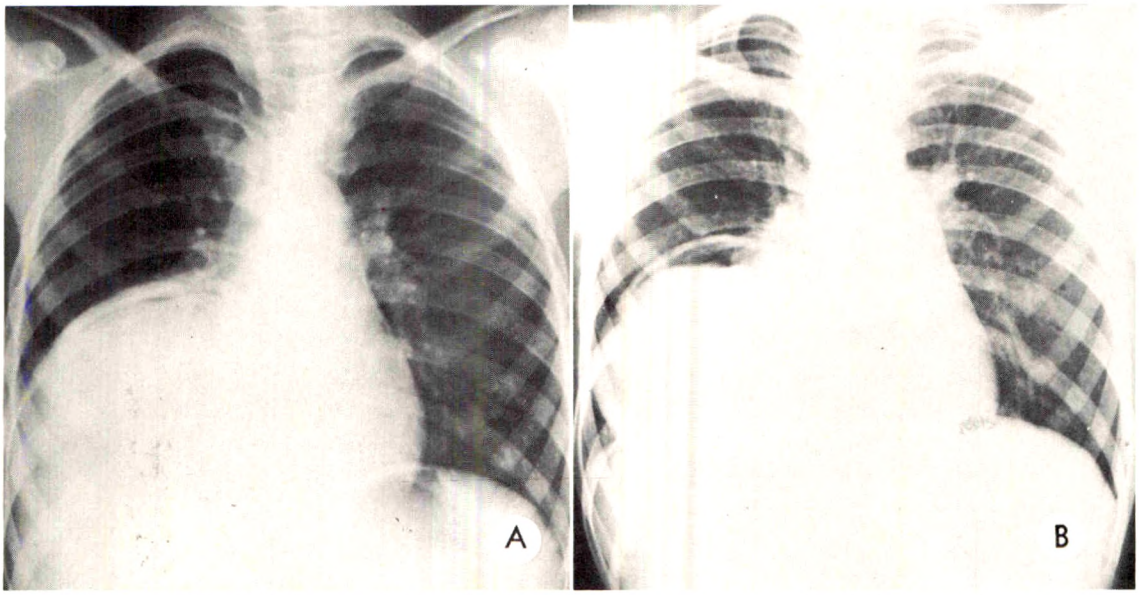


Fig. 8. *Crescent Sign.* (*A*) Roentgenogram showing an early crescent sign with air between the pericyst and the unruptured endocyst. (*B*) Few days later there is a larger air crescent with smooth endocyst.

an air fluid level with no demonstrable floating membranes or daughter cysts. The wall of the pericyst may become quite thick and the whole picture may be indistinguishable from ordinary pyogenic abscess. The expectorant, however, may contain disintegrated pieces of the membranes, degenerated scolices and more commonly hooklets and pus cells.

Hydatid lung abscesses with thick walls and large air fluid levels were seen in 4 patients, 1 with disintegrating floating mem-

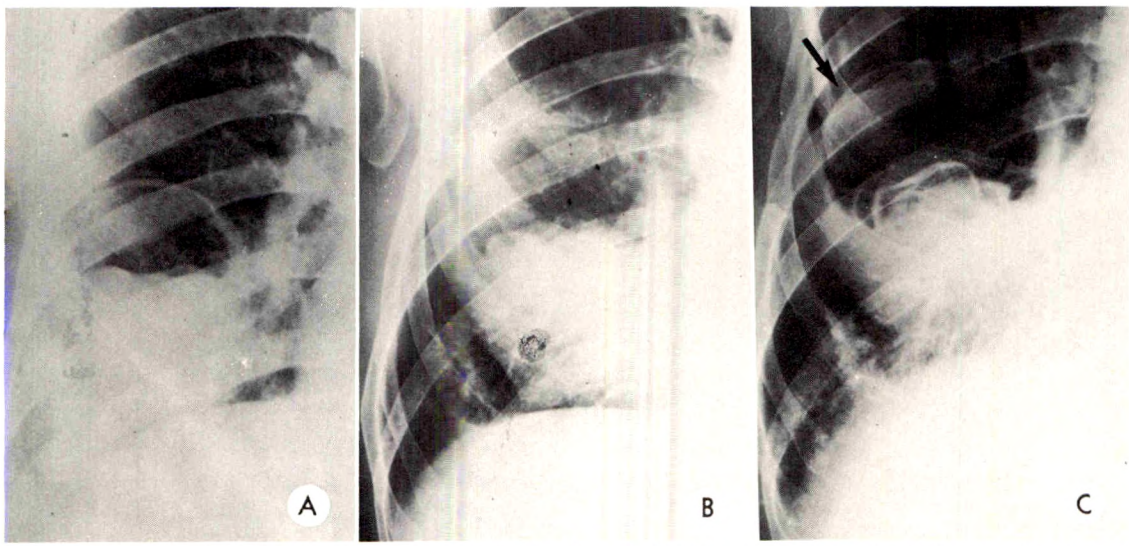


Fig. 9. *Double Arch Sign.* (*A*) Roentgenograms showing air fluid level between the pericyst and endocyst and in the endocyst (*B*) with 2 air fluid levels in the endocyst (*C*) Later 2 arches outline the endocyst and a third the pericyst (arrow). Pneumonitis, fibrotic and atelectatic changes present in the right base were confirmed at operation.

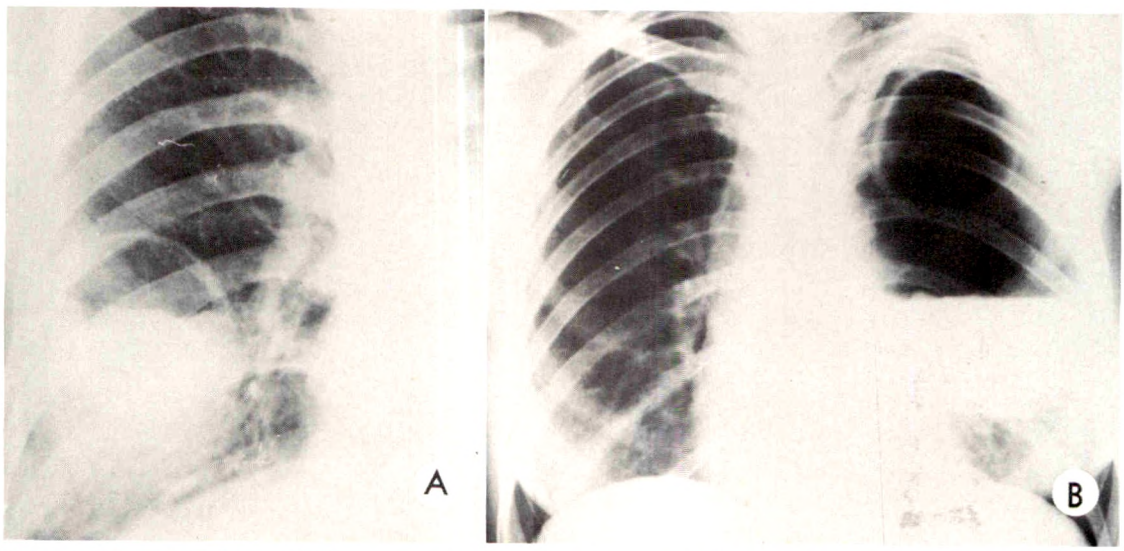


FIG. 10. *Water Lily Sign.* (*A*) Roentgenogram showing the wavy endocyst membrane floating over hydatid fluid. (*B*) There is a large hydatid abscess with thick pericyst wall and floating of the disintegrating endocyst membrane.

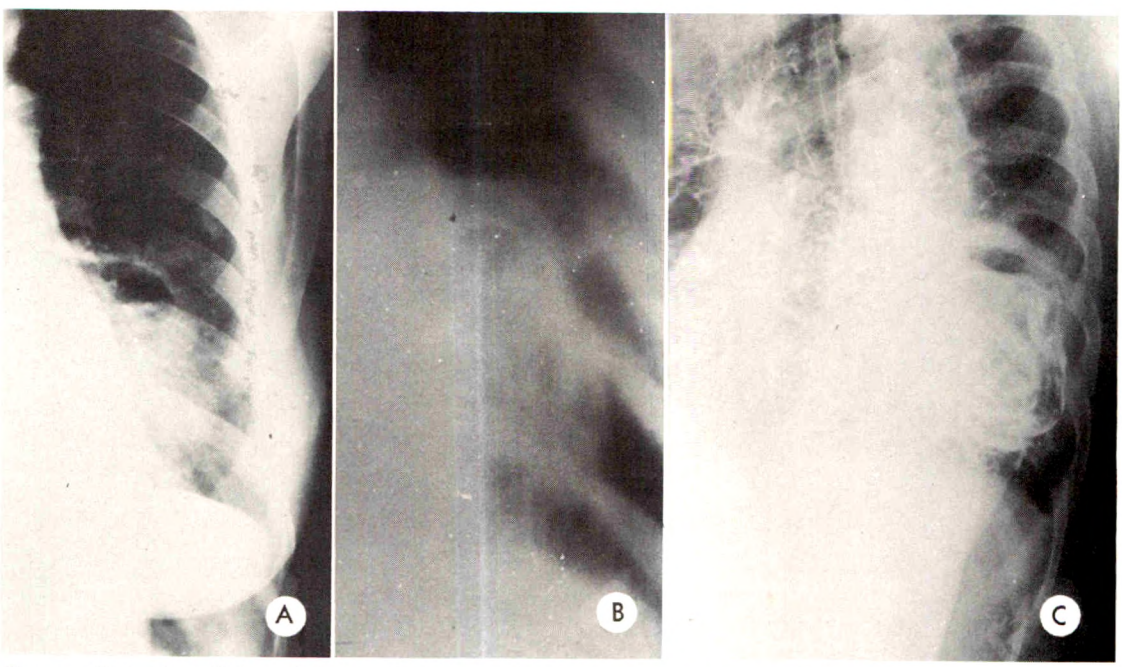


FIG. 11. *"Membrane" Sign.* (*A*) Roentgenogram showing a ruptured cyst with the pulmonary meniscus sign. (*B*) Tomogram showing the membranes as opaque curves against air as contrast and giving the membranes an "onion-peel" appearance. (*C*) Bronchogram showing the endocyst membrane as a lucent undulating line thus determining the hydatid nature of the pulmonary meniscus sign.

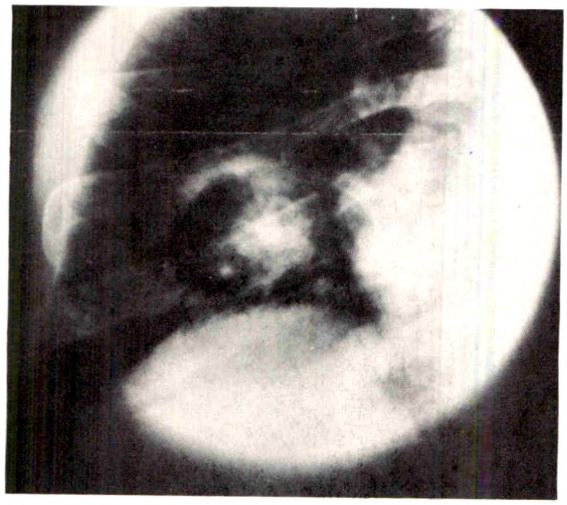


Fig. 12. *Daughter Cyst Sign.* Roentgenogram showing a circular daughter cyst within the pericyst (confirmed at operation).

branes (Fig. 10*B*), however, smaller, 2–3 cm. cavities with fluid levels were seen in 10 patients (Fig. 5*B*).

Empty Pericyst, and Pericyst Effusion

Spontaneous disappearance of hydatid cyst of the lung following rupture is reported to be up to 10 per cent.[18,25] The non-infected pericyst may also disappear in the course of few months without leaving any trace,[6,13] but more commonly may become infected.[18]

We have seen 2 patients with empty pericysts, who gave a history of coughing out membranes and salty fluid. These simulate very closely the congenital air cysts of the lungs or bulla (Fig. 2*C*; and Fig. 5*D*). Empty pericysts were commonly seen following operative removal of the endocyst (Fig. 2*C*), and cyst effusion required a few days of drainage. Two patients developed postoperative abscess and 1 patient bronchopleural fistula.

Atelectasis, Pulmonary Fibrosis, Bronchiectasis and Calcification

With large cysts compression phenomena, minor and segmental atelectatic changes can occur,[25,26] and with chronic pneumonia, dense scarring and minimal bronchiectasis may set in.[25,28] Calcification is extremely rare.[3,4]

Fibrotic changes were noted in 14 patients (28 per cent) (Fig. 9*C*), and mild bronchiectasis in 4 (Fig. 7*D*). Atelectatic changes were present in 14 patients (28 per cent), of which 2 were with segmental atelectasis, 2 with plate-like atelectasis, and 10 with sunburst pattern at the dependent periphery of the cysts (Fig. 5*A*; and Fig. 7*B*). There were 5 patients with displacement and crowding of lung markings (Fig. 3, *A* and *B*). No calcified hydatid cysts were noted.

Pleural Reaction, Effusion, and Empyema

Pleural reaction of varying degrees was noted in 30 patients (60 per cent) at operation, but only about half as much could be seen on roentgenograms. Pleural effusion due to infected cysts occurred in 2 patients (Fig. 13*B*), and empyema in 2, one of which showed a bronchopleural fistula. Few cases of spontaneous pneumothorax with and without tension have been reported in the literature.[1,3,5]

Multiple Hydatid Cysts

Multiple large round shadows in the lungs are said to be virtually pathognomonic of hydatid disease.[26] We have had 5 such patients (Fig. 5*A*), but multiple small cysts may simulate metastasis, Wegener's granulomatosis, other granulomas and cavitating metastasis.[23,26] We had 8 patients in this category, demonstrating cysts with air fluid levels (Fig. 5*B*), ruptured cysts with surrounding pneumonitis among other intact cysts (Fig. 5*C*), grossly intact cysts that showed pneumonitis at tomography (Fig. 5*E*), and cysts with hydatid membranes that could be seen (Fig. 6*A*). The presence of a thin wall of the pericyst, demonstrable membranes and pneumonitis scattered among intact cysts were considered important features in favor of hydatid disease.

Recurrent Hydatid Cysts, Secondary and Exogenous Re-infections

Secondary hydatid cysts are the result of implants or blood dissemination follow-

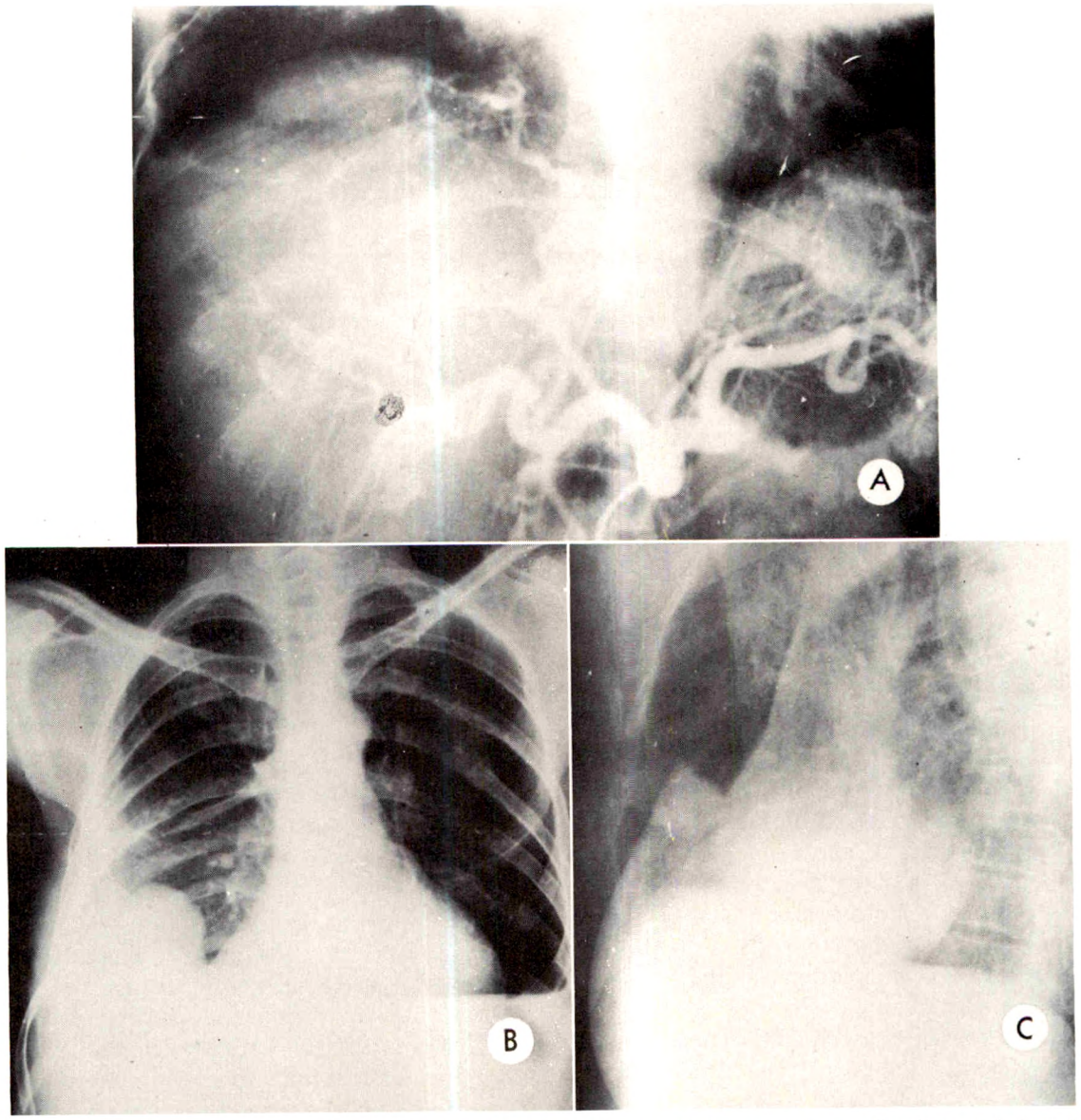


FIG. 13. *Diaphragmatic, Pleural, and Subpleural Hydatid Cysts.* (*A*) Roentgenogram showing an infected hydatid cyst involving the diaphragm and the parietal pleura with blood supply from phrenic arteries. (*B*) Pleural hydatid cyst believed to be an implant from previous operation of hydatid cysts of the right lung and liver. (*C*) Subpleural location of a small hydatid cyst with minimal pleural reaction.

ing rupture of a cyst.[14,29,30] Its incidence may be as high as 20 per cent.[19]

Secondary hydatid cyst implants on pleura were seen in 2 patients, one following spontaneous rupture, and the other following rupture of a cyst of the liver at operation (Fig. 13*B*). In 3 other patients, however, new cysts appeared in the lungs several years after operative removal of intact hydatid cysts of the lungs. These may represent re-infection by ingestion of ova.

EXTRAPULMONARY THORACIC AND EXTRATHORACIC CYSTS

Only a small number, 2–5 per cent, of thoracic cysts are reported to develop in

the mediastinum, pleura[21,22] and diaphragm,[5,29] but as high as 16 per cent of pulmonary cysts are reported to have concurrent liver cysts.[5]

In our series, the liver was involved in 16 patients (30 per cent) (Fig. 6), the plura in 3 (Fig. 13*B*), the diaphragm in 1 (Fig. 13*A*), the left ventricle in 1 (Fig. 14), the chest wall in 1 (implant), the spleen in 2, the peritoneum in 1, the kidney in 1, the brain in 1 and the breast in 2. A rim of curvilinear density of hydatid of the breast was found to be diagnostic of hydatid cyst (Fig. 15, *A* and *B*). There was 1 patient with hepato-bronchial fistula with bile expectoration, and 1 with floating cyst in the pleural cavity. There were no cases of miliary pleural hydatid granulomatosis.[22] Pleural hydatids had to be differentiated from mesothelioma.[23] Previous operation and serology provided clues to the diagnosis.

SUMMARY

Roentgen analysis of 50 cases of hydatid disease of the lungs is presented.

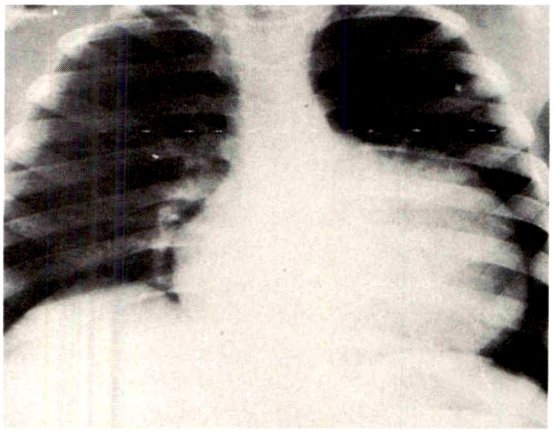


FIG. 14. *Hydatid Cyst of the Heart.* Roentgenogram showing an enlarged left ventricle. At operation, a hydatid cyst of the left ventricle was removed. (Courtesy of Dr. M. Slim.)

The hydatid cysts of the lungs were commonly solitary, well circumscribed, polycyclic, unruptured, at the lung bases, and at the posterior segments.

They exhibited different rates of growth in the same patient with a maximum growth of 7 cm. in 7 months. The presence of 2 or more large well circumscribed masses

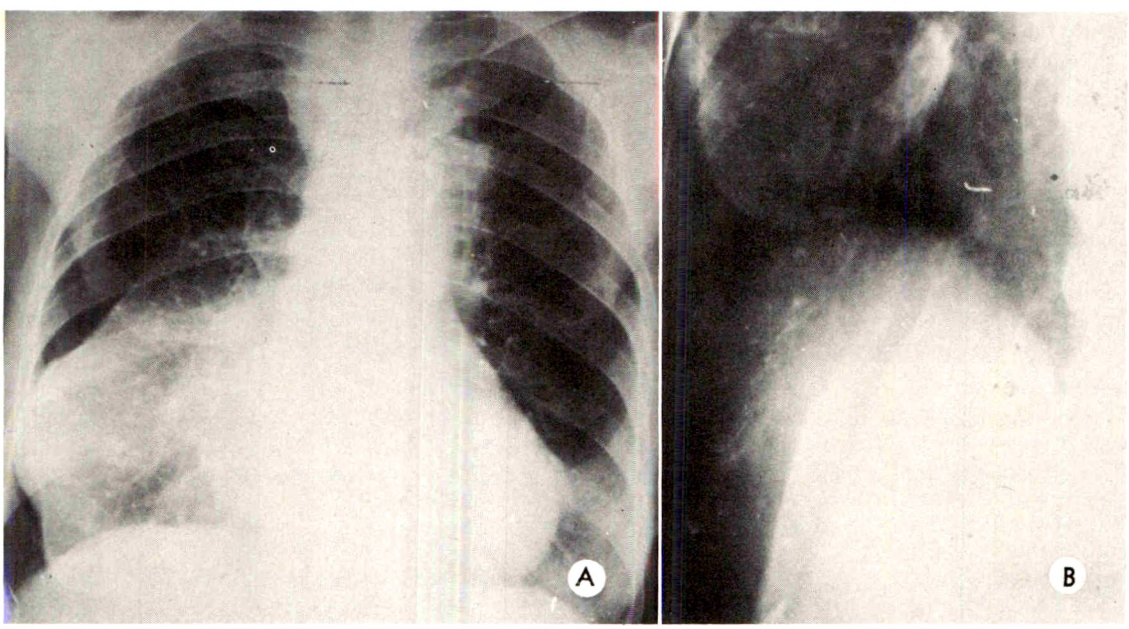


FIG. 15. *Hydatid Cyst of the Breast.* (*A*) Roentgenogram showing enlarged right breast with a rim of dense curvilinear shadow of hydatid cyst. An infected hydatid is also present near the aortic knob. (*B*) Heavy penetrated view showing the dense rim of the cyst. No calcifications were seen at histology.

were found to be virtually pathognomonic of simple hydatid cysts. Characteristics of multiple small cysts are presented.

The roentgen manifestations of rupturing and ruptured hydatid cysts included the classical roentgen signs; *i.e.*, the crescent sign, the double arch sign, and the water lily sign. The "membrane" sign on bronchography and tomography and the "daughter cyst" sign are described and illustrated.

Included also are cases showing: empty pericyst; pericyst effusion; hydatid lung abscess; pleural reaction; pleural effusion; and empyema. Accompanying types of pneumonitis included the peri-pericyst allergic and bacterial pneumonitis and aspiration bronchopneumonia. Other complications and sequelae included atelectasis, fibrosis, bronchiectasis, bronchopleural and hepato-bronchial fistulae.

Other aids in diagnosis of hydatid cyst of the lungs included: concurrent characteristic dense rim shadows and calcifications in the liver, spleen and kidneys; liver scan; clinical history; serology; and sputum microscopy. A rim of dense curvilinear shadow in the breast was dignostic of hydatid cyst of the breast.

The incidences of secondary and exogenous re-infections and concurrent extrapulmonary hydatidosis are reported.

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REFERENCES

1. BAKIR, F., and AL-OMERI, M. M. Echinococcal tension pneumothorax. *Thorax*, 1969, *24*, 547–556.
2. BARRETT, N. R., and THOMAS, D. Pulmonary hydatid disease. *Brit. J. Surg.*, 1944, *38*, 39–45.
3. BLOOMFIELD, J. A. Protean radiological manifestations of hydatid infestation. *Australian Radiol.*, 1966, *10*, 330–343.
4. BONAKDAPOUR, A. Echinococcus disease: report of 112 cases from Iran and review of 611 cases from United States. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 660–667.
5. BORRIE, J. Fifty thoracic hydatid cysts. *Brit. J. Surg.*, 1962, *50*, 268–283.
6. CHRYSOSPATHIS, P. Echinococcus cysts of lung. *Dis. Chest*, 1966, *49*, 278–283.
7. FELSON, B. Chest Roentgenology. W. B. Saunders Company, Philadelphia, 1973.
8. FINLAYSON, D. M., and FERGUS, A. Hydatid disease in Toronto. *Canad. M. A. J.*, 1963, *88*, 1004–1009.
9. FOSTER, F. H. Hydatid disease in New Zealand. *New Zealand M. J.*, 1958, *57*, 562–568.
10. FRAYHA, G. J. Studies on hydatid disease in Lebanon. *Lebanon. M. J.*, 1970, *23*, 135–150.
11. FRASER, R. G., and PARE, J. A. P. Diagnosis of the Diseases of the Chest. W. B. Saunders Company, Philadelphia, 1970.
12. GARABEDIAN, G. A., MATOSSIAN, R. M., and SUIDAN, F. G. Correlative study of immunological tests for diagnosis of hydatid disease. *Am. J. Trop. Med.*, 1959, *8*, 67–71.
13. JIDEJIAN, Y. Hydatid disease. *Surgery*, 1953, *34*, 155–167.
14. JONATHAN, O. M. Hydatid disease in North Wales. *Brit. M. J.*, 1960, *1*, 1246–1253.
15. KATZ, A. M., and PAN, C. T. Echinococcus disease in United States. *Am. J. Med.*, 1958, *25*, 759–770.
16. KEGEL, R. F. C., and FATEMI, A. Ruptured pulmonary hydatid cyst. *Radiology*, 1961, *76*, 60–64.
17. MATOSSIAN, R. Personal communication.
18. MCPHAIL, J. L., and ARORA, T. S. Intrathoracic hydatid disease. *Dis. Chest*, 1967, *52*, 772–781.
19. NICKS, R. Thoracic hydatid cyst. *Med. J. Austral.*, 1967, *1*, 999–1004.
20. PIPKIN, A. A., RIZK, E., and BALIKIAN, J. P. Echinococcosis in Near East and its incidence in animal hosts. *Trans. Roy. Soc. Trop. Med. & Hyg.*, 1951, *45*, 253–260.
21. RAKOWER, J., and MILWIDSKY, H. Primary mediastinal echinococcosis. *Am. J. Med.*, 1960, *29*, 73–83.
22. RAKOWER, J., and MILWIDSKY, H. Hydatid pleural disease. *Am. Rev. Resp. Dis.*, 1964, *90*, 623–631.
23. REEDER, M. M. RPC of month from AFIP: hydatid cyst of lung. *Radiology*, 1970, *95*, 429–437.
24. ROUX, B. T. Pulmonary hydatid disease. *Thorax*, 1972, *27*, 365–367.
25. SADRIEH, M., DUTZ, W., and NAVABPOOR, M. S. Review of 150 cases of hydatid cyst of lung. *Dis. Chest*, 1967, *52*, 662–666.
26. SCHLANGER, P. M., and SCHLANGER, H. Hydatid disease and its roentgen picture. AM. J. ROENTGENOL. & RAD. THERAPY, 1948, *60*, 331–347.
27. SLIM, M. S., KHAYAT, G., NASR, A. T., and JIDEJIAN, Y. D. Hydatid disease in childhood. *J. Pediat.*, 1971, *6*, 440–448.

28. Srouji, M., Mulhim, R., and Wilson, J. L. Hydatid cyst of lung with bronchographic evaluation of treatment by internal suture of pericyst. *J. Thoracic & Cardiovasc. Surg.*, 1958, *35*, 779–794.
29. Taiana, J. A. Thoracic hydatid echinococcosis: diagnosis and treatment. *Dis. Chest*, 1966, *49*, 8–14.
30. Waddle, N. Pulmonary hydatid disease: review of 478 cases reported in Louis Barnett Hydatid Registry of Royal Australian College of Surgeons. *Australian & New Zealand J. Surg.*, 1950, *19*, 273–290.
31. Wilson, J. F., Diddams, A. C., and Rausch, R. L. Cystic hydatid disease in Alaska: review of 101 autochthonous cases of Echinococcus granulosus infection. *Am. Rev. Resp. Dis.*, 1968, *98*, 1–15.

PULMONARY INVOLVEMENT IN ADULTS WITH CYSTIC FIBROSIS*

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WHEN first described, cystic fibrosis was believed to be uniformly fatal in early childhood. Until a few years ago, it was rarely seen after adolescence.[23] However, Shwachman[20] recently reported that 200 patients in his clinic have now reached 17 years of age, which contrasts with only 3 patients reported 15 years earlier. This dramatic change is attributed to our better understanding of the varied manifestations of the disease, the recognition that pancreatic insufficiency is not essential for diagnosis, and to improved diagnostic techniques, notably the sweat test, as well as to improved treatment.

Because cystic fibrosis is being encountered with increasing frequency today in young adults,[2,3,10,15,18,19,26] it must be a major consideration in the diagnosis of chronic pulmonary disease in such patients. Yet, there is little in the radiologic literature regarding its presentation in this age group.

In recent years we, also, have observed a growing number of adults with this diagnosis in our clinics. Furthermore, we have noted that their chest findings on roentgenography do not necessarily parallel the classic textbook descriptions which have been based mainly on experience with young children. Along with others, we have noted that as the child grows, the lungs supplant the pancreas and gastrointestinal tract as the major target organ. The above facts have motivated us to review the characteristic pulmonary changes observed after adolescence.

MATERIAL AND METHOD

Fifty-five patients attending our cystic fibrosis clinics at Hahnemann Hospital and Harrisburg Polyclinic Hospital have passed their 17th birthday. This total consists of 24 females and 31 males and accounts for approximately one-fourth of all patients followed during this period. These individuals ranged from neonate through 21 years of age at the time of initial diagnosis, and were between 17 and 31 years on their last visit. Six have been followed from 10 to 20 years and 24 others for 5 or more years. Six patients, between 17 and 31 years of age, have died.

In each instance, the diagnosis has been confirmed by a pilocarpine iontophoresis sweat test. On analyzing the chest roentgenograms of these young adults, we have examined the evolution of their pathologic changes and have correlated these with other pertinent findings.

ROENTGEN FINDINGS

Almost all of our patients have demonstrated increased interstitial markings or cyst-like changes or a combination of the two. The earliest abnormality generally observed is accentuation and haziness of the vascular shadows, predominantly in the upper lobes. Later, the interstitial markings tend to become more irregular, discontinuous and, eventually, may appear reticulonodular (Fig. 1*A*). The fact that they rarely regress completely and are later accompanied by upward hilar retraction, attests to their partially fibrotic nature.

Bronchiectasis exists in one form or another in nearly every case. Clustered, small cyst-like areas representing ectatic bronchi or bronchioles on end appear first, localizing mainly in the parahilar regions. As their walls thicken and interstitial in-

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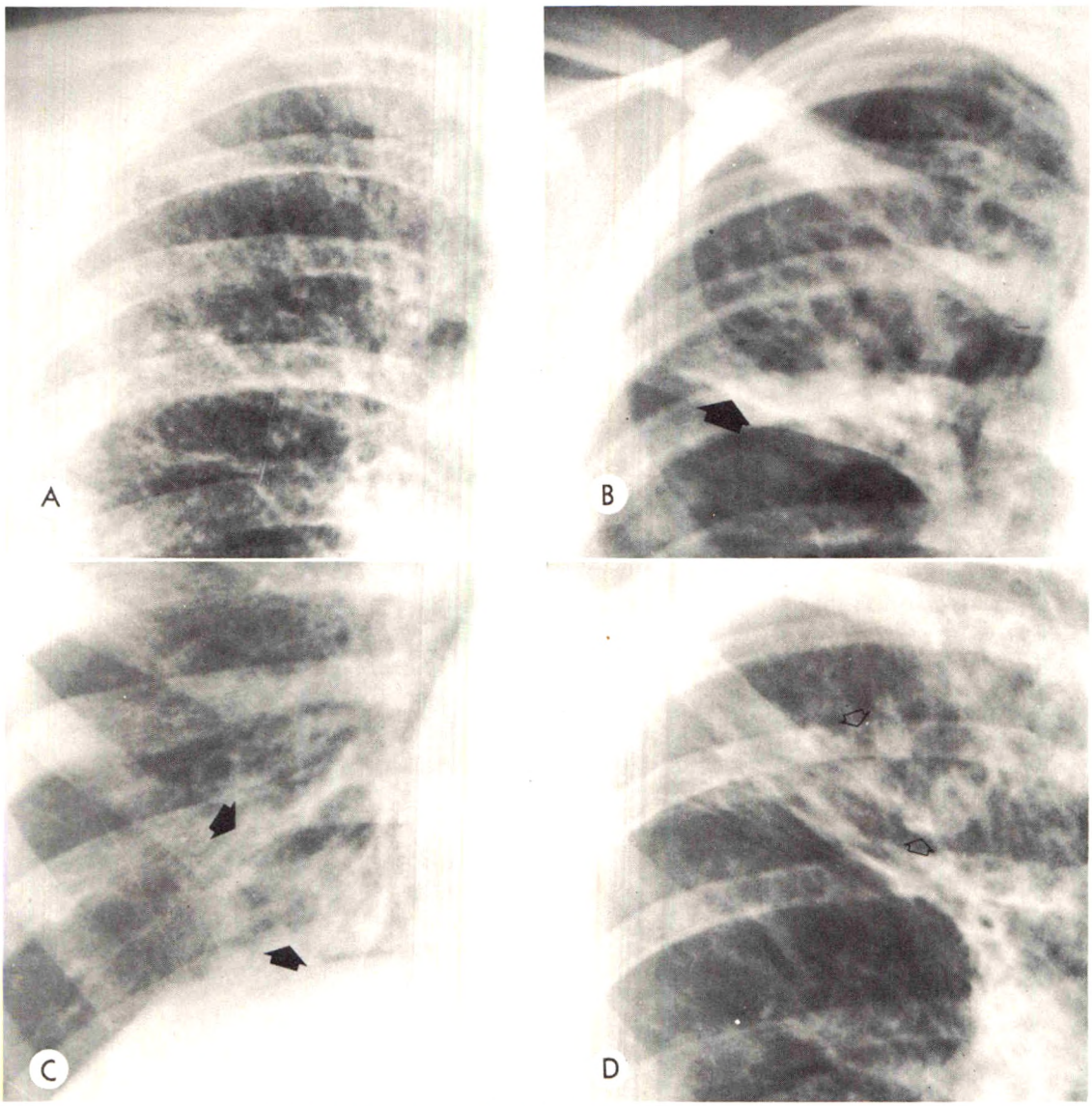


Fig. 1. Pulmonary patterns in cystic fibrosis. (*A*) Mixed interstitial infiltration. (*B*) Honeycombing. Arrow points to nearby thick-walled, dilated bronchus seen in profile rather than *en face*. Note elevated fissure immediately below, indicating volume loss in right upper lobe. (*C*) Basilar bronchiectasis. (*D*) Transient nodular densities (arrows) amidst air-containing, dilated bronchi.

filtration progresses, a honeycomb appearance may ensue (Fig. 1*B*). In advanced stages a bizarre, distorted picture suggesting swiss cheese is produced.

Transient nodular densities (Fig. 1*D*), occasionally occupy these spaces only to be replaced subsequently by lucencies again. Such transition was observed in 12 of our patients at least once during their course. Correlative pathology studies by Hodson and France[8] reveal that such evanescent nodularities consist of endobronchial collections as well as peribronchial abscesses, the latter draining by rupture into adjacent bronchi. When bronchiectasis involves the bases (Fig. 1*C*), its appearance again varies with the amount of secretions contained by the air passages. Dilated bronchi were verified by bronchography (Fig. 2) in only 3 cases, since the procedure

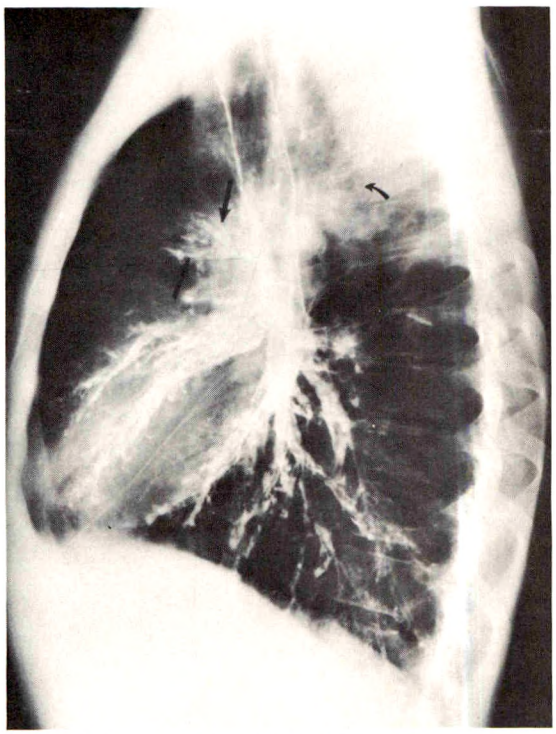


FIG. 2. Advanced bronchiectasis in anterior segment of upper lobe (straight arrows). The bronchi in subapical segment are obstructed by secretions (curved arrow). Despite recurrent respiratory infections since childhood, this patient was not diagnosed until age 21, at the time of the bronchography.

is seldom performed in these patients, but it was widely distributed in each of our autopsied cases.

Pulmonary hyperinflation occurred in roughly one-half of our patients and, with the single exception shown on Figure 3, *A* and *B*, it invariably accompanied widespread parenchymal infiltration (Fig. 5). This is opposed to the situation in infancy, where emphysema may be the first evidence of disease.[4,11,12,22] Increased retrosternal air space often accompanied upper lobe bronchial and peribronchial involvement, and lower lobe hyperinflation tended to follow fibrosis of the upper lobes. A number of patients with advanced disease also had giant apical bullae. Small bullae were invariably present at necropsy. Seventeen of our cases had roentgenologic findings which fulfill Simon's criteria[21] for generalized panacinar emphysema, Grade 3, but judging from our own and other postmortem studies,[6] most of this hyperinflation appears to be due to obstructive or compensatory overexpansion.

The predominant roentgenographic patterns observed in our cases at the time of their most recent examination are tabu-

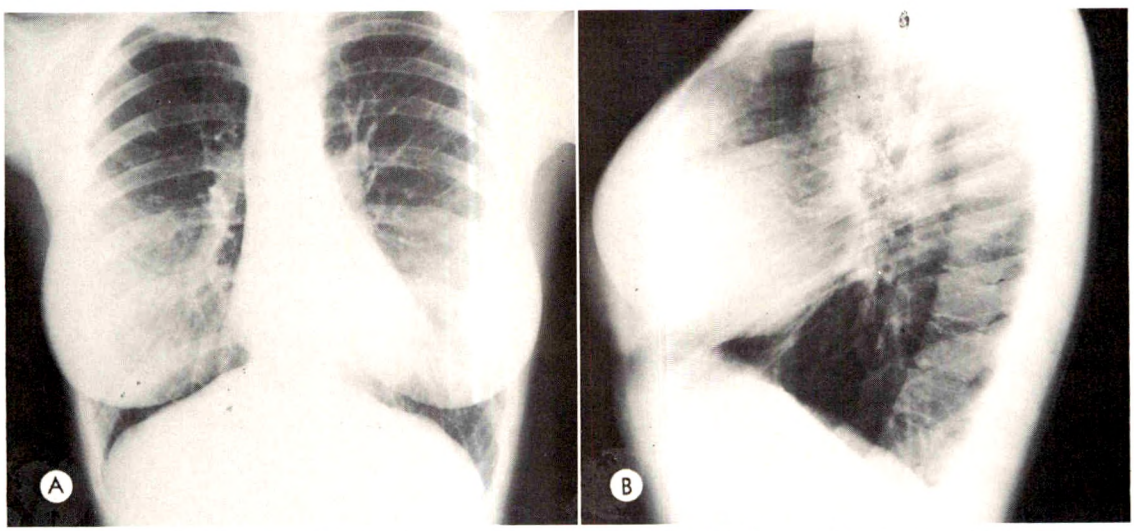


FIG. 3. (*A* and *B*) Marked generalized emphysema despite minimal infiltrates in a 24 year old woman who first developed a cough 4 years earlier. Her mid maximal expiratory flow rate (MMEFR) was only 19.6 per cent of normal.

Table I

PREDOMINANT PATTERNS IN POST-ADOLESCENT CYSTIC FIBROSIS

Degree of Involvement	Mixed Interstitial	Cystic Honeycomb	Transient Nodular	Hyperinflation
Mild	12	9	1	11
Moderate	26	24	1	14†
Advanced	7	11	0	3†
Total (55)*	45	44	2	28
Per Cent	(82)	(80)	(4)	(51)

* Since more than 1 type of lesion may exist in any 1 patient, particularly in later stages, the subtotals under each vertical column add up to more than the total number of 55 patients. For this same reason patients with composite lesions of minimal degree were only 7 in number, those with moderately severe changes totaled 30, etc. (see text).

† Fulfill Simon's criteria[21] for widespread panacinar emphysema, Grade 3.

lated on Table 1. We have arbitrarily graded the degree of involvement on the basis of severity and distribution. Our compilation indicates that only 7 patients were minimally affected, 30 had moderately severe changes, and 18 patients had advanced lesions of one or more types.

PULMONARY COMPLICATIONS

The main pulmonary arteries were disproportionately large in 26 patients (Table II). This manifestation of pulmonary hypertension occurred mostly in individuals with moderately or far advanced pulmonary disease. Enlarged hilar vessels could not be readily separated from the frequently coexisting hilar and tracheobronchial lymphadenopathy. As is not unusual in cor pulmonale, the cardiac silhouette remained small until the appearance of right heart failure, a preterminal event which occurred in 6 patients (Fig. 4, *A* and *B*).

Table II

PULMONARY COMPLICATIONS IN 55 CASES

Complication	Patients
Cor pulmonale	26
Pneumonitis	16
Right heart failure	6
Hemorrhage*	7
Pneumothorax	4
Effusion	2
Atelectasis	1

* Unlike other complications, hemorrhagic sites were never seen roentgenographically.

Clinical pneumonitis was difficult to identify on roentgenography. It was detectable in 16 instances, usually as small patchy peribronchial infiltrates. Major segmental or lobar consolidation was found only 3 times. Pleural effusion was more uncommon, being encountered in only 2 patients in whom it was minimal in amount. Coagulase positive *Staphylococcus* and *Pseudomonas* were the most common offending organisms cultured from sputum.

Hemoptysis occurred in 7 patients, 3 of whom had advanced mixed interstitial disease. Unfortunately, the bleeding sites could never be localized roentgenographically. The ominous significance of this complication has been pointed out previously by one of the authors.[9] This impression was supported by 2 patients included in this series who died shortly after such an episode. One case was our only Black patient, a 31 year old woman, who was admitted with meconium ileus equivalent 4 months after massive hemoptysis, and who died 4 days following admission.

Pneumothorax occurred 6 times in 4 patients, all of whom had advanced disease

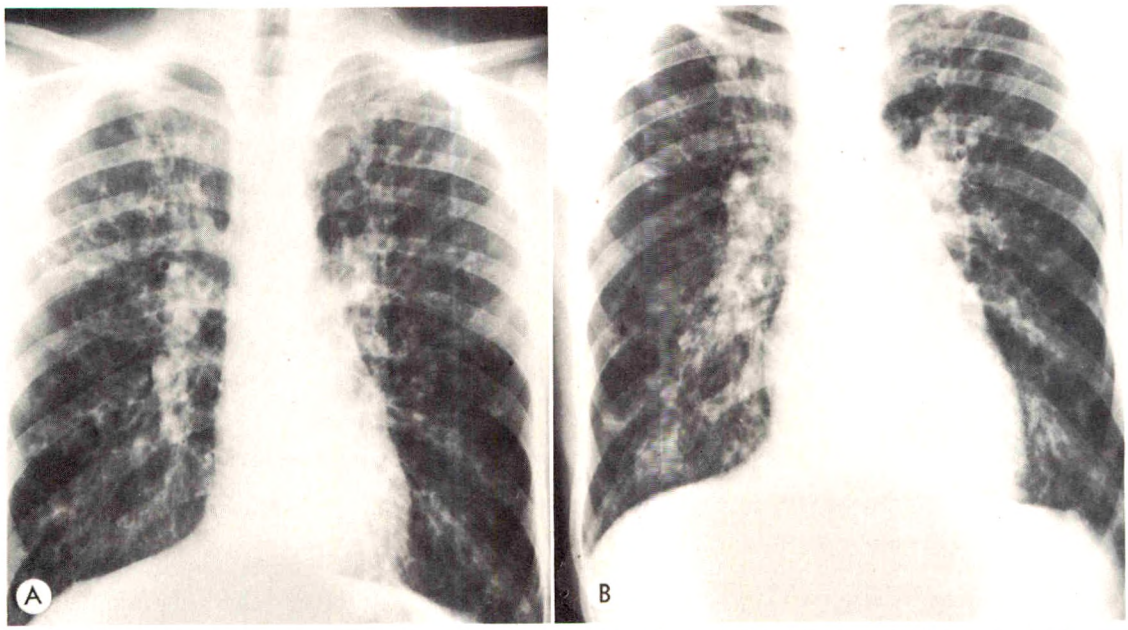


FIG. 4. (*A* and *B*) Right heart failure in a 20 year old male with cystic fibrosis diagnosed in early childhood. He also had esophageal varices, duodenal changes, and marked clubbing. Note the marked increase in heart size over the 3 month period between *A*, (March 6, 1972) and *B* (June 6, 1972). Autopsy 12 days after *B* revealed right ventricular hypertrophy and dilatation.

and/or hyperinflated lungs. Two of these also had large apical bullae. In view of the marked parenchymal scarring, it is not surprising that re-expansion of these lungs was sometimes considerably delayed (Fig. 5; and 6).

Atelectasis was identifiable on roentgenography in only a single patient, but segmental collapse may well have been obscured in other cases by surrounding diseased lung. Clubbing, on the other hand, was readily apparent in most patients with at least moderately advanced pulmonary disease.

CORRELATIVE PULMONARY FUNCTION STUDIES

Pulmonary function studies were performed at regular intervals in most patients, and as frequently as a month apart in some. These studies included forced vital capacity (FVC), mid maximal expiratory flow rate (MMEFR), forced expiratory volume in 1 second (FEV_1), and peak expiratory flow rate (PEFR). The results were recorded as percentages of expected



FIG. 5. Right pneumothorax (arrows) in a 17 year old male complicating advanced lung disease and pulmonary hypertension. He was diagnosed at $5\frac{1}{2}$ years and by age 11 already had moderately severe interstitial disease and hyperinflation. This lung resisted re-expansion for 3 months.

normal levels.

As observed in other studies,[17,27] the MMEFR, which assesses airway obstruction, was the most sensitive index of impaired function and coincided most closely with roentgen findings. Those with mild or minimal roentgen changes generally had normal levels. However, in two patients with low MMEFRs at the time that their roentgenograms were minimally abnormal, the MMEFR proved to be the forecaster of deterioration noted on roentgenograms soon thereafter. In all cases the decline of both the MMEFR and FEV_1, another measure of airway obstruction, paralleled progressive pulmonary disease manifested roentgenographically. They were invariably low in patients with moderate or advanced roentgen findings. Only in advanced cases, where the FEV_1 was less than 30 per cent of normal and the MMEFR 15 per cent or less, was an appreciable effect on the FVC and PEFR noted. Neither the degree of localization nor the distribution of the disease process on roentgenography had appreciable influence on these criteria of pulmonary function.



FIG. 6. Appearance of lung at autopsy of same patient as shown in Figure 5, 2 years later. Examination revealed advanced bronchiectasis, widespread consolidation, peribronchiolar abscesses, focal atelectasis and emphysema, and many emphysematous bullae.

Several patients also had radioactive perfusion scans (Tc^{99m} labeled albumin) performed. In these individuals the areas of diminished perfusion correlated well with areas of maximum disease demonstrable roentgenographically (Fig. 7, *A* and *B*).

INVOLVEMENT OF OTHER SYSTEMS

In our adults, pancreatic and gastrointestinal involvement followed the lungs and sinuses in frequency. This agrees with previous experience which approximates an incidence of 84 per cent.[25] Findings included esophageal varices due to biliary cirrhosis, meconium ileus equivalent, rectal prolapse, and evidence of malabsorption such as steatorrhea. Splenomegaly was visualized in 8 patients, and there were occasional instances of cholecystitis. Intussusception and pneumatosis cystoides intestinalis, which also have been reported, were not encountered in this series.

In keeping with the well known fact that the islet cells of the pancreas are spared until relatively late, we found only 5 clinically overt diabetics among these 55 cases. Interestingly, perusal of the data failed to disclose any relationship between the extent and severity of pulmonary and extrapulmonary disease.

DISCUSSION

Cystic fibrosis is a protean disease which may afflict nearly every site of mucous secretion in the body. However, because it was first clinically recognized as a dis-

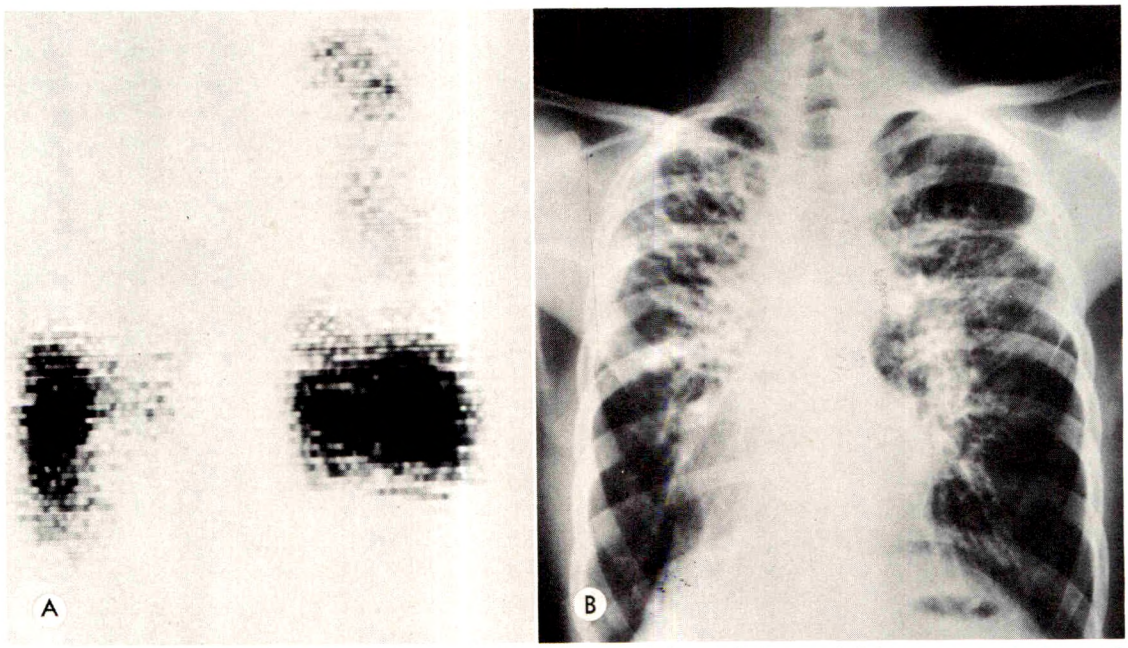


FIG. 7. Close correlation between markedly diminished upper lobe perfusion on (*A*) lung scan and advanced upper lobe interstitial and cystic disease on (*B*) roentgenogram of patient illustrated in Figures 5 and 6 shortly before death.

ease of the pancreas, it was called cystic fibrosis of the pancreas. The term mucoviscidosis which next gained popularity became inappropriate when it was appreciated that sweat glands, as well as mucous exocrine glands, were also involved. After infancy, interest is directed mainly to the lung, rather than the pancreas, inasmuch as it is almost always involved and has such a strong influence on the over-all condition and prognosis of the patient.

The disease is generally progressive (Fig. 8, *A–C*), and respiratory and secondary heart failure are the chief causes of death. Cystic fibrosis today is the most common lethal genetic disease of the Caucasian race,[13] with an incidence of approximately 1 in 1,600.

The basic respiratory tract lesion is obstructive airway disease leading to bronchiectasis. Bronchiectasis was seen in four-fifths of our patients. The process originates in excessive viscid secretions which plug peripheral air passages, thereby impeding drainage and ventilation, causing overinflation and atelectasis, and predisposing to infection. The ciliated bronchial epithelium becomes inactivated, hypertrophied and converted to goblet cells.[16] Chronic bronchitis and peribronchitis eventually occur, and a vicious cycle of progressive, irreversible changes ensues. Destructive emphysema increases with time, but remains relatively focal, mild and infrequent as compared to "dilatation emphysema" or hyperinflation.[1,6]

The clinical spectrum of cystic fibrosis expands with age. On the one hand are patients who are nearly asymptomatic. They are diagnosed late in life unless suspicion has been directed by an afflicted sibling. Findings in this group tend to be minimal initially and their roentgenograms may show only slight increase of perivascular markings resembling chronic bronchitis (Fig. 9, *A* and *B*). At the other extreme are patients who experience severe respiratory problems from early childhood, and who suffer from severe bronchiectasis and hyperinflation by puberty. Moderately severe and advanced cases have dominated our experience (Table 1). However, it is vital to detect the subclinical or mild case so that prophylactic treatment may be

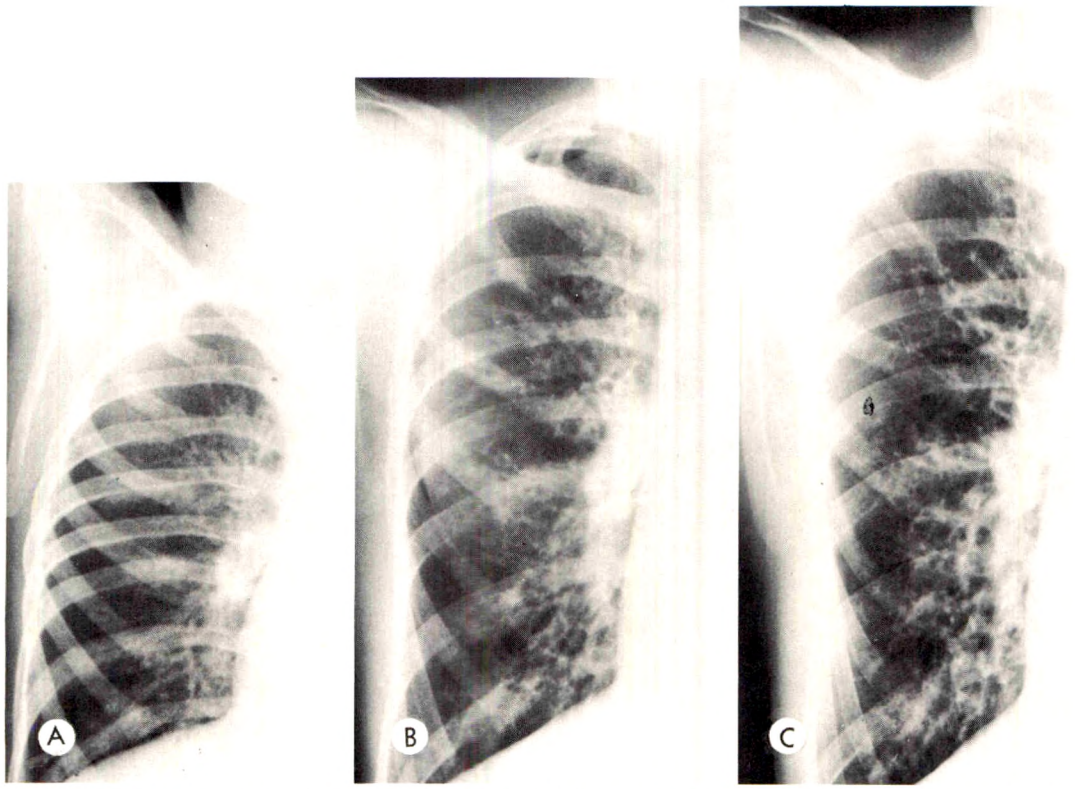


Fig. 8. (*A*) January 23, 1954. (*B*) June 22, 1966. (*C*) July 26, 1972. Roentgenograms obtained over an 18 year period in a male aged 23 years, first diagnosed at birth with meconium ileus. Studies beginning at age 4 (*A*) show gradual progression from mild interstitial disease to advanced cystic bronchiectasis and pulmonary hypertension (*C*).

initiated and subsequent deterioration retarded if not arrested.[5,19]

Cystic fibrosis often presents differently in young adults than in early life. As previously stated, in infancy generalized hyperinflation of the lungs may be the sole manifestation,[4,11,12,22] and collapse of an entire lobe or major segment, particularly an upper lobe, is not at all uncommon.[4,8,12] Emphysema was not a prominent feature in 7 adults reported by Coates,[3] although Tomashefski *et al.*[26] described perifocal and compensatory emphysema in their young adults. Differing interpretations of the term emphysema may explain this difference of opinion. Whereas we have found considerable hyperinflation and/or emphysema in close to one-half of our patients, such changes have almost invariably developed in the wake of advanced pulmonary infiltration.

Shrinkage or volume loss of involved parenchyma in our patients appeared to be primarily due to scarring rather than major atelectasis. Only focal atelectasis has been discovered in our patients coming to autopsy. It is conceivable that alveolar hypoplasia secondary to bronchial occlusion in early childhood contributes to the reduced lung volume.[21]

A number of observers,[3,24,26] including the authors, have been impressed by the predisposition of the upper lobes, particularly on the right side, to this disease process. This is a carry over from infancy at which time, as mentioned, atelectasis may selectively involve an upper lobe and irreversible bronchial damage may occur. In this connection, Gyepes *et al.*[7] have reported diminished perfusion and ventilation in the upper lobes on isotopic lung scannings before the advent of roentgenographic changes.

Other complications, notably pneumo-

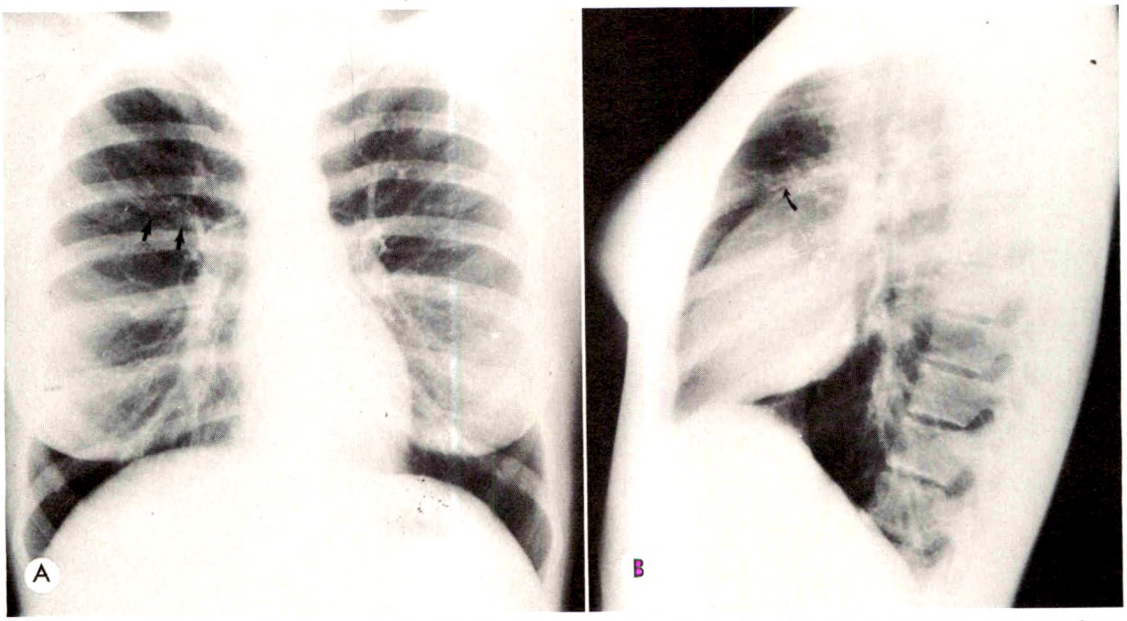


FIG. 9. (*A* and *B*) Patient with a mild cough for 10 years and various allergies. Chest roentgenograms show only minimal interstitial infiltrations and, possibly, bronchiectasis in the anterior segment of the right upper lobe (arrows). Sweat test was, nonetheless, positive.

thorax and hemoptysis, rarely occur before puberty. Cor pulmonale, in particular, is an accompaniment of late disease[14] and, likewise, tends to be more common in the older age group. The importance of recognizing right heart failure resides in the fact that it so often precedes death. The minimal incidence of pleural effusion or empyema at all ages reflects the remarkable resistance of patients with cystic fibrosis to the respiratory pathogens they carry. Interestingly, neither of the 2 patients in the present series who developed effusions had advanced disease.

DIFFERENTIAL DIAGNOSIS

A young adult with chronic pulmonary disease should bring to mind the possibility of cystic fibrosis. The diagnosis is supported by the presence of a cystic or interstitial process preferentially involving the upper and mid lung fields, and may be confirmed by chlorides in excess of 80 mEq./l. on sweat test. Pancreatic and gastrointestinal abnormalities are helpful but not a *sine qua non* in making the diagnosis.

On observing such roentgen findings one must rule out a number of other possibilities, prominent among which are tuberculosis and chronic bronchitis. The other entities listed in Table III are also chronic and predominantly interstitial disease processes. However, in none is bronchiectasis a major feature, and, in none, excepting chronic asthma and silicosis, does cor pulmonale commonly occur.

The so-called "dirty chest" often associated with asthma and chronic bronchitis may simulate mild cystic fibrosis, but would hardly be confused with the more advanced stage of the disease demonstrating irregular, coarse interstitial infiltrates. Tuberculosis poses the biggest problem in the differential diagnosis. However, widespread, irregular infiltrates in the absence of cavitation, calcification and effusion should rule out an acid fast infection. In our experience, tuberculosis occurs quite rarely in patients with cystic fibrosis.

SUMMARY

Cystic fibrosis is no longer rare in adults.

Table III

DIFFERENTIAL DIAGNOSIS OF PULMONARY CYSTIC FIBROSIS IN ADULTS

Disease	Similarity	Contrast
Tuberculosis	Upper lobes	Less uniform and diffuse; calcifies and cavitates; effusion
Chronic asthma with bronchitis	Hyperinflation	Infiltrates less prominent, more uniform, not "cystic"
Silicosis	Upper lobes; pleural involvement minimal	More nodular and often conglomerate infiltrates; eggshell hilar calcification
Sarcoidosis	Pleural involvement rare	More changeable, nodular and localized; paratracheal lymph nodes
Histiocytosis X	Honeycombing, upper lobes; pneumothorax	More uniform and nodular; hila not enlarged
Connective tissue disease	Variable patterns	More often basilar and alveolar

The lung is the main target organ and the basic lesion is bronchial obstruction leading to bronchiectasis. Interstitial infiltrates and cyst-like changes are characteristic on roentgenography.

Complications include pulmonary hypertension, pneumonitis, hemorrhage, and pneumothorax.

Little correlation exists between respiratory tract and pancreatic and gastrointestinal involvement, despite similarities in pathogenesis.

Pulmonary cystic fibrosis in adults does not always resemble the condition in children, and must be differentiated from other chronic interstitial diseases.*

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* Since submitting this paper for publication we have confirmed the diagnosis of cystic fibrosis in 3 additional adults, thereby raising the total number to 58.

REFERENCES

1. Bedrossian, C. W. M. Personal communication.
2. Cabanel, G., Voge, R., and Rambaud, P. Mucoviscidosis in adult: predominantly bronchial expression. *J. franç. méd. et chir. thorac.*, 1967, *21*, 245–256.
3. Coates E. O., Jr. Characteristics of cystic fibrosis in adults: report of seven patients. *Dis. Chest*, 1966, *49*, 195–204.
4. di San'Agnese, P. A. Bronchial obstruction with lobar atelectasis and emphysema in cystic fibrosis of pancreas. *Pediatrics*, 1953, *12*, 178–190.
5. Doershuk, C. F., Mathews, L. W., Tucker, A. S., and Spector, S. Evaluation of prophylactic and therapeutic program for patients with cystic fibrosis. *Pediatrics*, 1965, *36*, 675–688.
6. Esterly, J. R., and Oppenheimer, E. H. Cystic fibrosis of pancreas: structural changes in peripheral airways. *Thorax*, 1968, *23*, 670–675.
7. Gyepes, M. T., Bennett, L. R., and Hassakis, P. C. Regional pulmonary blood flow in cystic fibrosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 567–575.
8. Hodson, C. J., and France, N. E. Pulmonary changes in cystic fibrosis of pancreas: radiopathological study. *Clin. Radiol.*, 1962, *13*, 54–61.
9. Holsclaw, D. S., Grand, R. J., and Shwachman, H. Massive hemoptysis in cystic fibrosis. *J. Pediat.*, 1970, *76*, 822–838.
10. Jones, J. S. Adult cystic fibrosis (mucoviscidosis): fatty replacement of pancreas in woman aged 47 years. *Brit. J. Dis. Chest*, 1970, *64*, 25–36.

11. KEATS, T. E. Generalized pulmonary emphysema as isolated manifestation of early cystic fibrosis of pancreas. *Radiology*, 1955, *65*, 223–226.
12. KIRKPATRICK, J. A., JR., and CAPITANIO, M. A. Pulmonary manifestations of systemic diseases in infants. *Seminars in Roentgenol.*, 1972, *7*, 149–172.
13. MCKUSICK, V. A. Mendelian Inheritance in Man: Catalogues of Autosomal Dominant, Autosomal Recessive, and X-linked Phenotypes. Johns Hopkins Press, Baltimore, 1966, p. 167.
14. Moss, A. J. Cor pulmonale in cystic fibrosis. In: Cystic Fibrosis. Part I. Edited by E. Rossi, and E. Stolle. S. Karger AG, Basel, 1967, pp. 187–193.
15. NOVY, M. J., TYLER, J. M., SHWACHMAN, H., ESTERDAY, C. L., and REID, D. E. Cystic fibrosis and pregnancy. *Obst. & Gynec.*, 1967, *30*, 530–536.
16. REID, L., and DE HALLER, R. Bronchial mucous glands: their hypertrophy and change in intracellular mucus. In: Cystic Fibrosis. Part I. Edited by E. Rossi, and E. Stoll. S. Krager AG, Basel, 1967, pp. 194–199.
17. REILLY, B. J., FEATHERBY, E. A., WENG, T. R., CROZIER, D. N., DUIC, A., and LEVISON, H. Correlation of radiological changes with pulmonary function in cystic fibrosis. *Radiology*, 1971, *98*, 281–285.
18. ROSENOW, E. D., III, and LEE, R. A. Cystic fibrosis and pregnancy. *J.A.M.A.*, 1968, *203*, 227–230.
19. SHWACHMAN, H., KULCZYCKI, L. L., and KHAW, K. T. Studies in cystic fibrosis: report on sixty-five patients over 17 years of age. *Pediatrics*, 1965, *36*, 689–699.
20. SHWACHMAN, H. Cystic fibrosis. In: Disorders of the Respiratory Tract in Children. Volume I. Pulmonary Disorders. Edited by E. L. Kendig, Jr. W. B. Saunders Company, Philadelphia, 1972, pp. 524–547.
21. SIMON, G. Plain radiograph in relation to lung physiology. *Radiol. Clin. North America*, 1973, *11*, 3–16.
22. SINGLETON, E. B., and WAGNER, M. L. Radiologic Atlas of Pulmonary Abnormalities in Children. W. B. Saunders Company, Philadelphia, 1971, p. 136.
23. SPENCER, H. Pathology of the Lung. Second edition. Pergamon Press, Publisher, New York, 1968, pp. 149–151.
24. STUE, O. Pulmonary changes in mucoviscidosis. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1963, *99*, 625–631.
25. TAUSSIG, L. M., SALDINO, R. M., and DI SANT'AGNESE, P. A. Radiographic abnormalities of duodenum and small bowel in cystic fibrosis of pancreas (mucoviscidosis). *Radiology*, 1973, *106*, 369–376.
26. TOMASHEFSKI, J. F., CHRISTOFORIDIS, A. J., and ABDULLAH, A. K. Cystic fibrosis in young adults. *Chest*, 1970, *57*, 28–36.
27. ZAPLETAL, A., MOTOYAMA, E. K., GIBSON, L. E., and BOUHUYS, A. Pulmonary mechanics in asthma and cystic fibrosis. *Pediatrics*, 1971, *48*, 64–72.

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THE ADULT RESPIRATORY DISTRESS SYNDROME*

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THE term "adult respiratory distress syndrome" is used to describe a serious and often fatal condition which may develop in any patient subjected to severe trauma, major surgery or critical illness.[1,2,4,7,12,20,30] While the precise etiology of the primary pulmonary injury remains obscure, the syndrome presents a characteristic clinical, roentgenologic and pathologic picture which has been described under a variety of terms largely determined by the particular predisposing situation, or the clinical or pathologic bias of the author. Some of the more commonly used synonyms for this syndrome are listed in Table I.

TABLE I

SYNONYMS FOR THE ADULT RESPIRATORY DISTRESS SYNDROME

Shock lung
Post-traumatic pulmonary insufficiency
Hemorrhagic lung syndrome
Stiff lung syndrome
Solid lung syndrome
Adult hyaline membrane disease
Respirator lung
Congestive atelectasis
Progressive pulmonary consolidation
Pump lung
Post-transfusion lung
Transplant lung
Post-perfusion lung
?Respiratory insufficiency associated with oxygen toxicity, fat embolism syndrome and disseminated intravascular coagulation

The incidence of this respiratory distress syndrome appears to have increased dramatically in recent years. While this may be partly due to the wider availability of modern techniques for the recognition of respiratory failure, it is probably mainly a reflection of the success of sophisticated resuscitation procedures which permit survival of many patients who previously would have died earlier following massive trauma, major surgery or critical medical illness. Recognition of the syndrome is of major concern to all involved in the management of patients in an intensive care unit, and the radiologist is an important member of this team.

The present communication is essentially concerned with a discussion of the adult respiratory distress syndrome with emphasis on the roentgenologic aspects of this entity and is based upon a review of the findings in 75 cases seen during the years 1968 to 1973 (Table II).

CLINICAL FEATURES

The clinical presentation and course are relatively constant and predictable. Initially, there is a period of severe shock or extensive tissue injury resulting from a variety of causes such as massive trauma, hemorrhage, major surgery, sepsis or burns followed by successful resuscitation. This, in turn, is followed by a latent period of variable duration, which may last for several hours or days but is generally in the region of 12 to 24 hours, at which time clinical evidence of the respiratory problem becomes manifest with the development of tachypnea, dyspnea and cyanosis accompanied by a decrease in the arterial oxygen tension. Initially the lungs are dry to auscultation and tracheobronchial secretions are minimal. Despite the institution of assisted or controlled ventilation, there is frequently a progressive diminution in arterial oxygenation and lung compliance requiring high oxygen concentrations in the inspired gas mixture and high ventilator pressures. Secondary pulmonary sepsis is common and a fatal outcome is not unusual. More recently, with the earlier recog-

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TABLE II

SUMMARY OF FINDINGS IN 75 PATIENTS WITH ADULT RESPIRATORY DISTRESS SYNDROME

Clinical Features	Roentgenologic Findings	Pathologic Findings
Primary insult Resuscitation Latent period		Vascular congestion, interstitial edema
Onset of clinical symptoms, tachypnea, dyspnea, cyanosis, hypoxia	Normal, ?Interstitial edema	Progressive congestion, interstitial edema, foci of intra-alveolar edema and hemorrhage
Progression of clinical symptoms and hypoxia requiring assisted ventilation	First definite changes patchy, ill-defined bilateral densities	More diffuse intra-alveolar edema and hemorrhage
Persistent arterial oxygen desaturation, progressive loss of lung compliance, arteriovenous shunting	Increase in extent and coalescence of bilateral densities without pleural effusion	Solid lungs, thickened alveolar septa, hyaline membranes
As above with onset of fever. Increased tracheobronchial secretions, rigors and rapid downhill course	Diffuse bilateral opacification of lungs frequently with abscesses, pneumothorax and/or pneumomediastinum	Prominent hyaline membranes, inflammatory changes, microabscesses or gross necrosis, fibrosis

nition of this syndrome, and the application of continuous positive pressure breathing with positive end expiratory pressure (PEEP), there has been an improved prognosis with reversal of the pulmonary lesion and clinical recovery in many patients.

PATHOLOGIC FINDINGS

The pathologic changes found at autopsy are relatively constant and stereotyped,[2,19,20,23] and depend to a large degree on the duration of the pulmonary insufficiency prior to death as well as the therapeutic procedures adopted. If death occurs within 24 to 48 hours following the onset of the clinical syndrome, the lungs are found to be heavy, airless and grossly hemorrhagic, commonly described as "beefy" or "liver-like." Microscopically, there is marked capillary congestion, interstitial edema, foci of intra-alveolar edema and hemorrhage and micro-atelectases. If death occurs after a period of 5 to 7 days, the lungs appear less hemorrhagic, exhibiting a grayish color, but are again noted to be heavy, solid and airless. Microscopically, there is evidence of hypertrophy and hyperplasia of the alveolar lining cells with prominent hyaline membranes lining the alveolar ducts and sacs and intra-alveolar proteinaceous or fibrinous deposits. If the patient survives more than 1–2 weeks, there is usually fibroblastic proliferation involving both the alveolar and interstitial tissues and almost invariably evidence of secondary pulmonary infection superimposed on the basic pathologic picture described above. Since the pulmonary sepsis is commonly due to hospital-acquired gram negative bacteria, there is frequently evidence of gross tissue necrosis or microabscesses.

ROENTGENOLOGIC FINDINGS

These tend to be constant and stereotyped in nature, following a more or less predictable pattern.[17] Correct interpretation of the roentgen changes obviously demands recognition by the radiologist of the existence of this syndrome as a distinct entity and is only possible if the relatively nonspecific findings on any single roent-

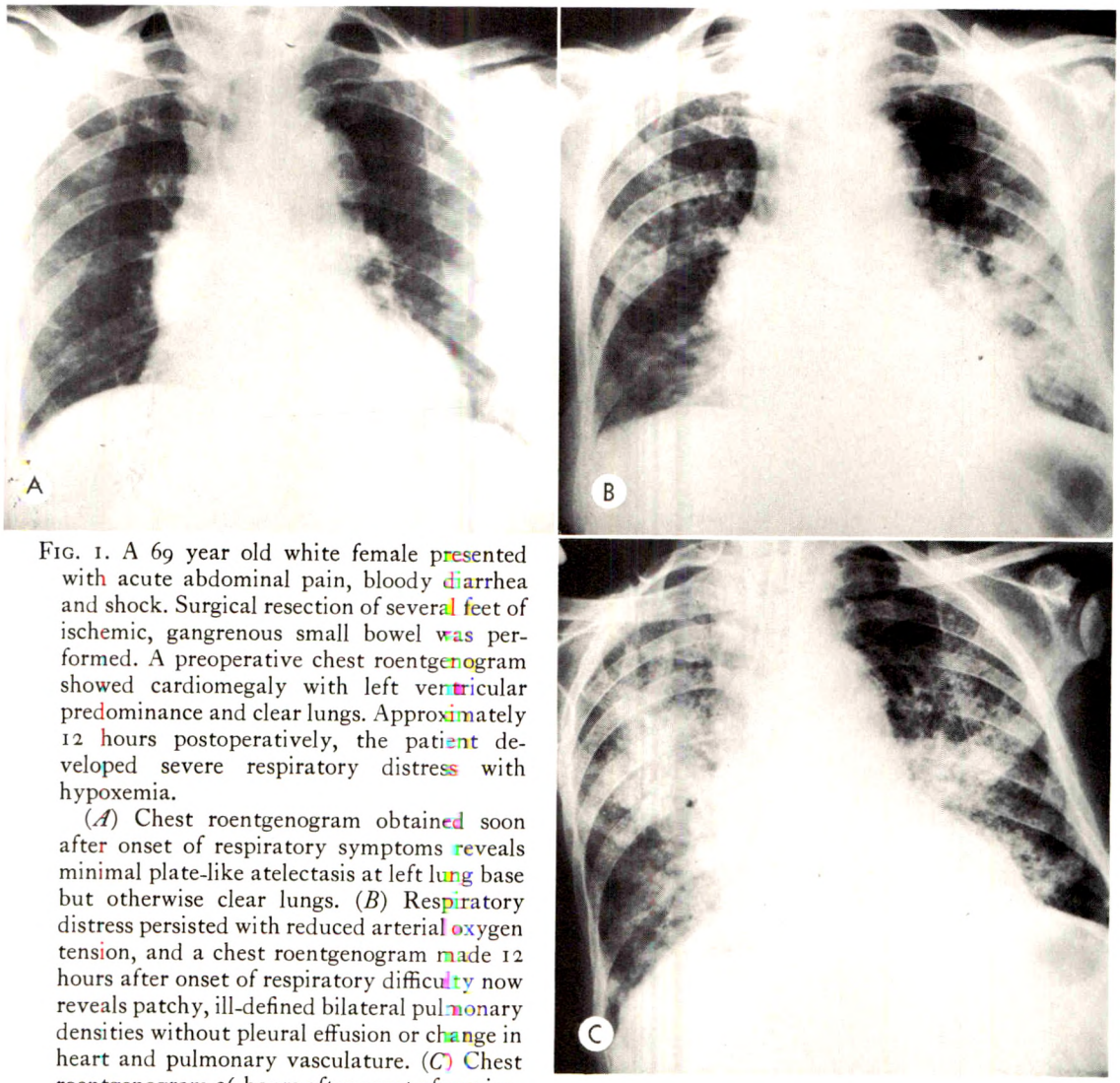


FIG. 1. A 69 year old white female presented with acute abdominal pain, bloody diarrhea and shock. Surgical resection of several feet of ischemic, gangrenous small bowel was performed. A preoperative chest roentgenogram showed cardiomegaly with left ventricular predominance and clear lungs. Approximately 12 hours postoperatively, the patient developed severe respiratory distress with hypoxemia.

(*A*) Chest roentgenogram obtained soon after onset of respiratory symptoms reveals minimal plate-like atelectasis at left lung base but otherwise clear lungs. (*B*) Respiratory distress persisted with reduced arterial oxygen tension, and a chest roentgenogram made 12 hours after onset of respiratory difficulty now reveals patchy, ill-defined bilateral pulmonary densities without pleural effusion or change in heart and pulmonary vasculature. (*C*) Chest roentgenogram 36 hours after onset of respiratory distress reveals marked extension and coalescence of pulmonary densities. Death occurred approximately 12 hours after this roentgenogram.

genogram are strictly related to: (1) the appropriate clinical setting (respiratory distress occurring in a patient subjected to a period of shock from whatever cause, followed by successful resuscitation and a latent period); (2) the specific clinical findings at the time of the chest roentgenogram; and (3) the sequential changes observed on serial chest roentgenograms. If these criteria are strictly applied, a distinct roentgen pattern emerges (Fig. 1, *A–E*; 2, *A–D*; and 3, *A–F*). In all patients there is a delay in the appearance of roentgen changes following the onset of clinical symptoms. Almost invariably the initial chest roentgenogram, obtained at a time when marked tachypnea, dyspnea and cyanosis are clinically evident, appears normal (Fig. 1*A*). In a patient in the appropriate clinical setting this finding in itself is an important early clue to the diagnosis, since massive pulmonary embolism without infarction is possibly the only other condition likely to present with a similar combination of

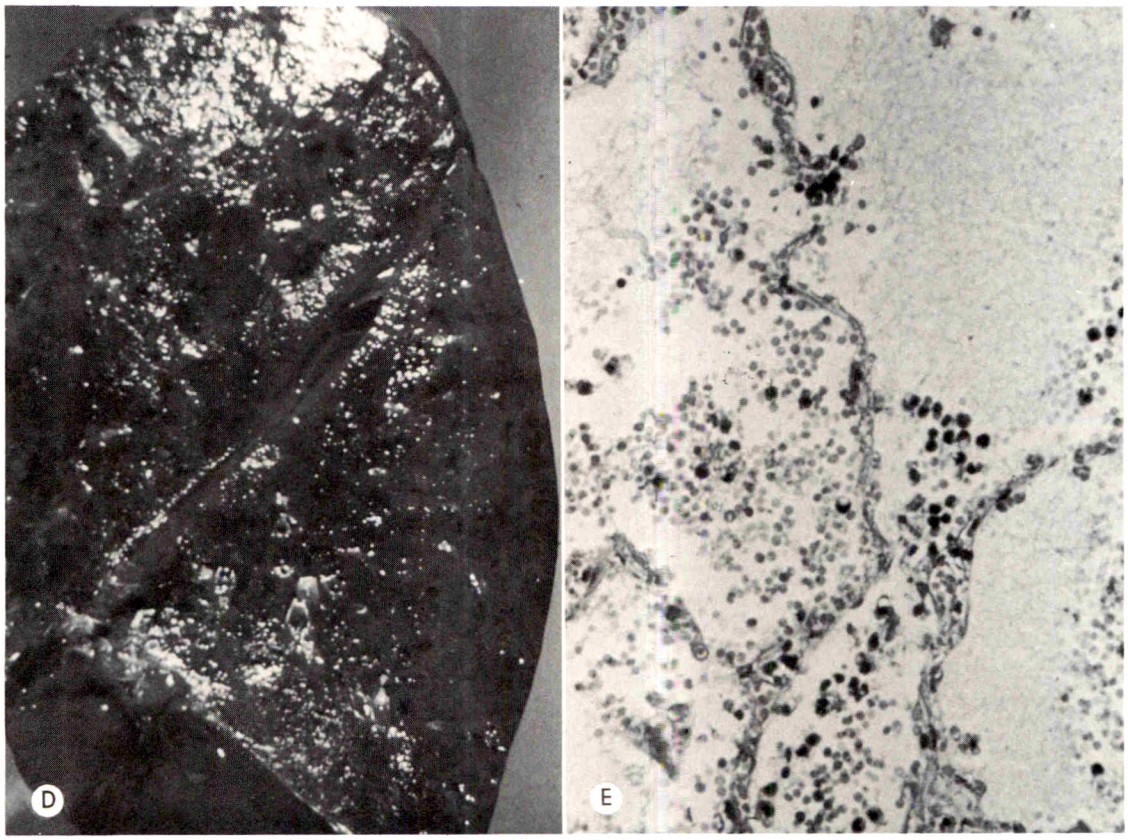


FIG. 1. (*D*) Autopsy specimen of lung showing solid, "liver-like" appearance. Both lungs were heavy, airless and grossly hemorrhagic. (*E*) Microscopic examination reveals capillary congestion, interstitial edema and foci of intra-alveolar edema and hemorrhage.

clinical and roentgenologic findings. Occasionally, there may be roentgen evidence of interstitial edema on the initial roentgenogram, but, in the author's experience, this is very uncommon. In a careful retrospective review, unequivocal roentgen evidence of interstitial edema, especially Kerley lines, was identified in only 5 of the 75 cases. The first definite roentgen abnormality generally appears about 12 to 24 hours after the onset of the clinical syndrome and consists of patchy, ill-defined, bilateral pulmonary densities (Fig. 1*B*; and 3*B*). These may resemble pulmonary edema, but are not accompanied by clinical evidence of heart failure or roentgen evidence of change in heart size and pulmonary vasculature or pleural effusions. If digitalis and/or diuretics are administered there is no rapid improvement in the roentgen picture. Instead, concomitant with a progressive diminution in the arterial oxygen tension, there is a gradual or relatively rapid increase in the extent of the pulmonary densities (Fig. 1*C*), and if death occurs within 24 to 48 hours, pathologic examination reveals heavy, airless, "liver-like" lungs (Fig. 1*D*) with microscopic evidence of capillary congestion, interstitial edema and foci of intra-alveolar edema and hemorrhage (Fig. 1*E*). If the patient survives longer, serial chest roentgenograms reveal further extension and coalescence of the bilateral pulmonary densities which exhibit a predominantly "alveolar" pattern (Fig. 2*B*). If death occurs after 5 to 7 days, pathologic examination reveals heavy, solid, grayish lungs (Fig. 2*C*) with microscopic evidence of prominent hyaline membranes, hyperpla-

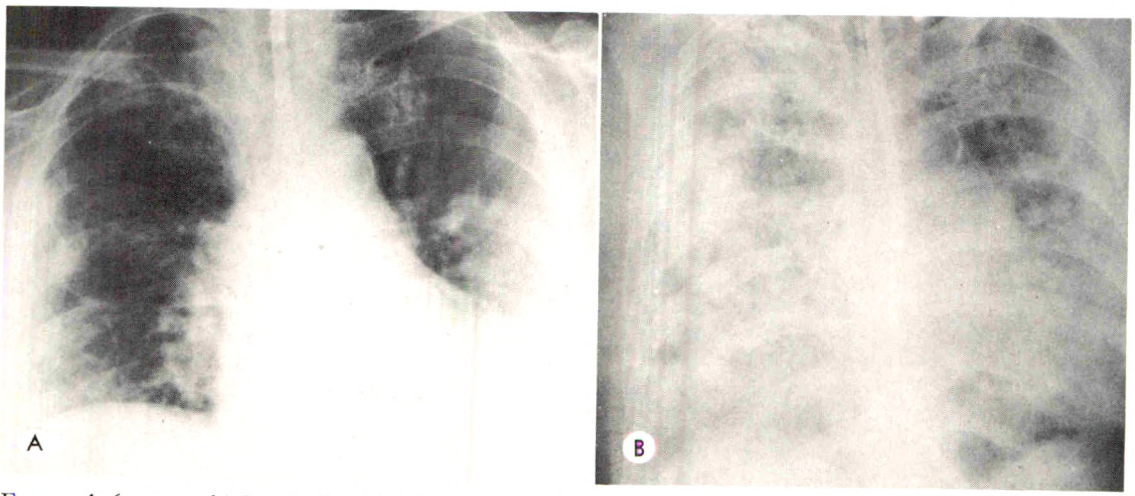


Fig. 2. A 65 year old female involved in an automobile accident sustained severe abdominal injury with ruptured spleen, pelvic fractures and severe shock. Admission chest roentgenogram revealed clear lungs and old, healed fractures of right 5th and 6th ribs. Approximately 18 hours following laparotomy and splenectomy, severe respiratory distress developed. An initial chest roentgenogram was normal.

(*A*) Repeat chest roentgenogram approximately 12 hours after onset of respiratory symptoms reveals bilateral ill-defined patchy densities. Controlled mechanical ventilation was instituted without clinical response. Progressive diminution in arterial oxygen tension and lung compliance with development of arteriovenous shunting required high oxygen concentrations in inspired gas mixture. There was a gradual increase in extent and coalescence of the densities. (*B*) Chest roentgenogram 7 days later reveals total bilateral opacification of lungs.

sia of the alveolar lining cells and intra-alveolar fibrinous exudate (Fig. 2*D*). After about 7 to 10 days the radiologist's main concern is directed towards recognition of complications. These include pulmonary oxygen toxicity, secondary pulmonary sepsis, pneumomediastinum and pneumothorax.

PULMONARY OXYGEN TOXICITY

There is abundant experimental evidence to indicate that the prolonged administration of highly concentrated oxygen is harmful to the lungs of a variety of laboratory animals.[10] Studies in man have been largely confined to observations on healthy volunteers[15] or retrospective analysis.[21] While few prospective studies in humans are available and the conclusions in these somewhat conflicting,[3,26] there is, nevertheless, general agreement that the prolonged administration of oxygen in high concentration is likely to result in severe lung injury. Although oxygen toxicity cannot be implicated as the cause of the initial pulmonary insult in patients with the adult respiratory distress syndrome, it may well aggravate the pulmonary damage when used in concentrations in excess of 50 to 60 per cent for prolonged periods. Roentgenologic abnormalities associated with pulmonary oxygen toxicity have been described in both infants[22] and adults.[13,16] In patients with the adult respiratory distress syndrome, the administration of high concentrations of oxygen may account for a worsening roentgenologic picture and any progressive increase in the extent of the pulmonary densities should alert the radiologist to the possibility of oxygen toxicity. Since the roentgenologic changes *per se* are relatively nonspecific, the possibility of pulmonary oxygen toxicity should only be considered when there is a strict correlation of the roentgen changes with the mode of therapy employed.

PULMONARY SEPSIS

Secondary bacterial infection of the lungs is a frequent complication in patients

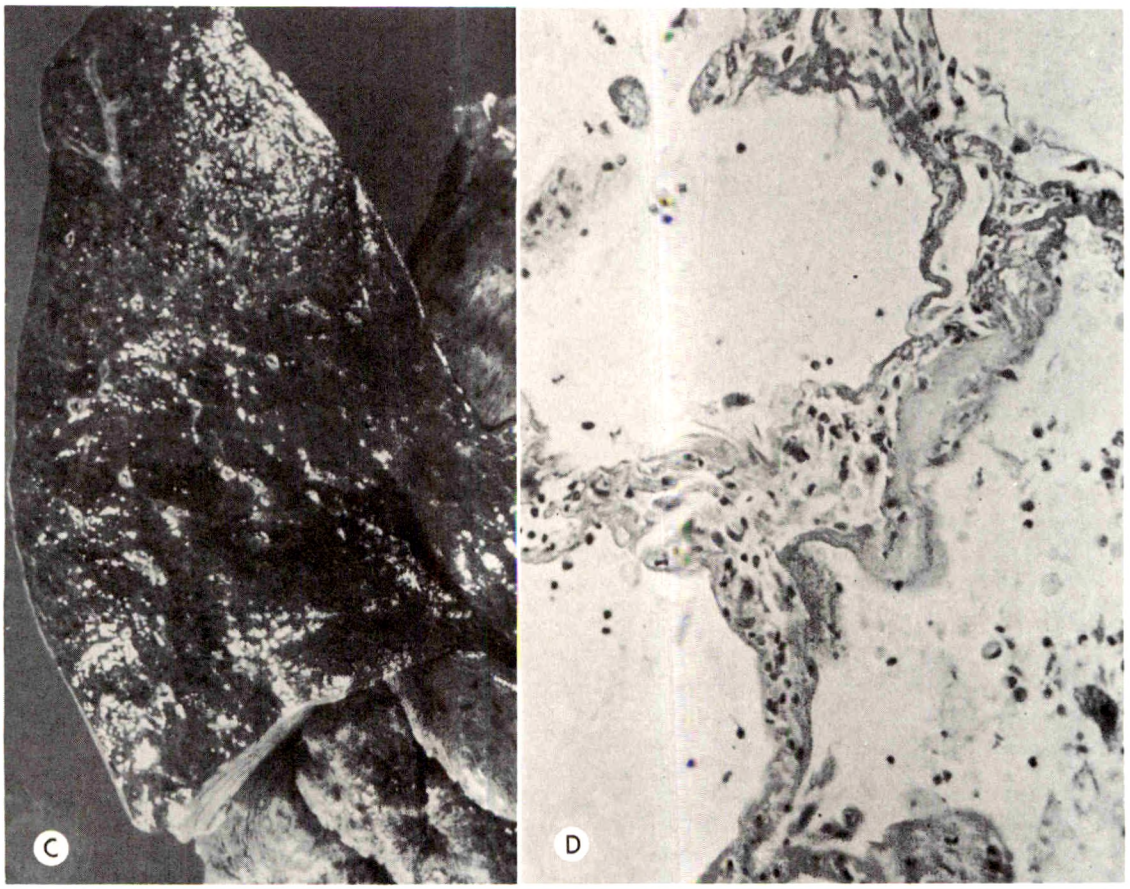


FIG. 2. (*C*) Gross specimen of right lung at autopsy examination. Both lungs were heavy, airless, grayish in color and uniformly solid. (*D*) Photomicrograph showing thickened alveolar septa with prominent hyaline membranes and intra-alveolar fibrinous exudate.

with the adult respiratory distress syndrome who survive more than 7 to 10 days.[17,20] In the vast majority of cases infection is due to hospital-acquired gram negative organisms. In our experience[17,18] *Pseudomonas aeruginosa* is by far the commonest offender, either alone or in combination with *Klebsiella*, *Aerobacter* and *Proteus*. Fungal infections are much less common, but may be a late manifestation in some instances, especially when multiple broad-spectrum antibiotics and corticosteroids have been administered. While various sites such as skin wounds, burns, the urinary tract and vascular catheters may provide the source for organisms, secondary bacterial invasion of the lungs in these patients generally results from colonization of the trachea and is most likely to occur after a tracheostomy has been performed. Knowledge of this background setting should alert the radiologist to the likelihood of secondary bacterial infection as a cause for further deterioration in the roentgenologic picture. The changes observed may consist of: (1) a progression in the extent of the alveolar pulmonary infiltrates (Fig. 3*C*) associated clinically with the development of fever, chills, increased tracheal secretions, polymorphonuclear leukocytosis and repeated positive cultures of the offending organism from the tracheal aspirate; (2) the development of abscess cavities.[18] This represents the most definitive roentgenologic sign of super-added pulmonary infection (Fig. 3*C*) and is not uncommon in gram negative bacterial pneumonias. The cavities may be small or

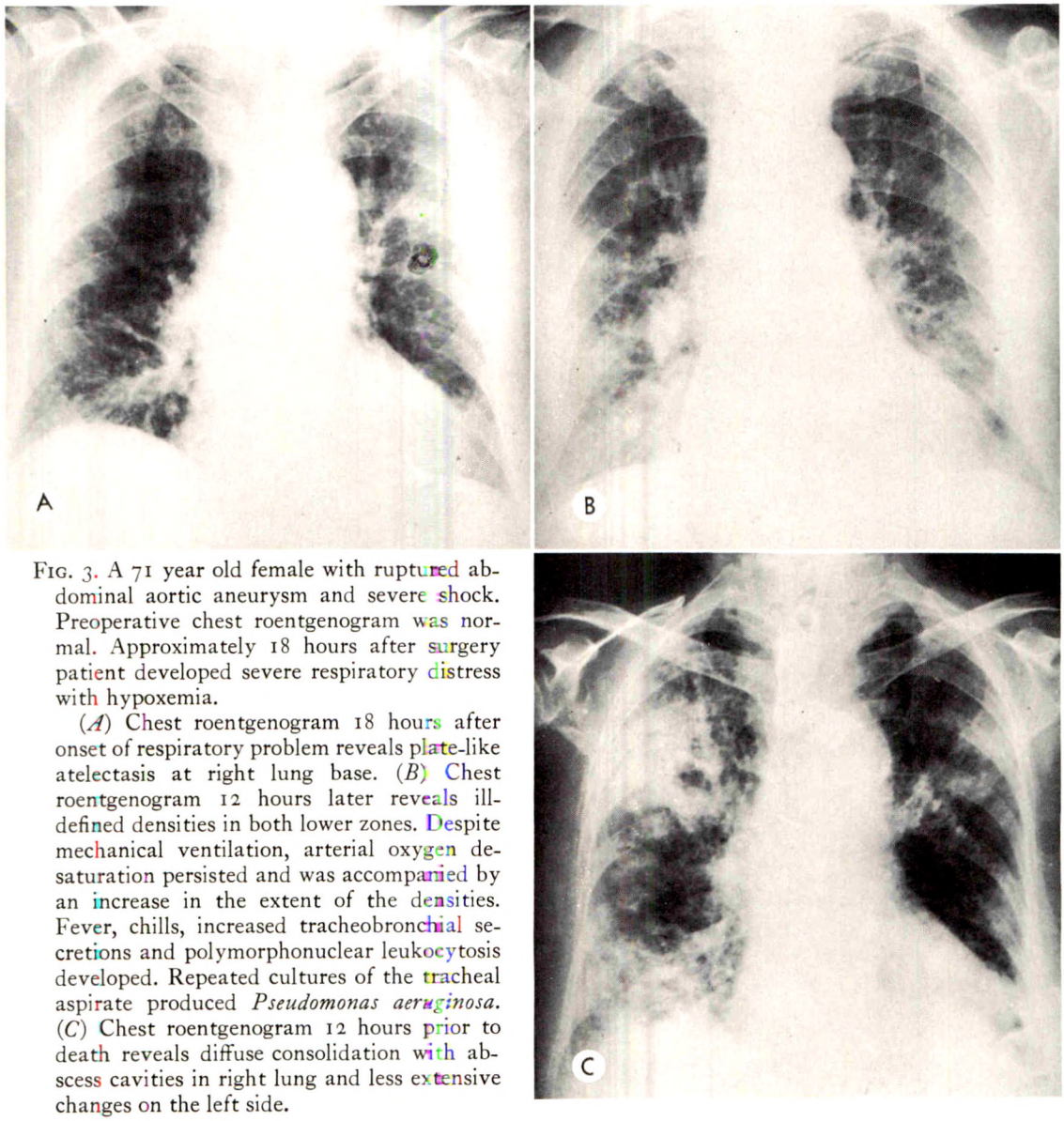


Fig. 3. A 71 year old female with ruptured abdominal aortic aneurysm and severe shock. Preoperative chest roentgenogram was normal. Approximately 18 hours after surgery patient developed severe respiratory distress with hypoxemia.

(*A*) Chest roentgenogram 18 hours after onset of respiratory problem reveals plate-like atelectasis at right lung base. (*B*) Chest roentgenogram 12 hours later reveals ill-defined densities in both lower zones. Despite mechanical ventilation, arterial oxygen desaturation persisted and was accompanied by an increase in the extent of the densities. Fever, chills, increased tracheobronchial secretions and polymorphonuclear leukocytosis developed. Repeated cultures of the tracheal aspirate produced *Pseudomonas aeruginosa*. (*C*) Chest roentgenogram 12 hours prior to death reveals diffuse consolidation with abscess cavities in right lung and less extensive changes on the left side.

large, single or multiple; (3) a mottled or nonhomogeneous appearance of the diffuse lung consolidation (Fig. 4, *A* and *B*). While this finding may be produced by any peripheral air-space consolidation which is superimposed on chronic obstructive lung disease—the so-called "swiss-cheese" appearance—its development in lungs which were previously normal or the site of homogeneous opacification should arouse strong suspicion of secondary gram negative bacterial pneumonia, especially that due to *Pseudomonas aeruginosa*. At autopsy, such lungs generally exhibit numerous necrotic nodules (Fig. 3*D*) or widespread micro-abscesses; (4) the development of pleural effusion (empyema). The diffuse pulmonary densities which occur in patients with the adult respiratory distress syndrome are characteristically unaccompanied by pleural effusions, and the development of a pleural effusion should raise the possibility of a complication such as pulmonary infection with empyema, pul-

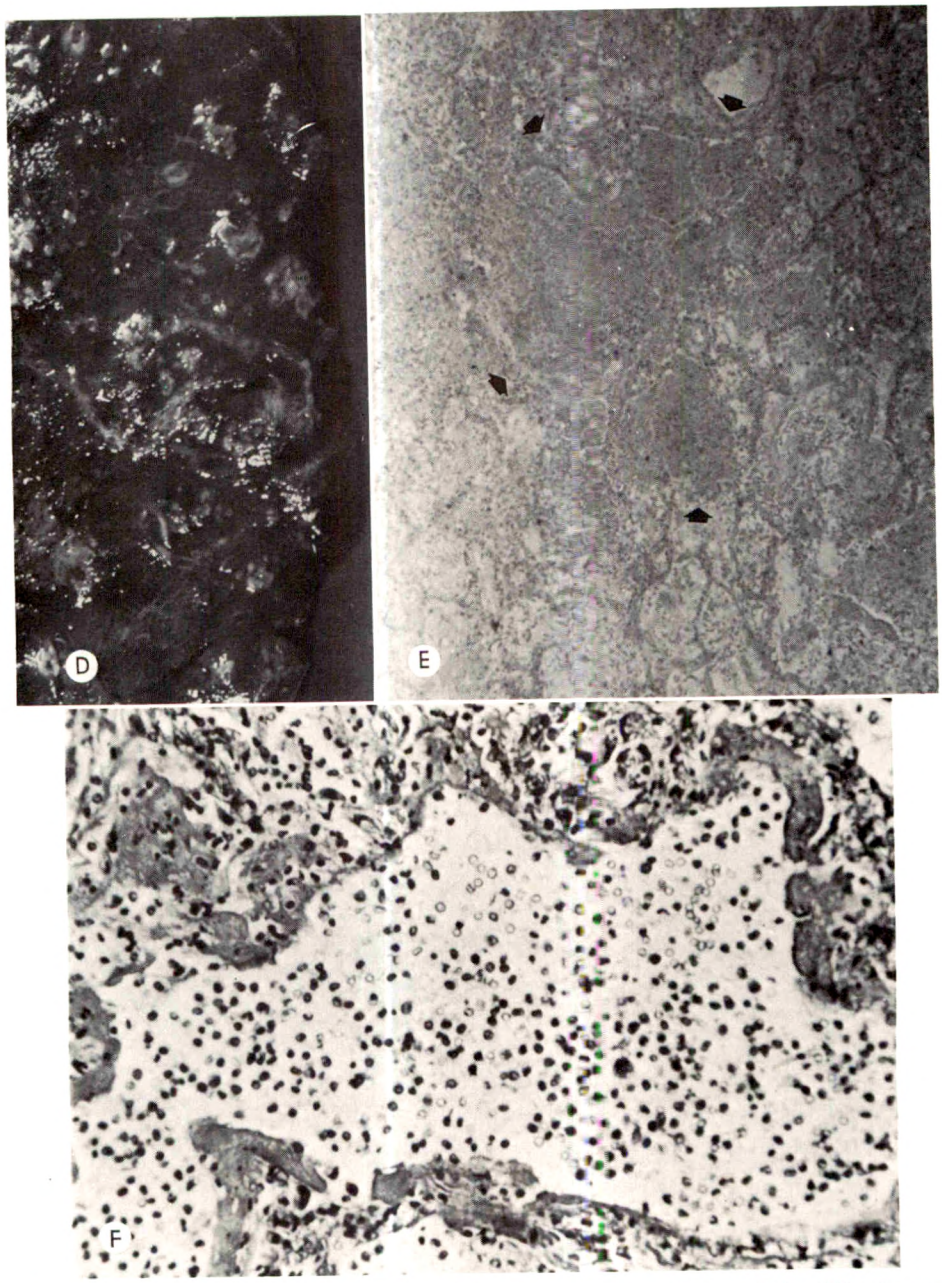


FIG. 3. (*D*) Autopsy specimen of lung showing numerous necrotic nodules within diffusely consolidated, airless lung. (*E*) Microscopic examination reveals large area of necrosis (arrows), thickened alveolar septa, hyaline membranes and fibroblastic proliferation. (*F*) High power photomicrograph showing diffuse infiltrate of polymorphonuclear cells in addition to the basic pathologic picture seen in Figure 2*D*.

monary embolism with infarction or cardiac failure.

PNEUMOMEDIASTINUM, PNEUMOTHORAX AND INTERSTITIAL PULMONARY EMPHYSEMA

Since loss of compliance of the lungs is such a prominent feature in the adult respiratory distress syndrome, high ventilator pressures are often required to obtain satisfactory arterial oxygenation. While pneumomediastinum and pneumothorax are well-recognized complications of inter-

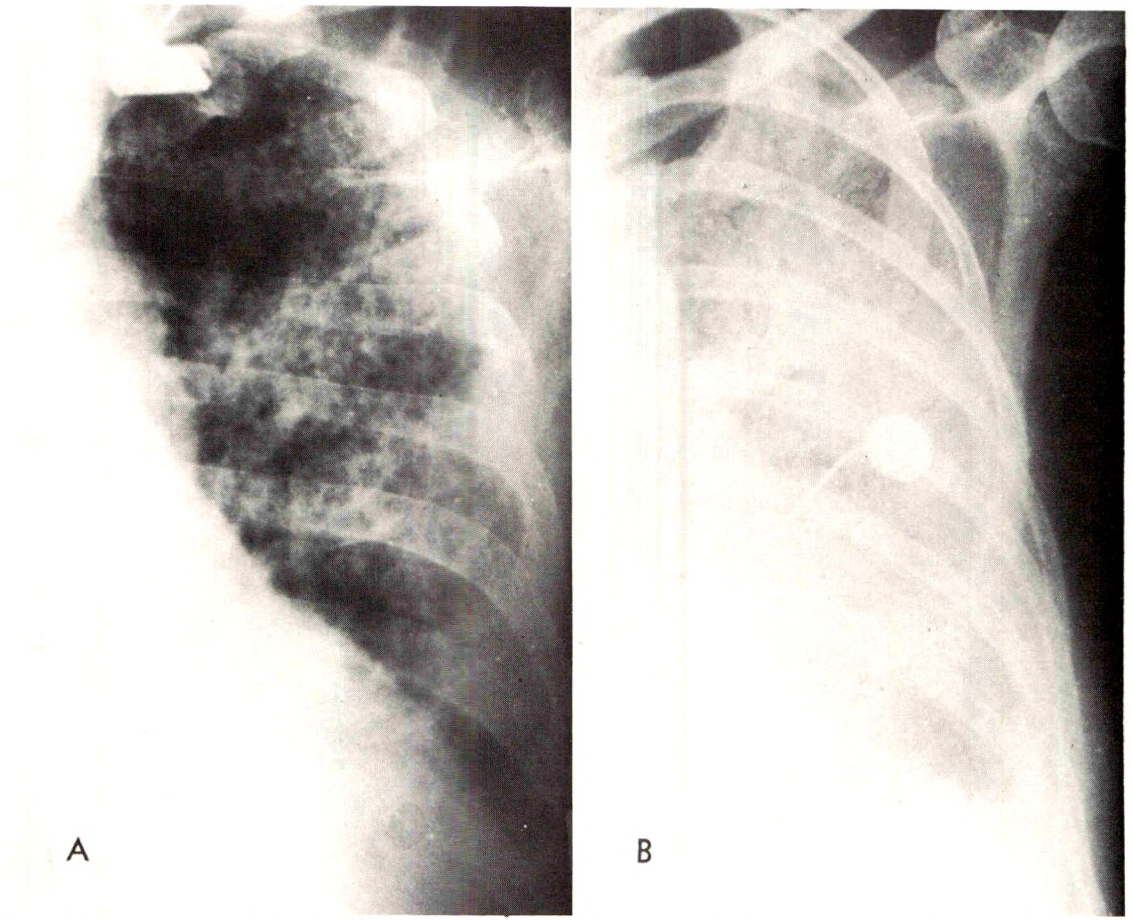


Fig. 4. (*A*) Note mottled, non-homogeneous pattern of pulmonary densities commonly seen with gram negative pneumonia. Compare with (*B*) which shows uniform, homogeneous opacification produced by alveolar pulmonary edema or various pneumonias not due to gram negative organisms. Changes seen in *A* may, however, be produced by any peripheral air-space consolidation superimposed on severe chronic obstructive lung disease.

mittent positive pressure breathing in these patients, there has been a marked increase in the incidence of pneumomediastinum and pneumothorax following the introduction and widespread use of continuous positive pressure ventilation with PEEP. The possibility of pneumothorax, especially tension pneumothorax, should always be considered when there is a sudden marked deterioration in the patient's condition (Fig. 5). Interstitial pulmonary emphysema may accompany or precede the pneumomediastinum or pneumothorax. Because of the diffuse opacification of the lungs in the adult respiratory distress syndrome, interstitial pulmonary emphysema may be clearly visible as it produces numerous rounded or irregular radiolucencies within the diffusely consolidated areas and is sometimes associated with fairly large subpleural air "cysts" (Fig. 6, *A–C*; and 7, *A* and *B*). Such air cysts may be transient in nature, tending to resolve rapidly, but occasionally they may continue to expand and give rise to large tension cavities (Fig. 7*B*). Roentgenologic recognition of interstitial pulmonary emphysema is important, as it is often a sign of impending pneumomediastinum and/or pneumothorax (Fig. 6*C*). It may simulate the mottled radiodensities and radiolucencies produced by gram negative pneumonias, or, when ex-

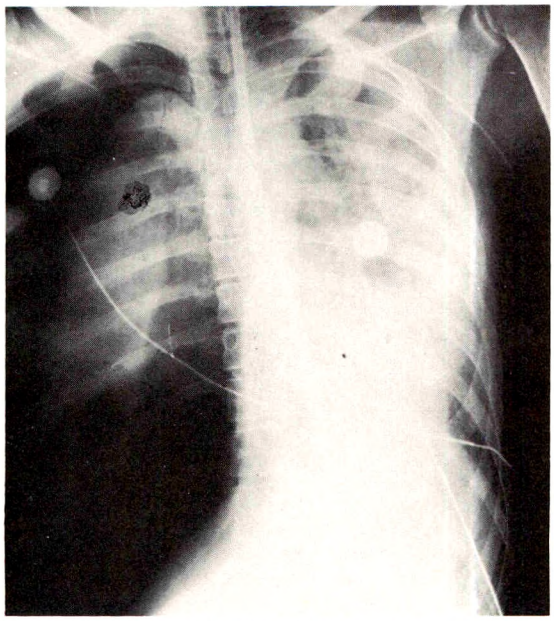
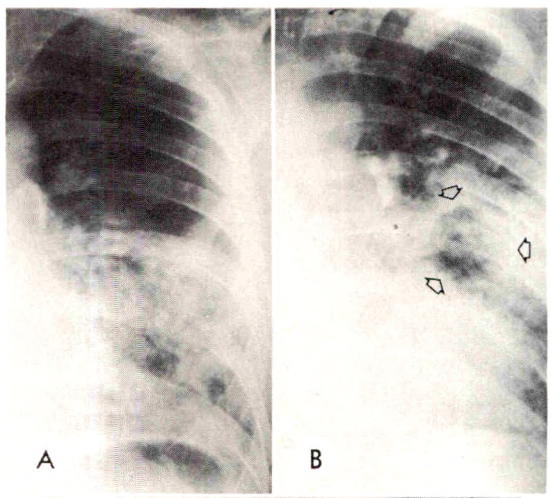


FIG. 5. Patient with the adult respiratory distress syndrome following drug overdosage developed sudden, marked deterioration following institution of continuous positive pressure breathing with positive end expiratory pressure. Note right-sided tension pneumothorax in addition to bilateral pulmonary opacification.

tensive and occurring within a diffusely consolidated area, may give rise to a false impression of roentgenologic improvement at a time when there is clinical evidence of deterioration in the patient's condition (Fig. 6*C*).

ROENTGENOLOGIC DIFFERENTIAL DIAGNOSIS

A fairly large number of conditions may be responsible for acute respiratory distress in patients who have been subjected to massive trauma, major surgery or critical medical illness. These include: pulmonary edema secondary to cardiac failure; overzealous intravenous fluid administration or head injury; massive aspiration; pulmonary contusion or hemorrhage; primary infection of the lungs; atelectasis due to mucus plugs or blood; pulmonary thromboembolism; fat embolism; and pneumothorax.



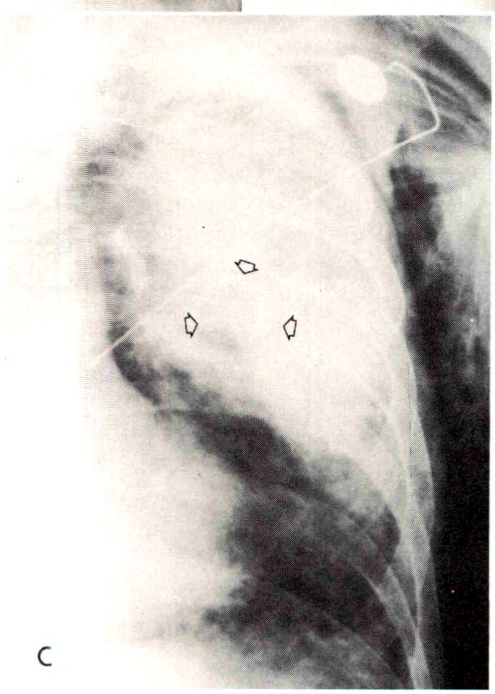


FIG. 6. A 28 year old male with acute respiratory distress following acute hemorrhagic pancreatitis. Serial chest roentgenograms revealed progressive opacification of both lungs.

(*A*) Localized view of the left lung 3 days after institution of continuous positive pressure ventilation and positive end expiratory pressure showing diffuse pulmonary density associated with small radiolucencies suggestive of interstitial pulmonary emphysema. (*B*) Same lung 24 hours later showing development of large subpleural air cyst (arrows) in lingula. (*C*) At 36 hours there is extensive interstitial pulmonary emphysema in the lower zone giving the false impression of clearing of consolidation. In addition there is now mediastinal and subcutaneous emphysema. Note lateral displacement of calcified lymph node. The air cyst (arrows) is still present and the left lobe is now totally opacified.

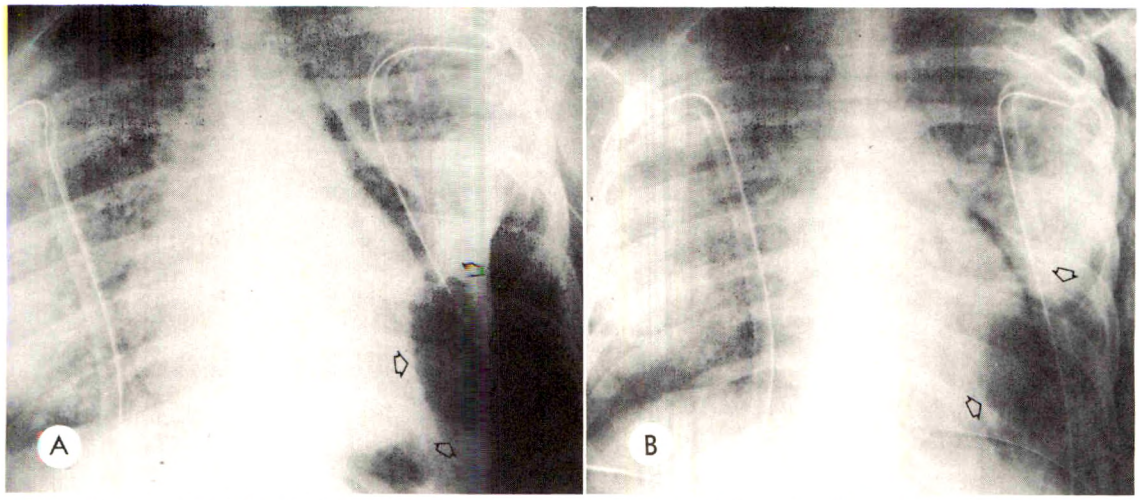


FIG. 7. A 30 year old female developed acute respiratory distress with peritonitis and shock following self-induced abortion. Three days after the institution of continuous positive pressure breathing and positive end expiratory pressure the patient developed bilateral pneumothoraces.

(*A*) Chest roentgenogram after introduction of bilateral chest tubes shows diffuse opacification of lungs and residual left-sided pneumothorax. Note large air cyst (arrows) in left lower lobe. (*B*) Chest roentgenogram 48 hours later showing small residual left pneumothorax, subcutaneous emphysema and marked expansion of air cyst (arrows) in left lower lobe.

Some of these conditions such as pneumothorax and atelectasis generally offer no roentgenologic diagnostic problem.

The more important conditions to be considered in the roentgenologic differential diagnosis of the adult respiratory distress syndrome are:

(1) *Pulmonary edema.* The roentgenologic changes produced by acute pulmonary edema may be indistinguishable from those observed in the adult respiratory distress syndrome. Differentiation is largely based upon a correlation of the clinical and roentgenologic findings and the response to therapy. Acute pulmonary edema is characterized by its variability and rapid change in extent and distribution of the pulmonary densities with therapy or change in position of the patient, whereas in the adult respiratory distress syndrome there is generally a progressive increase in the extent of the pulmonary densities despite treatment for pulmonary edema. Serial chest roentgenograms are frequently necessary in order to make the differentiation. The presence of cardiomegaly, pulmonary vascular engorgement and pleural effusion will favor a diagnosis of pulmonary edema.

(2) *Massive aspiration.* Acute respiratory distress associated with roentgenologic evidence of bilateral pulmonary densities may occur as a result of the massive aspiration of blood or acid gastric contents, especially in patients who are comatosed or semi-comatosed as a result of trauma, surgery or medical illness. However, the pulmonary densities are almost invariably present on the initial chest roentgenogram (Fig. 8) and this finding taken in conjunction with the appropriate clinical history will usually indicate the correct diagnosis. If the roentgenologic abnormalities appear relatively late in the course of the clinical syndrome and after the patient has been intubated with a cuffed endotracheal tube, it is unlikely that massive aspiration can be held responsible for the roentgen changes.

(3) *Primary pulmonary infection.* Localized unilateral infiltrates due to primary pulmonary infection generally provide no difficulty in differential diagnosis. Bilateral, patchy or confluent pulmonary densities may be produced by a variety of primary pneumonias. The diagnosis is generally

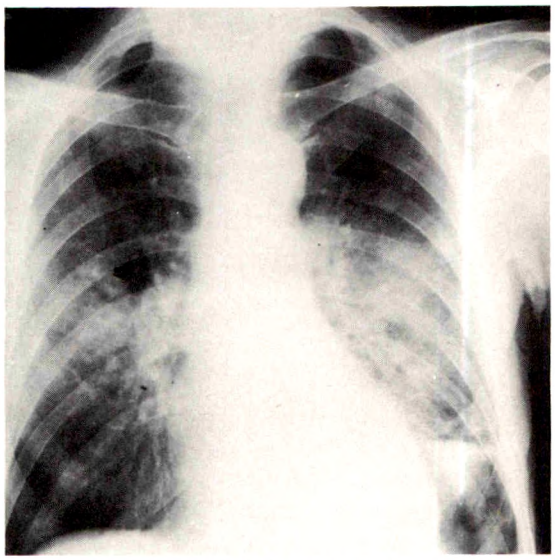


FIG. 8. A 56 year old alcoholic male was transferred to the intensive care unit with a history of acute gastrointestinal hemorrhage, vomiting and respiratory distress.

Chest roentgenogram reveals an elevated left hemidiaphragm secondary to gas-distended bowel and bilateral ill-defined pulmonary densities. The history and the presence of the densities on the initial chest roentgenogram strongly suggest a diagnosis of massive aspiration. Note absence of nasogastric and endotracheal tubes.

established by a correlation of the roentgenologic findings with clinical evidence of fever, sputum production, laboratory data, culture of the organisms from the sputum and response to antibiotic therapy. Secondary bacterial infection of the lungs is very common in the adult respiratory distress syndrome but it generally develops relatively late in the course of the syndrome and its roentgenologic aspects have been previously discussed.

(4) *Pulmonary contusion and hemorrhage.* Contusion of the lung secondary to direct trauma may produce roentgen evidence of pulmonary densities. Diagnosis is based upon the history of trauma and the localized nature of the densities which are usually present on the initial chest roentgenogram or appear very soon thereafter, and which tend to clear relatively rapidly. Severe blast or crush injuries of the thorax may result in diffuse, bilateral pulmonary densities secondary to hemorrhage and/or edema. Such cases may proceed with a clinical and roentgenologic course identical to that seen in the adult respiratory distress syndrome secondary to nonthoracic trauma.

(5) *Pulmonary thromboembolism.* The bed-ridden injured, post-surgical or critically ill patient is not only a prime candidate for the development of the adult respiratory distress syndrome but also faces the hazards of phlebothrombosis and pulmonary embolism. This possibility should always be considered when respiratory distress develops in such a patient. Pulmonary embolism without infarction may be associated with an essentially normal chest roentgenogram and thus constitutes the most important differential diagnostic possibility in the early phase of the adult respiratory distress syndrome. Careful scrutiny of the initial chest roentgenogram obtained following the onset of respiratory distress may reveal one or more areas of pulmonary oligemia with or without associated dilatation of the right ventricle and main pulmonary artery segment. In such cases a radionuclide lung scan and/or pulmonary angiogram may be required for confirmation or exclusion of pulmonary thromboembolism. If infarction occurs as a result of thromboembolism, the chest roentgenogram is likely to reveal one or more pleural-based densities associated with pleural effusion and in such cases differentiation from the adult respiratory distress syndrome is generally not difficult.

(6) *Fat embolism.* Pulmonary fat embolism may give rise to a clinical syndrome of respiratory insufficiency indistinguishable from the adult respiratory distress syndrome. While pathologic evidence of fat globules in the pulmonary capillaries is common following major trauma,[11,25] the incidence of the clinical fat embolism syndrome is much less frequent and there is still some controversy regarding the exact pathophysiologic mechanism of pulmonary fat embolism. Recent studies[9,29] suggest toxicity of free fatty acids as the primary causal factor producing damage to the capillary endothelium and alveolar septa

with resultant intra-alveolar hemorrhage, interstitial and intra-alveolar edema. A clinical background of major bone or soft tissue injury and a delayed onset of respiratory distress following injury and resuscitation is common to both syndromes. The roentgenologic findings in pulmonary fat embolism are largely due to intra-alveolar hemorrhage and edema and as such consist of diffuse, bilateral ill-defined densities (Fig. 9) without pleural effusion and with a normal cardiac silhouette and pulmonary vascular pattern.[9] The densities have no specific distribution and are essentially the same as those seen in the adult respiratory distress syndrome. The diagnosis of the fat embolism syndrome is largely based upon a clinical complex consisting of a combination of pulmonary, systemic and neurologic symptoms and signs associated with a petechial rash, thrombocytopenia and the presence of fat in the urine.

ETIOLOGY

The precise etiology of the initial pulmonary injury in patients with the adult respiratory distress syndrome is still undetermined and a detailed discussion of the various hypotheses is beyond the scope of the present paper. The stereotyped pathophysiologic changes which occur after many different causes suggest that either there is a common mechanism or the ability of the lung to react to different insults is very limited. Most workers feel that damage to the pulmonary microvasculature represents the initial change, but the exact site and causes are still disputed and have been variously ascribed to ischemic damage during shock, release of vasoactive substances such as catecholamines, histamine, serotonin, prostaglandins, kinins or vasoactive peptides from injured tissues or blood constituents.[7,28] Some investigators are strong proponents of the theory that disseminated intravascular coagulation is a major contributory factor.[6,14,24] Oxygen toxicity, surfactant deficit and overhydration during resuscitation have also been incriminated, but, although all of these factors may play a role in aggravating the pulmonary damage, it is unlikely that they represent the initiating pulmonary insult in the adult respiratory distress syndrome.

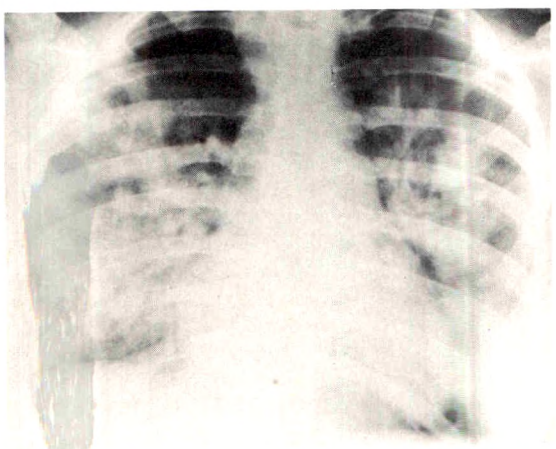


Fig. 9. A 29 year old female sustained bilateral femoral and tibial fractures in an automobile accident. Approximately 20 hours after immobilization of the fractures she developed respiratory distress with tachypnea, dyspnea and cyanosis. Soon thereafter she became irritable, confused and stuporous. Physical examination revealed a petechial rash on the chest and neck and fat was noted in the urine.

Chest roentgenogram shows extensive bilateral, pulmonary densities.

Autopsy revealed extensive intra-alveolar edema and hemorrhage secondary to pulmonary fat embolism.

SUMMARY

The term "adult respiratory distress syndrome" is used to describe a serious and often fatal condition which may develop in any patient subjected to massive trauma, major surgery or serious medical illness.

Despite the obscure nature of the primary pulmonary insult, the syndrome presents a constant and stereotyped clinical, roentgenologic and pathologic picture.

In the present paper a brief outline of the clinical and pathologic features is given and the roentgenologic findings and differential diagnosis are discussed in some detail with illustrative examples selected from a large series of cases seen during a 6 year period.

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REFERENCES

1. ASHBAUGH, D. G., BIGELOW, D. B., PETTY, T. L., and LEVINE, B. E. Acute respiratory distress in adults. *Lancet*, 1967, *2*, 319–323.
2. AYRES, S. M., MUELLER, H., GIANNELLI, S., JR., FLEMING, P., and GRACE, W. J. Lung in shock: alveolar capillary gas exchange in shock syndrome. *Am. J. Cardiol.*, 1970, *26*, 588–594.
3. BARBER, R. E., LEE, J., and HAMILTON, W. K. Oxygen toxicity in man: prospective study in patients with irreversible brain damage. *New England J. Med.*, 1970, *283*, 1478–1484.
4. BERGOFSKY, E. H. Adult acute respiratory insufficiency syndrome following non-thoracic trauma: lung in shock. *Am. J. Cardiol.*, 1970, *26*, 619–621.
5. BERRIGAN, T. J., JR., CARSKY, E. W., and HEITZMAN, E. R. Fat embolism: roentgenographic-pathologic correlation in 3 cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 967–971.
6. BLAISDELL, F. W., LIM, R. C., JR., and STALLONE, R. J. Mechanism of pulmonary damage following traumatic shock. *Surg., Gynec. & Obst.*, 1970, *130*, 15–22.
7. BLAISDELL, F. W., and SCHLOBOHM, R. M. Respiratory distress syndrome: review. *Surgery*, 1973, *74*, 251–262.
8. BREDENBERG, C. E., JAMES, P. M., COLLINS, J., ANDERSON, R. W., MARTIN, A. M., JR., and HARDAWAY, R. M., III. Respiratory failure in shock. *Ann. Surg.*, 1969, *169*, 392–403.
9. BRUECK, P., BURKE, J. F., and LAM, K. W. Pathophysiology of pulmonary fat embolism. *J. Thoracic & Cardiovasc. Surg.*, 1971, *61*, 949–955.
10. CLARK, J. M., and LAMBERTSEN, C. J. Pulmonary oxygen toxicity: review. *Pharmacol. Rev.*, 1971, *23*, 37–133.
11. GLAS, W. W., GREKIN, T. D., and MUSSELMAN, M. M. Fat embolism. *Am. J. Surg.*, 1953, *85*, 363–368.
12. GOMEZ, A. C. Pulmonary insufficiency in non-thoracic trauma. *J. Trauma*, 1968, *8*, 656–665.
13. HALL, R. M., and MARGOLIN, F. R. Oxygen alveolopathy in adults. *Clin. Radiol.*, 1972, *23*, 11–17.
14. HARDAWAY, R. M., JAMES, P. M., JR., ANDERSON, R. W., BREDENBERG, C. E., and WEST, R. L. Intensive study and treatment of shock in man. *J.A.M.A.*, 1967, *199*, 779–790.
15. HELVEY, W. M. Problem of man and milieu: prolonged exposure to pure oxygen. *Fed. Proc.*, 1963, *22*, 1057–1059.
16. JOFFE, N., and SIMON, M. Pulmonary oxygen toxicity in the adult. *Radiology*, 1969, *92*, 460–465.
17. JOFFE, N. Roentgenologic findings in post-shock and postoperative pulmonary insufficiency. *Radiology*, 1970, *94*, 369–375.
18. JOFFE, N. Roentgenologic aspects of primary *Pseudomonas aeruginosa* pneumonia in mechanically ventilated patients. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 305–312.
19. MARTIN, A. M., JR., SOLOWAY, H. B., and SIMMONS, R. L. Pathologic anatomy of lungs following shock and trauma. *J. Trauma*, 1968, *8*, 687–698.
20. MOORE, F. D., LYONS, J. H., JR., PIERCE, E. C., JR., MORGAN, A. P., DRINKER, P. A., MACARTHUR, J. D., and DAMMIN, G. J. Post-traumatic Pulmonary Insufficiency. W. B. Saunders Company, Philadelphia, 1969.
21. NASH, G., BLENNERHASSETT, J. B., and PONTOPPIDAN, H. Pulmonary lesions associated with oxygen therapy and artificial ventilation. *New England J. Med.*, 1967, *276*, 368–374.
22. NORTHWAY, W. H., JR., ROSAN, R. C., and PORTER, D. Y. Pulmonary disease following respiratory therapy of hyaline membrane disease: bronchopulmonary dysplasia. *New England J. Med.*, 1967, *276*, 357–368.
23. ORELL, S. R. Lung pathology in respiratory distress following shock in adult. *Acta path. et microbiol. scandinav.*, 1971, *79*, 65–76.
24. PUTNAM, C. E., MINAGI, H., and BLAISDELL, F. W. Roentgen appearance of disseminated intravascular coagulation (DIC). *Radiology*, 1972, *109*, 12–18.
25. SEVITT, S. Significance and classification of fat embolism. *Lancet*, 1960, *2*, 825–828.
26. SINGER, M. M., WRIGHT, F., STANLEY, L. K., BENSON, B. R., and HAMILTON, W. K. Oxygen toxicity in man: prospective study in patients after open-heart surgery. *New England J. Med.*, 1970, *283*, 1473–1478.
27. UNGER, J. DE B., ROSE, H. D., and UNGER, G. F. Gram negative pneumonia. *Radiology*, 1973, *107*, 283–291.
28. WEBB, W. R. Pulmonary complications of non-thoracic trauma: summary of National Research Council conference. *J. Trauma*, 1969, *9*, 700–711.
29. WEISZ, G. M., and STEINER, E. Cause of death in fat embolism. *Chest*, 1971, *59*, 511–516.
30. WILSON, R. F., KAFI, A., ASUNCION, Z., and WALT, A. J. Clinical respiratory failure after shock or trauma: prognosis and methods of diagnosis. *A.M.A. Arch. Surg.*, 1969, *98*, 539–549.

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PULMONARY COMPLICATIONS OF NARCOTIC ABUSE*

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DRUG abuse has, within the past decade, become a major medical and social problem. With the development of newer drugs, the clinical presentation and subsequent management of the overdose patient will present an even greater diagnostic challenge.

Although the field of toxicology has made notable advances, the physician in many circumstances will be unable to rely solely on this modality for a specific diagnosis in drug overdosage. The majority of hospitals are not equipped to perform these tests and, even if the equipment and personnel were available, the time lag in obtaining the results is not realistic with regards to the clinical situation. Also, laboratory values may often be misleading due to possible additive or synergistic effects, even if the individual drug is present in less than toxic levels.

However, the chest roentgenogram is often the initial clue to the altered pathophysiology and reveals the cause of the patients respiratory difficulty. The purpose of this study is to document the lung roentgenographic changes associated with the various forms of drug abuse and to determine to what extent the radiologist can aid the clinician in both the diagnosis and eventual prognosis of these patients.

MATERIAL AND METHOD

We reviewed the admissions to our intensive care unit for drug overdoses during the period of 1970–1973. A total of 72 patient records was reviewed. All roentgenograms were interpreted independently by 2 staff radiologists. Clinically, pulmonary edema was established by the physicians observations and/or auscultatory findings. Patients were selected from this group if the following criteria were satisfied: (1) no complicating medical history, *i.e.*, congestive heart failure, convulsions, etc.; (2) adequate identification of drug used in overdose; (3) satisfactory clinical records; and (4) initial chest roentgenograms within 1–2 hours of admission and adequate follow-up chest roentgenograms.

RESULTS

(Table 1)

By applying the above criteria, 45 patients were judged suitable for our study. Twenty-four were admitted with overdoses of barbiturates, tranquilizers and other sedatives. Their clinical presentation ranged from a mild respiratory depression and dull sensorium to an apneic, cyanotic and comatose condition. None of these patients had either clinical or roentgenographic signs of pulmonary edema on their initial chest roentgenogram or subsequent roentgenograms. Three patients presented with or developed clinical and roentgenographic signs of aspiration pneumonia which resolved over a 3–10 day interval.

The remaining 21 patients had documented overdoses of heroin, solely or in combination with methadone. Eight of these patients (38 per cent), all of whom were initially apneic, had admission chest roentgenograms which demonstrated pulmonary edema. The pattern of pulmonary edema ranged from the classic "bat-wing" perihilar, alveolar appearance, to a prominent interstitial pattern.

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TABLE I

RESULTS IN TOTAL OF 45 PATIENTS

Drug	No. of Patients	Pulmonary Edema Roentgenographically	Pulmonary Edema Clinically	Resolution Time	Complications
Barbiturates, etc.	24	0	0	—	Aspiration pneumonia (3)
Heroin with or without methadone	21	8	5	24–48 hours	Bacterial and/or aspiration pneumonia (3)

Only 5 had substantial clinical findings to support the diagnosis of pulmonary edema, the remaining three having suggestive physical findings. Nevertheless, all 8 patients showed roentgenographic resolution of the pumonary edema within 24–48 hours of admission (Fig. 1, *A* and *B*). Clinical improvement preceded the roentgenographic resolution in all patients. The 3 patients who had only roentgenographic signs of pulmonary edema had definite clearing of their initial prominent interstitial pattern on subsequent studies. Three patients with clinical and roentgenographic evidence of pulmonary edema developed either a localized aspiration or bacterial pneumonia, although no diagnostic blood or sputum cultures were obtained. The roentgenograms of these patients showed gradual improvement over a period of 3–10 days, but the initial roentgenographic and florid clinical presentation of pulmonary edema, nevertheless, resolved within 24–48 hours. Central venous pressure (CVP) measurements when recorded (2 of the 8 patients) were never above 5 cm. H_2O.

DISCUSSION

The patient with a drug overdose, whether narcotic or non-narcotic, often presents clinically either comatose or semicomatose, with miotic pupils and a marked respiratory depression.[5,6,17] Treatment frequently consists of the use of Nalline (nalorphine hydrochloride) or Narcan (naloxone hydrochloride), oxygen therapy and intubation with mechanical ventilatory assistance. The decision to use either Nalline or Narcan is clinically important. Although both drugs may have dramatic awakening effects when used in heroin overdoses, Nalline can antagonize the respira-

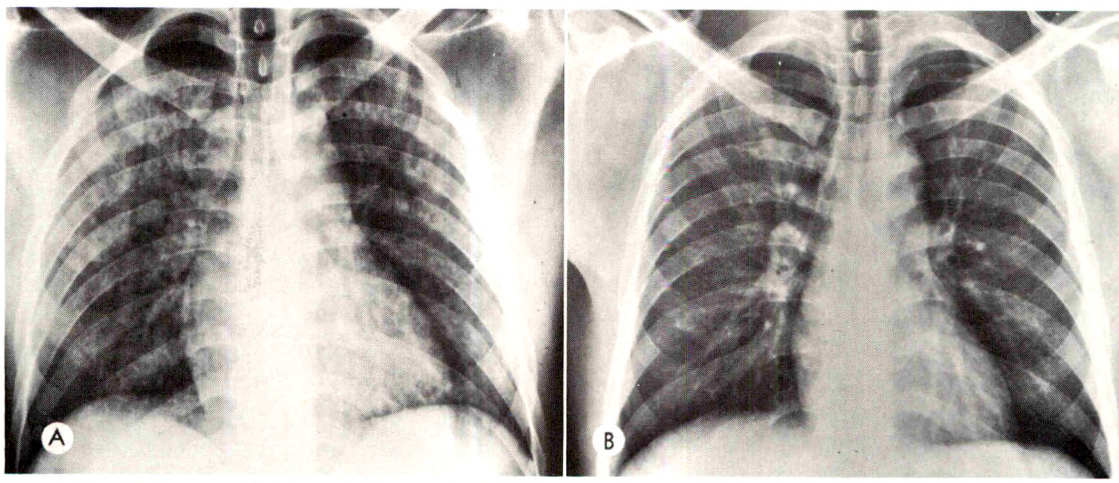


FIG. 1. Rapid clearing of pulmonary edema in a drug addict. (*A*) Anteroposterior supine chest roentgenogram taken on admission showing moderate pulmonary edema. (*B*) Upright posteroanterior chest roentgenogram taken 40 hours later. The chest is now normal.

tory depression in barbiturate and cocaine overdoses.

Aspiration pneumonia occurs frequently in this clinical situation and is a result of an absent or decreased cough reflex due to the central nervous system (CNS) depression. Pulmonary edema, which occurs in overdoses involving heroin, methadone and Darvon (propoxyphene hydrochloride), has many proposed etiologies, the more tenable being: (1) severe hypoxia; (2) increased capillary permeability secondary to a direct toxic effect on the endothelial cell layer of the pulmonary capillaries: (3) myocardial stress secondary to either a direct toxic effect by a contaminant or to the severe hypoxia; (4) allergic or idiosyncratic reaction to the drug; or (5) hypersensitivity reaction to a contaminant.

Considering increased capillary permeability secondary to a direct toxic effect, Frand and associates[5] have shown that the edema fluid obtained from some of these patients had a protein composition similar to plasma. Certain chemical poisons such as α-naphthyl thiourea, war gases and sodium deoxycholate can affect the capillary permeability. This results in increased protein in the edema fluid, supposedly by a direct toxic effect on the alveolar-capillary membrane.[1,4] However, the finding of serum proteins in the edema fluid is not unique to these patients. It can be found, although in lesser amounts, in patients with pulmonary edema and secondary hypoxia from other causes, *i.e.*, congestive heart failure.

The finding of both a normal heart size on chest roentgenograms and normal to low central venous pressure measurements in the majority of patients with heroin and/or methadone induced pulmonary edema makes transient myocardial ischemia or failure a less likely explanation.[5]

Considering Etiologies 4 and 5 together, the variables involved here are innumerable. Not only does the strength of heroin vary from supplier to supplier, area to area, season to season, but also the contaminants involved in "cutting" the narcotic such as quinine, lactose, talc, etc., vary in a similar manner. This contaminant variation is supported by the observation of a "quinine-induced myocarditis" noted in autopsies only in certain geographic areas of the country. Also, individual addict response may change quite markedly depending, for example, on whether he has recently abstained. This would lessen his tolerance for the drug on being re-challenged. Host response can also differ from day to day, reflecting the drug addict's tenuous nutritional and emotional status. The potential combinations of these factors create an enormous (if not impossible) task if one was to attempt to isolate the possible hypersensitive inducing agent.

Halpern and Rho,[9] in a large autopsy series performed on narcotic addicts, reported severe congestion in all organs except the CNS. Gardner[7] reported similar autopsy findings in the lungs in acute deaths secondary to heroin and/or methadone overdoses. The similarity between these findings and those in patients whose demise was secondary to an idiosyncratic reaction to a non-narcotic drug, *i.e.*, penicillin, suggests a common etiology. On the other hand, the autopsy findings in victims of drug overdose are not significantly different from those found on autopsies of patients dying of any acute death.[10] It is also generally believed that the pulmonary congestion and edema may actually become more intense as the time interval from death to autopsy is increased. The occurrence of pulmonary edema in methadone overdoses, which is synthesized in a pure form and therefore contaminant free, combined with the lack of the usual clinical findings of a hypersensitive reaction in these patients, *i.e.*, laryngeal edema, wheezing, etc., militate against both the contaminant and allergic etiologies.

However, hypoxia is a common denominator in all the proposed etiologies and can by itself produce all the clinical and pathologic findings as described in the acute drug overdose deaths. The pulmonary edema experienced by persons exposed to

high altitudes is a combination of hypoxia, exercise and the individual's vascular response, but the main factor is probably hypoxia.[19–21] Severe hypoxia can also cause increased capillary permeability and myocardial stress and/or transient failure. In regard to this, Frand *et al.*[5] noted that the extent of hypoxemia in patients with heroin-induced pulmonary edema was greater than in patients with pulmonary edema due to ischemic heart disease. They felt that this degree of hypoxemia may reflect the extent of pulmonary edema, the heroin-induced hypoventilation, or both. How does one explain both the increased hypoxia and increased incidence of pulmonary edema? One factor may be the rapidity with which the hypoxia occurs, for example, via the intravenous administration used by the heroin addict. This may in essence "shock" the lung and CNS and not allow for a compensatory mechanism to operate effectively and maximally. Pulmonary edema rarely occurs in the barbiturate overdose patient who may hypoventilate to the same degree as the patient with narcotic overdose. The organs involved in the barbiturate overdoses have a relatively longer period of time to adjust to the initial but gradual insult due to the oral route of administration. If pulmonary edema does occur in this latter group, it is usually due to an overzealous attempt to diurese these patients and a resultant volume overload.

Another mechanism that may be evoked to explain the occurrence of the more severe hypoxemia and the increased incidence of pulmonary edema in the narcotic overdose patient is an effect by the drug at the alveolar-capillary level that hinders oxygen transport. We are presently performing experiments in our laboratory using intravenous methadone in dogs to further evaluate this transport system.

To date, we can only speculate concerning these various etiologies for the pulmonary edema associated with drug overdose. No one (including ourselves) has been able to reproduce these findings in the experimental animals, even under conditions of severe hypoxia.

Besides pulmonary edema and aspiration pneumonia, the chest findings may also reveal other roentgenographic features to which these patients are susceptible: (1) bacterial pneumonia unrelated to coma or aspiration; (2) pulmonary septic emboli either secondary to a right-sided endocarditis or an infected injection site, both of which are due to the unsterile conditions used (Fig. 2, *A* and *B*); (3) pulmonary

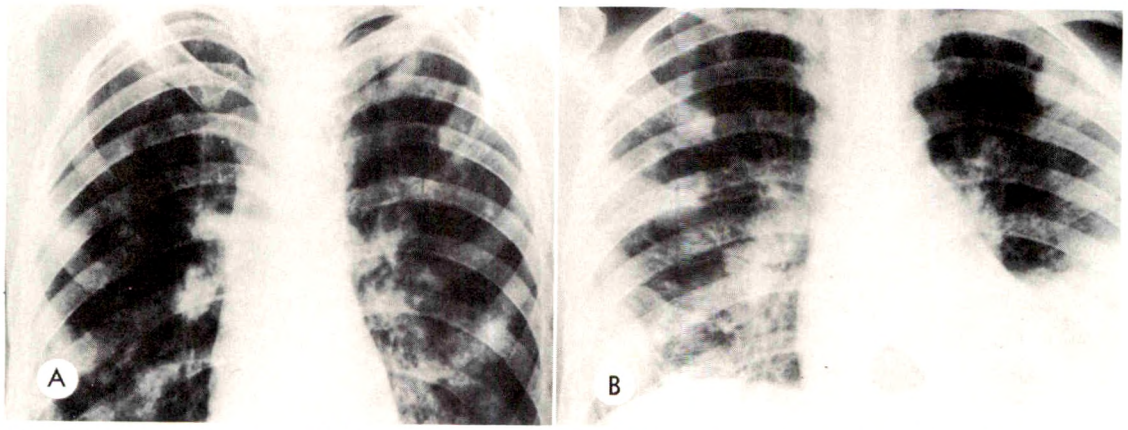


FIG. 2. Septic pulmonary emboli in a drug addict with bacterial tricuspid endocarditis. (*A*) Initial chest roentgenogram shows multiple, small scattered infiltrates, some with early cavitation. (*B*) Chest roentgenogram 5 days later showing a new shower of septic pulmonary emboli and the development of an empyema.

fibrosis, particularly in the long-term addict; and (4) pulmonary talc granulomatosis secondary to the use of oral tablets injected intravenously, *i.e.*, Ritalin (methylphenidate hydrochloride—USP) and "blue velvet."*

The combination of edema fluid as a culture medium, the inability of these patients to normally expand their lungs, their propensity for aspiration and their generally poor nutritional status can account for the frequent appearance of a superimposed bacterial pneumonia.[8] The occurrence of pulmonary fibrosis and talc granulomatosis can be explained by the type of contaminants used prior to injection in "cutting" the drug, *i.e.*, quinine, talc, lactose etc., or in the filtering of it, *i.e.*, cotton fibers, both of which may subsequently gain access to the blood stream. Talc and starch are also used as fillers in medications manufactured for oral use and are, therefore, injected intravenously as in "blue velvet" addiction. Although gross evidence of these sequelae are infrequently detected roentgenographically, they are very common findings histologically, especially in the chronic addict (Fig. 3).[2,3,11,18,22] Clinical studies by Soin *et al.*[16] have shown that asymptomatic narcotic addicts have a high incidence of abnormal lung scans, mainly consisting of diffuse, non-segmental defects. Studies by Karliner and co-workers[14] on a similar group of asymptomatic long-term addicts have shown a significant reduction in diffusing capacity, which suggests that these patients may suffer from functionally detectable but clinically asymptomatic chronic lung disease.

The findings in our study are similar to those previously reported, except for the lower incidence of pulmonary edema occurring in our group. This probably reflects the more stringent criteria used in selecting our patients. Also, one must realize that the incidence of pulmonary edema is probably even lower since the majority of drug abuse patients are never admitted to the hospital. Most studies essentially bias their findings in favor of pulmonary edema by studying this select group, *i.e.*, admission to intensive care unit. Nevertheless, there is little doubt concerning the increased incidence of these complications in both the acute overdose patient and the chronic narcotic addict.

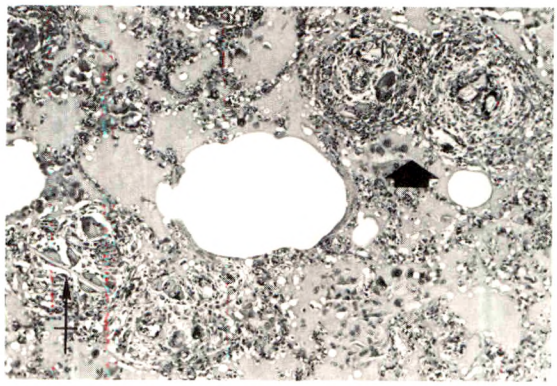


Fig. 3. Lung in a chronic drug addict. Arrow shows chronic granuloma in and surrounding small pulmonary arteries. Talc particles were demonstrated by polarized light. Cross-hatched arrow shows fiber (probably cotton) in another pulmonary artery. Note the surrounding pulmonary edema. (Autopsy specimen, Hematoxylin and Eosin ×130.)

Therefore, one can, with some confidence, establish general guidelines concerning the interpretation of these patients' chest roentgenograms both in the acute and chronic situation:

1. If the initial chest roentgenogram suggests pulmonary edema, one should very strongly suspect a heroin, methadone or Darvon overdose, either alone or in combination. Pulmonary edema rarely, if ever, is present on the admission chest roentgenogram of a non-narcotic overdose.
2. If the initial chest roentgenogram in a known narcotic overdose is clear or has an infiltrate, the radiologist should suggest a subsequent study to rule out the delayed occurrence of pulmonary edema. Heroin-induced pulmonary edema has been reported to occur 6–10 hours after admission.[13,17]
3. If a roentgenogram obtained 24–48

* "Blue velvet" is an expression used by the narcotic addict for tripelennamine (Pyribenzamine) with or without narcotics.

hours after admission shows an atypical pulmonary edema pattern, an aspiration or superimposed bacterial pneumonia should be suspected. Since narcotic-induced pulmonary edema has no distinctive roentgenographic pattern and usually clears within 48 hours, other complications become more probable if the chest roentgenogram has not cleared within this time period.

4. With a clinical history of spiking or high temperatures and a changing pulmonary pattern roentgenographically, septic emboli must be suggested.
5. If the initial chest roentgenogram suggests pulmonary hypertension in a young individual, a clinical history involving the intravenous use of oral drugs should be sought and the possibility of pulmonary talc granulomatosis then considered.

In conclusion, the radiologist can make a significant contribution to the proper medical management of the drug abuse patient if he is aware of the various complications that can occur and the general time sequence that these complications may be manifest roentgenographically.

SUMMARY

The admissions for drug overdoses to the medical intensive care unit of our institution for the past 3 years were reviewed. Forty-five patients were included in this study and separated according to the type of drug overdosage, clinical presentation and the presence of pulmonary edema.

Only those patients that had taken an overdose of heroin and/or methadone had pulmonary edema either clinically or by chest roentgenography. Of the 21 patients who were treated for overdosage of one or combinations of these 2 drugs, 8 had pulmonary edema on their chest roentgenogram. However, only 5 out of the 8 patients had physical findings of pulmonary edema. All patients with pulmonary edema had clearing on the chest roentgenograms within 24–48 hours.

Radiologists can make a significant contribution in the diagnosis of the narcotic abuse patients, particularly in the acute overdose situation, by being aware of the temporal sequence of the roentgenographic manifestations.

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REFERENCES

1. Borgstrom, K. D., Ising, U., Linde, E., and Lunderquist, A. Experimental pulmonary edema. *Acta radiol.*, 1960, *54*, 97–117.
2. Citron, B. P., Halpern, M., McCarron, M., Lundberg, G. D., McCormick, R., Pincus, L. J., Tatter, D., and Haverback, B. J. Necrotizing angitis associated with drug abuse. *New England J. Med.*, 1970, *283*, 1003–1010.
3. Douglas, F. G., Kafilmont, K. J., and Patt, N. L. Foreign particle embolism in drug addicts: respiratory pathophysiology. *Ann. Int. Med.*, 1971, *75*, 865–872.
4. Drinker, C. K. Pulmonary Edema and Inflammation. Harvard University Press, Cambridge, 1950, p. 106.
5. Fraud, U. P., Shim, C. S., and Williams, M. H. Heroin-induced pulmonary edema: sequential studies of pulmonary function. *Ann. Int. Med.*, 1972, *77*, 29–35.
6. Fraud, U. P., Shim, C. S., and Williams, M. H. Methadone-induced pulmonary edema. *Ann. Int. Med.*, 1972, *76*, 975–979.
7. Gardner, R. Methadone misuse and death by overdosage. *Brit. J. Addict.*, 1970, *65*, 113–118.
8. Gelfand, M. L., Hammer, H., and Hevizy, T. Asymptomatic pulmonary atelectasis in drug addicts. *Dis. Chest*, 1967, *52*, 782–787.
9. Halpern, M., and Rho, Y. M. Deaths from narcotism in New York City: incidence, cir-

cumstances and post mortem findings. *New York J. Med.*, 1966, *66*, 2391–2408.

10. Henry, L. D., and DiMaio, V. J. Pulmonary edema and propoxyphene. Letter to Editor. *J.A.M.A.*, 1971, *215*, 2115.
11. Hopkins, G. B., and Taylor, D. G. Pulmonary talc granulomatosis: complication of drug abuse. *Am. Rev. Resp. Dis.*, 1970, *101*, 101–104.
12. Hultgren, H. N., Lopez, C. E., Lundberg, E., and Miller, H. Physiologic studies of pulmonary edema at high altitude. *Circulation*, 1964, *29*, 393–408.
13. Johnson, B. A., James, A. E., Levey, M. S., and Campbell, J. H. Pulmonary edema caused by methadone. To be published.
14. Karliner, J. S., Steinberg, A. D., and Williams, M. H., Jr. Lung function after pulmonary edema associated with heroin overdose. *A.M.A. Arch. Int. Med.*, 1969, *124*, 350–353.
15. Katz, S., Stein, I. M., and Rulop, M. Heroin-induced pulmonary edema. *Am. Rev. Resp. Dis.*, 1972, *106*, 472–474.
16. Soin, J. S., McKusick, K. A., and Wagner, H. N., Jr. Regional lung function abnormalities in narcotic addicts. *J.A.M.A.*, 1973, *224*, 1717–1720.
17. Steinberg, A. D., and Karliner, J. S. Clinical spectrum of heroin pulmonary edema. *A.M.A. Arch. Int. Med.*, 1968, *122*, 122–127.
18. Viswanathan, R., Jain, S. K., and Subramanian, S. Pulmonary edema of high altitude. II. Clinical, aerohemodynamic and biochemical studies in group with history of pulmonary edema of high altitude. *Am. Rev. Resp. Dis.*, 1969, *100*, 334–341.
19. Viswanathan, R., Jain, S. K., and Subramanian, S. Pulmonary edema of high altitude. III. Pathogenesis. *Am. Rev. Resp. Dis.*, 1969, *100*, 342–349.
20. Viswanathan, R., Jain, S. K., and Subramanian, S. Pulmonary edema of high altitude. I. Production of pulmonary edema in animals under conditions of simulated high altitude. *Am. Rev. Resp. Dis.*, 1969, *100*, 327–333.
21. Wendi, V. I., Puro, H. E., Shapiro, J., Mathews, W., and Wolf, P. L. Angiothrombotic pulmonary hypertension in addicts: "blue velvet" addiction. *J.A.M.A.*, 1964, *188*, 755–757.

PNEUMOCONIOSIS: INTER-READER VARIABILITY IN THE CLASSIFICATION OF THE TYPE OF SMALL OPACITIES IN THE CHEST ROENTGENOGRAM*

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COAL workers' pneumoconiosis is recognized by certain fairly distinctive roentgenographic appearances and a history of an appropriate occupational exposure. It is divided into 2 types, simple and complicated. The former is recognized by the extent and profusion of small opacities present in the chest roentgenogram.[8] These are divided primarily by their shape into regular and irregular types, and secondarily by their size. Regular opacities are thus designated according to their dimensions as either p, q, or r and irregular opacities as s, t, or u. The p type of opacity measures up to 1.5 mm. in diameter, the q between 1.5 and 3 mm. and the r beween 3 and 10 mm. In regard to irregular opacities, no dimensions are given; however, the s type opacity is described as fine, t as medium, and u as coarse or blotchy. The extent of the inter-observer variation that occurs when films are being categorized for pneumoconiosis has been recognized for some time; nevertheless, most studies have restricted themselves to quantifying the variation that occurs in categorizing the profusion of small opacities rather than to quantifying that which occurs when differentiating the types into p, q, r and s, t, and u.[3–6]

This study is an attempt to determine the degree of observer variation reported, when chest roentgenograms with pneumoconiosis are being categorized according to the type of small opacity present.

MATERIAL AND METHOD

Over the past 5 years the Appalachian Laboratory for Occupational Respiratory Diseases has been conducting a large epidemiologic survey of the respiratory status of a sample of working coal miners. Between 1969 and 1971 over 9,000 miners had chest roentgenograms taken in the first round of examinations. The second round started in July, 1972 and is presently continuing. Many of the participants have had films taken in both rounds. Four physicians have been involved in the interpretations of the films. Each used the UICC Classification; however, all 4 have not read the whole set of films taken in both rounds.

From the first round of examinations 8,523 chest roentgenograms were selected while a further 929 were selected from the second round. Films read as complicated pneumoconiosis were excluded. In the first round comparative readings were available from only 2 readers, C and D, but in the second round the sample was interpreted by C as well as by 2 other readers, A and B.

Inter-reader variability was quantitated by computing the percentage of disagreements among readers according to the type of opacity recorded. Readers A, B, C, and D were compared 2 at a time by cross-classifying their readings. For the reasons stated above, A and B could not be compared with D. An example of such a cross-classification is illustrated in Table 1 for readers A and C. Those films in which which there were disagreements as to type are counted in the off diagonal cells. Films were designated as "negative," if small opacities were not found. This should not be taken to imply that all roentgenograms with small opacities were read as simple pneumoconiosis, since a small number of

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TABLE I

CROSS-CLASSIFICATION OF READER A VERSUS READER C ACCORDING TO TYPE OF OPACITY

Reader A

		Negative*	Irregular			Regular			Irregular and Regular								
			s	t	u	p	q	r	ps	pt	pu	qs	qt	qu	rs	rt	ru
Negative*		**548**		33	4	4	148		2	1			10	1			
Irregular	s	2	**1**	1		2	3			1			2				
	t	7		**5**	1	1	18						2	1			
	u																
Regular	p	5		2		**2**	13						1				
	q	19		6			**44**	6					5	2			1
	r	1		1		1	2	**3**									
Irregular and Regular	ps	1				1											
	pt						2										
	pu																
	qs						2										
	qt	1					2	2								1	3
	qu																
	rs																
	rt																
	ru																

Of the 929 films read, 35 per cent lie in the off-diagonal cells, *i.e.*, the bold faced numbers.
* Roentgenograms in which small opacities were not found.

the total sample was read as subcategory 0/1.

RESULTS

The percentage of disagreements among readers in the classification of type is illustrated in Table II. It is apparent that the number of disagreements between C and D is enormous (86 per cent), between A and B moderate (38 per cent), between A and C moderate (35 per cent), and between B and C slight (19 per cent).

Table III shows the percentage of films which were read as negative and positive; *i.e.* with and without small opacities. The positives are broken down into regular, irregular, and both regular and irregular small opacities for each reader. Readers A, B, and C classified the majority of roentgenograms as negative; in contrast, D read the majority as positive. Of the positive films, regular nodules were most frequently recorded by A, B, and C, while irregular opacities were most frequently recorded by D.

Table IV contains the percentage of regular opacities classified as p, q, or r. Readers A, B, and C assign type q more frequently than p or r. A reads it almost exclusively (91 per cent), and B and C assign it 73 and 53 per cent of the time, respectively.

TABLE II

PERCENTAGE OF DISAGREEMENTS BETWEEN READERS IN THE CLASSIFICATION OF THE TYPE OF OPACITIES

Readers	Per Cent
2nd Round Sample	
A *vs.* B	38
A *vs.* C	35
B *vs.* C	19
1st Round Sample	
C *vs.* D	86

TABLE III

PERCENTAGE OF FILMS CLASSIFIED AS NEGATIVE* OR AS SHOWING THE PRESENCE OF REGULAR, IRREGULAR OR BOTH REGULAR AND IRREGULAR SMALL OPACITIES

Reader	Negative*	Regular	Irregular	Both
2nd Round Sample				
A	63	28	6	3
B	85	5	5	5
C	81	12	5	2
1st Round Sample				
C	82	10	6	2
D	13	0.5	47	39.5

* Roentgenograms in which small opacities were not found.

DISCUSSION

The results of the study indicate that inter-reader variation in the classification of the type of small opacity present in the films showing pneumoconiosis is appreciable. There is disagreement in a minimum of 1 out of 5 classifications and in a maximum of 1 out of 1.16. Although D almost never agrees with C (14 per cent), A, B, and C are not too dissimilar. The fact that D reads so many films as positive partly explains the inconsistencies between him and the other readers, but he also reports irregular opacities much more frequently. The average percentage of disagreements among A, B, and C is approximately 31 per cent, or 1 out of 3. Although A, B, and C all read predominantly regular opacities, A differs from the others in the more frequent assignment of type q.

Since the UICC Classification is used extensively to categorize chest films for coal workers' pneumoconiosis, the extent of the inter-observer variability that occurs with its use should be clearly appreciated. From previous studies, disagreements among readers as to the 4 major categories of profusion have been recorded in approximately 40 to 50 per cent of the roentgenograms.[5,6] When the elaborated 12 point scale is used, disagreement is even more frequent. Since there is a 40 per cent or more disagreement according to major category and a further 30 per cent according to the type of opacity, the use of a scale which combines profusion and type should lead to marked reader to reader variation. Thus there are 3 regular and 3 irregular type opacities which can be assigned individually or in combination, with the result that there are 63 possible combinations in which small opacities may be classified. The 15 categories employed in Table 1 should be considered the most frequent and could be combined with the UICC 12 point scale of profusion. With the exception of subcategories 0/- and 0/0 the scale would consist of 15 type classifications with each of the remaining ten profusion subdivisions, and this serves to emphasize the possible inter-reader variation and does much to explain the lack of consistency found in this study.

TABLE IV

THE FREQUENCY OF VARIOUS TYPES OF SMALL ROUNDED OPACITIES ACCORDING TO READER

Reader	*p* (per cent)	*q* (per cent)	*r* (per cent)
A	4	91	5
B	27	53	20
C	20	73	7

It has been suggested that progression of pneumoconiosis should be measured by trying to assess separately the increases in small regular and small irregular opacities. Over-all progression would be the sum of the individual progression of the regular and irregular opacities considered separately. If such a system is to be used, the plethora of possible combinations is bound to lead to undue inter-reader variability. On the other hand, if progression is re-

corded by estimating the combined increase of both opacities together, without trying to separate them, less variation should result.

Whether it is important in coal workers' pneumoconiosis to differentiate regular from irregular small opacities and to further subdivide them according to the size, is at present unknown. There is some evidence that the r type of regular opacity has a different pathology from the other regular opacities (p and q). Its etiology may be related to the presence of the rheumatoid diathesis.[1] In addition, miners with the p type of opacity have been shown to have a marginally lower diffusing capacity than do those with type q and r.[7] Nonetheless, it must be remembered that so far it has not been possible to show a pathologic distinction between the p and q type of radiographic opacity. Further studies are needed before it can be claimed that there is any useful purpose served by attempting to differentiate between the various types of small regular opacities.

Irregular opacities are observed much less frequently in coal workers' pneumoconiosis than they are in certain other pneumoconioses, *viz.* asbestosis. Moreover, while there is no doubt that they may be seen in coal workers' pneumoconiosis, the assumption that their presence is a consequence of the inhalation of coal dust is much more debatable. Again, as yet there is no pathologic explanation for the presence of irregular opacities in CWP; however, there is some evidence to support that cigarette smoking rather than retention of coal dust is responsible.[2]

CONCLUSION

The inter-observer variation that occurs in the differentiation of the various types of small opacities present in the chest roentgenograms of working miners was quantified. In approximately 2 out of 3 films there was disagreement as to the type of small opacities present. Since this variation is appreciable, epidemiologic studies that depend on the differentiation of these types of opacity should involve a majority opinion from several interpreters rather than relying on 1 reader only. In addition, the rigid training of readers periodically by the same standards should help to keep this variation within acceptable limits.

Finally, the progression of coal workers' pneumoconiosis should be quantified by assessing simultaneously the combined increase of both regular and irregular opacities. If progression is to be independently determined for each type of small opacity and then summed to obtain the over-all progression, the procedure will almost certainly lead to an unacceptable amount of inter-observer variation.

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REFERENCES

1. Caplan, A., Payne, R. B., and Withey, J. L. Broader concept of Caplan's syndrome related to rheumatoid factors. *Thorax*, 1962, *17*, 205–213.
2. Carilli, A. D., Kotzen, L. M., and Fischer, M. J. Chest roentgenogram in smoking females. *Am. Rev. Resp. Dis.*, 1973, *107*, 133–136.
3. Fletcher, C. M., and Oldham, P. D. Problem of consistent radiological diagnoses in coalminers' pneumoconiosis. *Brit. J. Indust. Med.*, 1949, *6*, 168–183.
4. Liddell, F. D. K. Elaboration of ILO classification of simple pneumoconiosis. *Brit. J. Indust. Med.*, 1969, *26*, 89–100.
5. Peters, W. L., Reger, R. B., and Morgan, W. K. C. Radiographic categorization of coal workers' pneumoconiosis by lay readers. *Environ. Res.*, 1973, *6*, 60–67.
6. Reger, R. B., Amandus, H. E., and Morgan, W. K. C. On diagnosis of coal workers' pneumoconiosis Anglo-American disharmony. *Am. Rev. Resp. Dis.*, 1973, *108*, 1186–1191.
7. Seaton, A., Lapp, N. L., and Morgan, W. K. C. Relationship of pulmonary impairment in simple coal workers' pneumoconiosis to type of radiographic opacity. *Brit. J. Indust. Med.*, 1972, *29*, 50–55.
8. UICC Committee. ILO U/C 1971 international classification of radiographs of pneumoconioses. *Med. Radiog. & Photog.*, 1972, *48*, 65–110.

GALLIUM 67 CITRATE SCANNING IN PATIENTS WITH PULMONARY SARCOIDOSIS*

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SINCE the initial observation in 1969 by Edwards and Hayes[1] that gallium 67 citrate accumulated in malignant neoplasms there have been reports of its accumulation in nonmalignant pulmonary lesions.[2–4,10–12] It has been suggested on the basis of a small series that there is a relationship between the activity of pulmonary sarcoidosis and the uptake of gallium 67 citrate.[3] A decrease in lung uptake of gallium 67 citrate following steroid therapy in sarcoidosis and other nonmalignant disease has been reported.[3–6]

The present study consists of 29 patients with biopsy proven sarcoidosis.

MATERIAL AND METHOD

From September 1972 to March 1973, 29 patients referred from the Sarcoidosis Clinic of Johns Hopkins Hospital had gallium 67 citrate scans. They were chosen on the basis of the following criteria: (1) the clinical course compatible with the diagnosis of sarcoidosis; (2) histopathologic demonstration of noncaseating granulomata by tissue biopsy and/or biopsy of a Kveim reaction.[7] All patients were Black. There were 20 females and 9 males, ranging in age from 20 years to 57 years, with a mean of 27.5 years. The patients received 3.0 millicuries of gallium 67 citrate intravenously and scans of an area extending from the nasion to the symphysis pubis were obtained 48 to 72 hours later. The scannings were performed with a rectilinear scanner having a 5 inch diameter sodium iodide crystal. Counts from 2 pulse height analyzers (PHA) were summed. The photo peaks were centered at 90 kev. and 242 kev. with window of 20 kev. and 175 kev., respectively. The patients were scanned supine and counts optimized at 200 counts per square centimeter using a medium energy (370 kev.) collimator.

Following the evaluation of the rectilinear scan, selected areas of interest were imaged with an Anger-type scintillation camera (Nuclear Chicago HP) equipped with 2 pulse height analyzers. One PHA was centered at the 184 kev. gallium 67 peak and the other at the 296 kev. gallium 67 peak. A 15 per cent window was used. The output from both PHAs was displayed on a cathode ray oscilloscope. Images were recorded on high contrast 35 mm. copy film (Eastman Kodak).

Accumulation of gallium 67 citrate in the pulmonary parenchyma and hilum was compared to the accumulation of gallium 67 in the liver and the soft tissue of the neck and shoulders. From our experience, the gallium 67 activity in the lungs is usually the same as that in the soft tissues of the neck and the shoulders in a normal person. In all patients, who were studied by whole body scans, there was uptake noted in the regions of the eyes, nose and submandibular glands. Breast uptake was noted in 5 nonlactating women. Liver uptake was noted in all patients who were examined by whole body scans. Spleen uptake was noted in 19 patients. Quantitative analysis of liver and spleen uptake was not made in this study. Clinical activity was classified as stable, progressive or undetermined. Symptoms were the primary determinant used for clinical activity. The disease was classified as progressive if the symptoms had developed or had become worse within 60 days of the deter-

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mining visit. The following symptoms were evaluated: shortness of breath; cough; chest pain; neuromuscular complaints; skin disease; and arthralgia or fever. Vital capacity, 1 second forced vital capacity, and steady state CO_2 diffusion capacity were measured in all patients. In 11 patients, the disease was stable, in 13 progressive and in 5 undetermined. Upright, postero-anterior and lateral chest roentgenograms were obtained at the time of the scan. The progression showing the chest roentgenograms resulted in 3 patients being changed from stable to the progressive category. The scan findings were evaluated independently of the clinical data and roentgenograms by 2 observers.

RESULTS

I. Accumulation of Gallium 67 Citrate in Lung Parenchyma

In our population of 29 patients, 23 had mild to severe parenchymal infiltrates in the chest roentgenograms (Table 1). Of these, 61 per cent (14/23) demonstrated parenchymal gallium 67 uptake in the area corresponding to the infiltrates demonstrated on the chest roentgenograms (Fig. 1, *A* and *B*). In no case did gallium 67 accumulate in an area of the lung shown to be normal on the chest roentgenograms. In 1 patient with bronchiectasis and pulmonary sarcoidosis, bronchiectasis may have been partially responsible for gallium 67 accumulation. Gallium 67 accumulation did not correlate with the roentgenographic pattern of infiltrates; *i.e.*, nodular *versus* linear. The specific lobe in which the infiltrate was located seemed also to have no bearing on the degree of gallium 67 uptake.

Fourteen of 29 patients in our series had abnormal pulmonary parenchymal uptake of gallium 67 (Table 1), and of this number, 57 per cent (18/14) were classified as progressive; *i.e.*, the symptoms were becoming worse. Thirty-six per cent (5/14) were clinically stable; we could not determine the status in the remaining patient.

Fifteen of our 29 patients (52 per cent) had no abnormal uptake of gallium 67 in the pulmonary parenchyma, and of these patients, 4/15 (27 per cent) were clinically progressive. Therefore, the uptake in the pulmonary parenchyma did not seen to correlate with progressively increasing symptoms of sarcoidosis.

II. Hilar Accumulation of Gallium 67

In our series of 29 patients, 16 (55 per cent) had hilar lymphadenopathy on the chest roentgenogram (Table 11). Of these, 94 per cent (15/16) had positive gallium 67 accumulation in the hilar area. However, 8 patients in our series had no hilar lymphadenopathy in the chest roentgenogram, but had abnormal gallium 67 uptake in the region of the hilar lymph nodes.

There were 23/29 patients (79 per cent) who had gallium 67 uptake in the pulmonary hilum (Table 11). Nine of the 23 patients

Table I

Gallium 67 Citrate Scan, Clinical Activity and Chest Roentgenogram

Gallium 67 Citrate Scan	Clinical Activity			Chest Roentgenogram	
	Progressive	Stable	Activity Undetermined	Parenchymal Change Present	Parenchymal Change Not Present
Abnormal uptake in pulmonary parenchyma (14)	8	5	1	14	0
No uptake in pulmonary parenchyma (15)	4	7	4	9	6

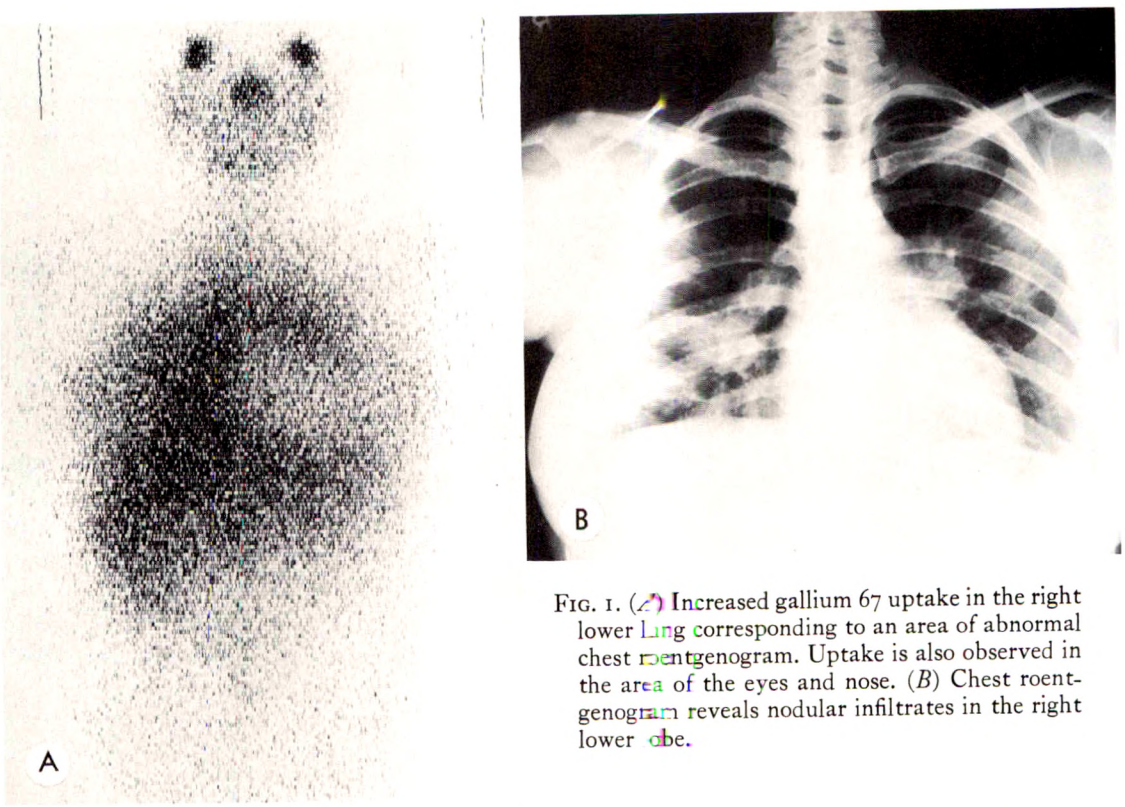


Fig. 1. (*A*) Increased gallium 67 uptake in the right lower lung corresponding to an area of abnormal chest roentgenogram. Uptake is also observed in the area of the eyes and nose. (*B*) Chest roentgenogram reveals nodular infiltrates in the right lower lobe.

(39 per cent) were classified as stable. Nine of the 23 patients (39 per cent) had progressive symptoms, and the remainder were not classified. Among the 29 patients in our study, 6 (20 per cent) did not have gallium 67 uptake in the hilar area. Of these 6, 3 were classified as progressive.

III. Gallium 67 Accumulation and Steroid Therapy

Corticosteroids often improve pulmonary function in sarcoidosis, at least during the period of administration.[8,9] Therefore, it seemed reasonable to ask whether monitoring of gallium 67 accumulation might be helpful in evaluating the response to steroid therapy. Three patients (C.M., L.M. and B.M.) had clinically progressive disease, positive chest roentgenograms, and abnormal accumulation of gallium 67 in the pulmonary parenchyma and hilar lymph nodes when studied prior to steroid therapy (Table III). After 5 to 7 months of steroid therapy, B.M.'s and C.M.'s chest

Table II

Gallium 67 Citrate Scan, Clinical Activity and Chest Roentgenogram

Gallium 67 Citrate	Clinical Activity			Chest Roentgenogram	
	Progressive	Stable	Activity	Lymph-adenopathy	Hilar Lymph-adenopathy Not Present
Abnormal uptake in pulmonary hilum (23)	9	9	5	15	8
No uptake in pulmonary hilum (6)	3	3	0	1	5

TABLE III

RESULT OF REPEAT GALLIUM 67 CITRATE SCAN

Name and Change in Clinical Status at Second Study	Steroid Therapy	Initial Study		Repeat Study	
		Scan	Chest Roentgenogram	Scan	Chest Roentgenogram
C.M. Clinically improved	15 mg./da.	2/14/73 Uptake in pulmonary hilum and parenchyma	2/14/73 Hilar lymphadenopathy: nodular parenchymal density	8/8/73 No uptake in the lung	8/8/73 Unchanged from 2/14/73
L.M. Clinically improved	20 mg./da.	3/14/73 Uptake in pulmonary hilum and parenchyma; liver uptake	3/14/73 Hilar lymphadenopathy; parenchymal	8/8/73 No uptake in the lung; liver uptake	8/8/73 Clear chest roentgenogram
B.M. Clinically improved	30 mg./da.	Hilar uptake positive	Hilar lymphadenopathy	No uptake	Hilar lymphadenopathy

roentgenograms remained unchanged, but there was no longer gallium accumulation in the pulmonary parenchyma or hilar lymph nodes in the repeat study. In patient L.M., not only did the gallium or accumulation in the lung disappear, but the chest roentgenogram returned to normal (Fig. 2, *A* and *B*).

Four out of 5 other patients who were on Prednisone therapy at the time of the scan had significant accumulation of gallium 67 in hilar lymph nodes and/or pulmonary parenchyma (Table IV). The 1 patient without gallium 67 accumulation in the lungs was on the largest dose of steroid therapy. The remaining 4 were on less than 15 mg. per day of Prednisone.

DISCUSSION

Prior to the present study, it was known that gallium 67 accumulated in the pulmonary parenchyma and the hilar lymph nodes of some patients with sarcoidosis. Evaluation of the degree of pulmonary uptake of gallium 67 was complicated by occasional abnormal uptake in the breast, and by accumulation of the tracer in the hilum which was difficult to differentiate from parenchymal uptake. Nevertheless, it seemed clear that gallium 67 uptake in the lung was not correlated with the location of the infiltrates or the pattern of infiltrates (nodular *versus* linear), although if the chest roentgenogram did not reveal parenchymal infiltrates, the gallium 67 scan of the lung parenchyma was always normal. The gallium 67 uptake in the lung parenchyma or hilar lymph nodes correlated poorly with the evidence of a clinical activity. However, roentgenographically visible hilar lymphadenopathy always accumulated gallium 67. In 8 patients, gallium 67 accumulated in the hilar lymph nodes despite a normal chest roentgenogram.

Conversion of the gallium 67 lung scan from positive to negative during Prednisone therapy has been reported previously in 1 patient with pulmonary sarcoidosis. In the present study, in 3 patients gallium 67 accumulation disappeared when the patient was treated with high dose steroid therapy ranging from 15 mg. per day to 30 mg. per day for 5 to 7 months. Although the number of patients is small, our data suggest that monitoring gallium 67 accumulation in pulmonary parenchyma or hilar lymph nodes in patients with pulmonary sarcoidosis may be of value, particularly when the mechanism of the gallium 67

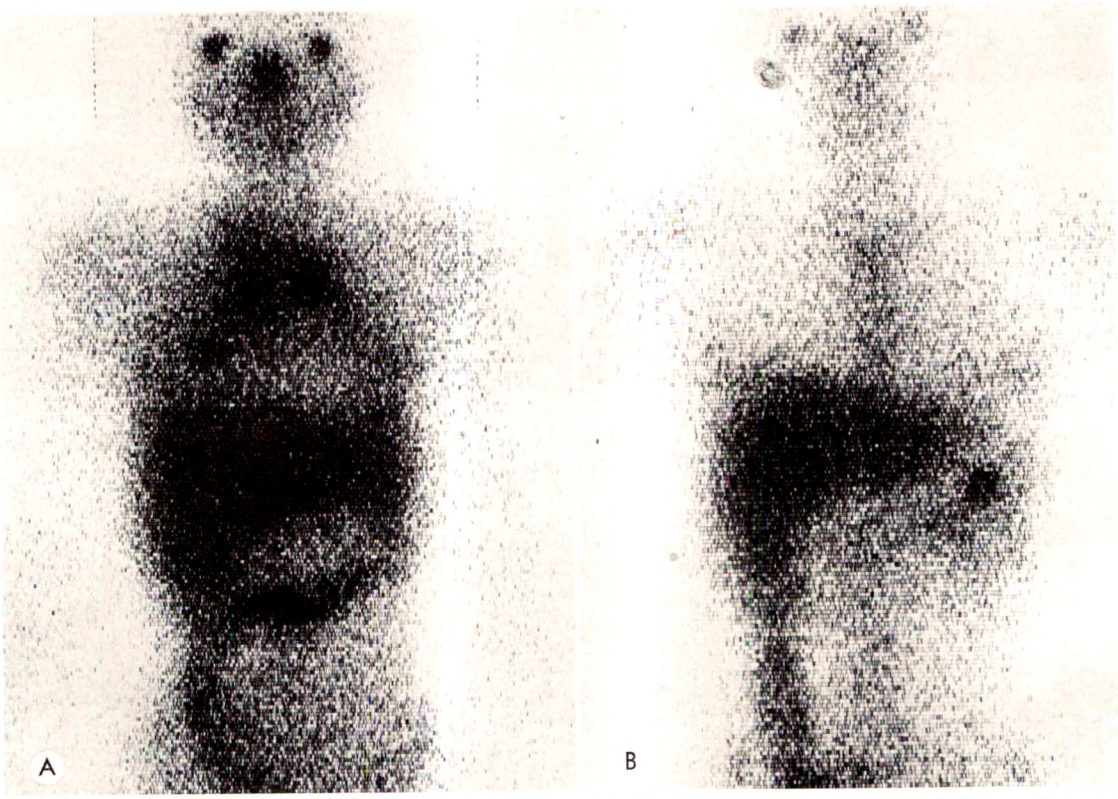


Fig. 2. L.M. (*A*) *March 14, 1973*. Gallium 67 scan reveals an uptake in the pulmonary hilum and parenchymal area. Liver uptake is also apparent. (*B*) *August 8, 1973*. Uptake of gallium 67 is no longer observed in the lung. Liver uptake is still present.

accumulation is better understood. Various hypotheses have been suggested. Gallium 67 may be incorporated into lysosomes of macrophages or it may be associated with the cell membrane of lymphocytes.[6] However, further study is needed.

Table IV

Result of Gallium 67 Citrate Lung Scan in Patient with Steroid Therapy

Patient	Steroid Therapy Dose	Gallium 67 Citrate Lung Scan	Clinical	Chest Roentgenograms
M.B.	20 mg./da.	No abnormal uptake	Stable	Parenchymal change present
Mu.B.	12.5 mg./da.	Abnormal hilar uptake	Stable	Parenchymal change present
B.A.	10 mg./da.	Abnormal hilar uptake	Stable	Normal
M.S.	5 mg./da.	Abnormal hilar and parenchymal uptake	Stable	Hilar lymphadenopathy present; parenchymal change present
H.M.	2.5 mg./da.	Abnormal hilar and parenchymal uptake	Stable	Hilar lymphadenopathy present; parenchymal change present

It seems possible that gallium 67 uptake may be the only sign of disease activity in some patients with pulmonary sarcoidosis.

SUMMARY

Twenty-nine patients with biopsy proven pulmonary sarcoidosis in various clinical stages of activity were studied with gallium 67 citrate scans.

If the chest roentgenogram was normal, we were not able to demonstrate any abnormal pulmonary accumulation of gallium 67, regardless of the patient's subjective complaints. Eight patients had abnormal gallium 67 accumulation in the hilar region with no roentgenographic evidence of hilar lymphadenopathy. Gallium 67 uptake in the pulmonary parenchyma or hilar regions correlated poorly with clinical activity.

Large doses of steroids decreased the uptake of gallium 67 in the pulmonary parenchyma and hilum in 3 patients. Although the mechanism of gallium 67 accumulation in pulmonary sarcoidosis remains to be determined, its measurement may prove to be a useful new index of disease activity before, during and after therapy.

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REFERENCES

1. Edwards, C. L., and Hayes, R. L. Tumor scanning with gallium-67 citrate scanning. *J. Nuclear Med.*, 1969, *10*, 103–105.
2. Higashi, T. Clinical evaluation of gallium-67 citrate scanning. *J. Nuclear Med.*, 1969, *10*, 103–105.
3. Langhammer, H., Glanbitt, G., Grebe, S. F., *et al.* Gallium-67 for tumor scanning. *J. Nuclear Med.*, 1972, *13*, 25–30.
4. Lavender, J. P., Lowe, J., Barker, J. R., *et al.* Gallium-67 citrate scanning in neoplastic and inflammatory lesions. *Brit. J. Radiol.*, 1971, *44*, 361–366.
5. McKusick, K. A. Gallium accumulation in pulmonary sarcoidosis. *J.A.M.A.*, 1973, *223*, 688.
6. Merz, T. Personal communication.
7. Proceedings of the International Congress on Sarcoidosis. *Am. Rev. Resp. Dis.*, 1961, *84*, 1–183.
8. Sharma, O. P., Cope, C., and Willings, M. H., Jr. Course of pulmonary sarcoidosis with or without corticosteroid therapy as determined by pulmonary function studies. *Am. J. Med.*, 1966, *41*, 541–551.
9. Stone, D. J., and Schwartz, A. Long term study of sarcoid and its modification by steroid therapy. *Am. J. Med.*, 1966, *41*, 528–540.
10. Van der Schoat, J. B., *et al.* Gallium-67 scintigraphy. Monte Carlo, October, 1972. ZAGA/SM: 164–219.
11. Van der Schoat, J. B., *et al.* Gallium-67 scintigraphy in lung diseases. *Thorax*, 1972, *27*, 543–546.
12. Winsor, D. W., Grebe, S. F., Sargent, N., *et al.* Pulmonary concentration of gallium-67 in pneumoconiosis. From abstract for scientific program.

PULMONARY MANIFESTATIONS IN WEGENER'S GRANULOMATOSIS*

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NECROTIZING lesions of small vessels characterize a complex group of diseases first described in 1866 by Küssmaul and Maier, under the name periarteritis nodosa.[50] From this group, Klinger and Wegener[48,49] isolated a clinical entity characterized by granulomatous involvement of the upper respiratory tract, the lungs and the kidneys,[25] which became known as "Wegener's granulomatosis" (W.G.). This disease, however, does not always present the classical triad described above; a large number of cases have been recognized in which the typical pathologic features are limited to the respiratory tract. If the findings are confined to the lung, the clinical course is more benign and there is a better response to therapy.[5,15] This led to the concept of "limited Wegener's granulomatosis,"[10,11,38] which can also be extended to cases with localized manifestations of this disease in which the lungs are uninvolved.

Two conditions are related to W.G.: "midline lethal granuloma" is a mutilating and ulcerating granulomatous disease limited to the nose and the facial tissues, with very poor prognosis. This disease is considered by some authors as a separate entity[43] whereas others consider it a localized manifestation of Wegener's granulomatosis.[4] Churg and Strauss described an allergic granulomatosis, closely related to W.G. in which an allergic etiology can be recognized, and the granulomas contain a large number of eosinophils.[10]

The purpose of this paper is to review briefly some of the recent developments in the understanding of necrotizing vasculitis of the Wegener type and to discuss the pulmonary roentgenographic findings, on the basis of which the radiologist may be able to contribute to an earlier diagnosis and an improved prognosis of these rare but fascinating disorders.

MATERIAL

The medical records of the University of Rochester Strong Memorial Hospital revealed 10 cases in which a diagnosis of Wegener's granulomatosis was made by the Pathology Department. There were 4 males and 6 females, with ages ranging from 26 to 70 years. Table 1 summarizes the clinical features and the sources of pathologic proof. Many organs showed clinical evidence of disease, but not all have been biopsied. Only 2 of the patients, Cases I and VI, have died, the others have been in remission for 4 months to 4 years.

REPORT OF CASES

CASE I.* A 70 year old woman was referred to the hospital because of a pneumonic infiltrate in the right middle lobe. She had had fever, weight loss and pleuritic pain for several weeks. Her past history revealed corneal ulcers 2 years earlier, progressive deafness and sanguinopurulent nasal discharge for 1 year and gradual nasal septum collapse over several months before admission. Sinus roentgenograms had shown clouding of the right maxillary antrum and a nasal biopsy was interpreted as compatible with lethal midline granuloma. She improved with steroid therapy; however, the right middle lobe (RML) became progressively more consolidated on roentgenography (Fig. 1, *A* and *B*) and a thoracotomy was performed. The resected RML showed diffuse vasculitis and granulomatous changes of the Wegener type. The patient recovered postoperatively, and was placed on cyclophosphamide. She later developed increasing renal problems, and died at home 2 years later. No autopsy was performed.

* This case was the subject of a previous report.[42]

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Table I

CLINICAL AND PATHOLOGIC DATA

Case	Sex	Age	Organs Clinically Affected									Sources of Pathologic Diagnosis
			Ears	Eyes	Nose	Sinuses	Mouth	Lung	Kidney	Skin	Joints	
I	F	70	X	X	X	X		X	X			Nose, Lung
II	M	63			X			X	X			Lung
III	F	28	X	X	X	X	X	X	X	X		Ear, Nose, Gingiva, Kidney
IV	F	52		X	X	X		X				Nose, Lung
V	M	61	X	X	X			X	X			Nose, Kidney
VI	M	46	X		X	X	X		X			Nose, Antrum, Palate, Kidney
VII	M	30		X	X					X	X	Orbit
VIII	F	52				X			X	X		Left calf
IX	F	70		X	X	X					X	Nasal septum
X	F	26	X						X	X	X	Kidney
Total= 10			5	6	8	6	2	5	7	4	3	

Case II. A 63 year old engineer was transferred from another hospital with a 6 week history of fever, chills, weight loss, cough and hemoptysis, which did not improve on antibiotic therapy. Chest roentgenograms had showed an infiltrate limited to the superior segment of the right lower lobe (RLL), which was unchanged on admission (Fig. 2, *A* and *B*). Fiberoptic bronchoscopy revealed a patent, but inflamed segmental bronchus and bronchial brushing was negative. Because of the suspicion of carcinoma, a thoracotomy was performed. The superior segment of the RLL was firm and consolidated and the right lower lobe was resected. The pathologic specimen showed a marked proliferative and vascular necrotizing process diagnostic of Wegener's granulomatosis. The patient is well 4 months later, except for chronic albuminuria.

Case III. A 28 year old woman first presented with nasal discharge, gingival bleeding, sinus

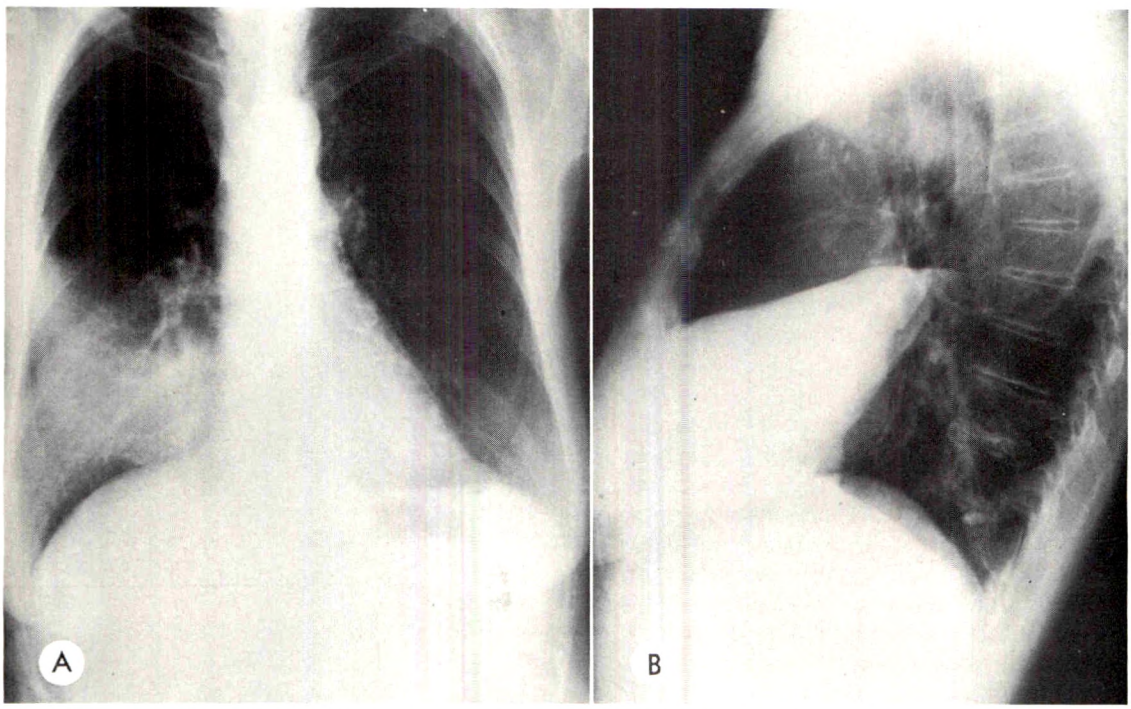


Fig. 1. Case 1. (*A*) On posteroanterior and (*B*) lateral chest roentgenograms, a complete consolidation of the right middle lobe (RML) can be seen.

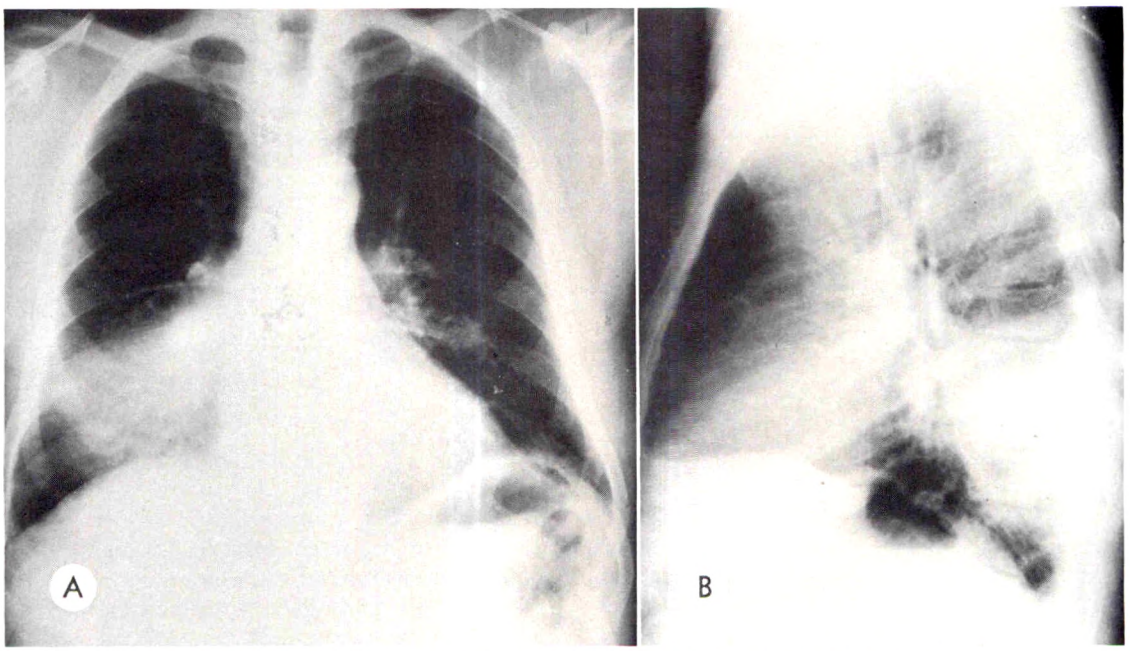


FIG. 2. Case II. (*A*) Posteroanterior and (*B*) lateral chest roentgenograms show a consolidation in the superior segment of the right lower lobe (RLL).

pain and rapidly progressive glomerulonephritis. Nasal, gingival and renal biopsies showed Wegener's granulomatosis. Over the following 6 months, the patient also developed bilateral otitis media, episcleritis, stiffness of the elbows, hands, knees and feet and a 3 cm. red area over the left heel. When admitted again because of fever and cough, a small nodule was seen in the left upper lobe, which subsequently cavitated (Fig. 3). Treatment with cytoxan was started and clinical improvement was accompanied by complete resolution of the pulmonary nodule. The chest roentgenogram has remained clear 1 year later, but the creatinine blood level shows intermittent elevation.

CASE IV. A 52 year old female was referred to the hospital for evaluation of diplopia, considered to be secondary to orbital involvement of Wegener's granulomatosis. This diagnosis had been made 2 years earlier, on biopsies of the nasal septum and on tissue from bronchoscopic material. A chest roentgenogram at the time (Fig. 4) showed 3 rather ill-defined nodules in the left lower lobe, one of which contained a small cavity. There was no evidence of kidney involvement. On therapy with high doses of steroids, the chest roentgenograms cleared and the patient improved. Diplopia also

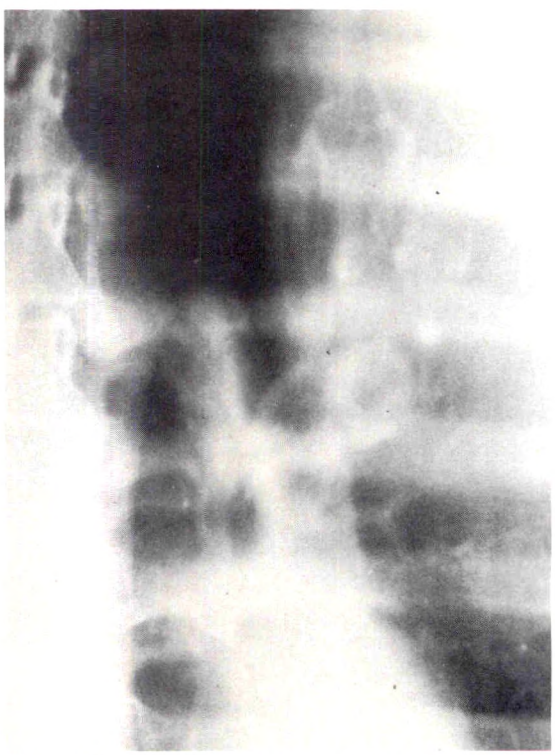


FIG. 3. Case III. An anteroposterior tomographic cut shows a solitary cavitating nodule in the left upper lobe (LUL).

yielded to steroids. The patient is doing well more than 3 years following her initial presentation and is on long-term cytoxan therapy.

Case v. A 61 year old mechanic was admitted to another hospital with a 2 month history of fatigue, nasal stuffiness, epistaxis, cough and hearing loss. The chest roentgenograms showed ill-defined bilateral apical infiltrates suggesting tuberculosis. There was proteinuria and hematuria and the blood urea nitrogen was 59 mg. per cent. The patient left the hospital against medical advice. His condition continued to deteriorate at home and he was readmitted 4 weeks later with progressive dyspnea and profound weakness. He was transferred to the University Hospital, where he arrived in severe respiratory and renal failure requiring tracheostomy and dialysis. The chest roentgenogram (Fig. 5*A*) revealed diffuse nodular infiltrates in both lungs. A biopsy of the left inferior turbinate and of the kidney showed necrotizing vasculitis with granuloma formation. Treatment with methylprednisolone and cytoxan brought about gradual improvement over a period of 4 weeks, during which his chest roentgenograms cleared (Fig. 5, *B–D*). Three months later he is doing well on cytostatic therapy.

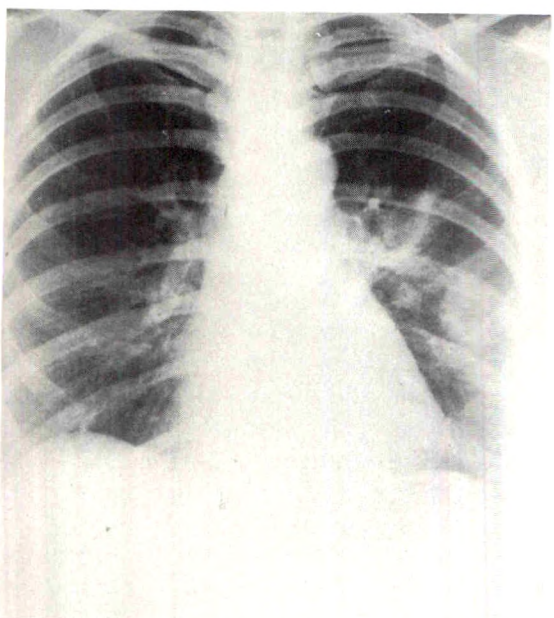


Fig. 4. Case iv. A posteroanterior chest roentgenogram shows 3 nodular lesions in the left lower lobe (LLL), 1 having a small central cavitation.

ROENTGEN FINDINGS

Two patients had a homogeneous consolidation of a lobe or a segment: the right middle lobe in Case i and the superior segment of the right lower lobe in Case ii. Both areas appeared slightly overexpanded on roentgenography. Following resection, there was no recurrence elsewhere in the chest.

Two patients showed a nodular pattern. Case iii had a well-defined solitary nodule, 1.5×2 cm. in size, in the left upper lobe, which, 2 weeks following its initial discovery, revealed a small cavity with a rather thick wall. Case iv presented multiple nodules in the left lower lobe, 2 of which were rather ill-defined and located close to the left hilum, while the largest measured 3.5×4 cm. and had a small cavity with thick walls surrounding it. Following steroid and cytostatic therapy, all these nodules disappeared slowly and roentgenograms obtained 6 months later showed normal lungs in both cases.

Case v had a widely disseminated reticulonodular, interstitial infiltrate which cleared gradually over a period of about 4 weeks (Fig. 5, *A–D*).

DISCUSSION

Wegener's disease has received increasing attention in the medical literature. It is part of a spectrum of diseases[1,4] characterized by necrotizing lesions of small blood vessels, which range from periarteritis nodosa and dermatomyositis, in which the angiitis element predominates through hypersensitivity angiitis, rheumatic arteritis and temporal arteritis, which have a mixture of angiitis and granulomatous elements to allergic angiitis and Wegener's granulomatosis, in which the element of granulomatosis predominates.[51]

W.G. appears to be more common than originally thought,[18] and may present in different clinical and pathologic forms. Carrington and Liebow[10] first described cases with involvement limited to the lungs. Cassan and associates[12] reported the

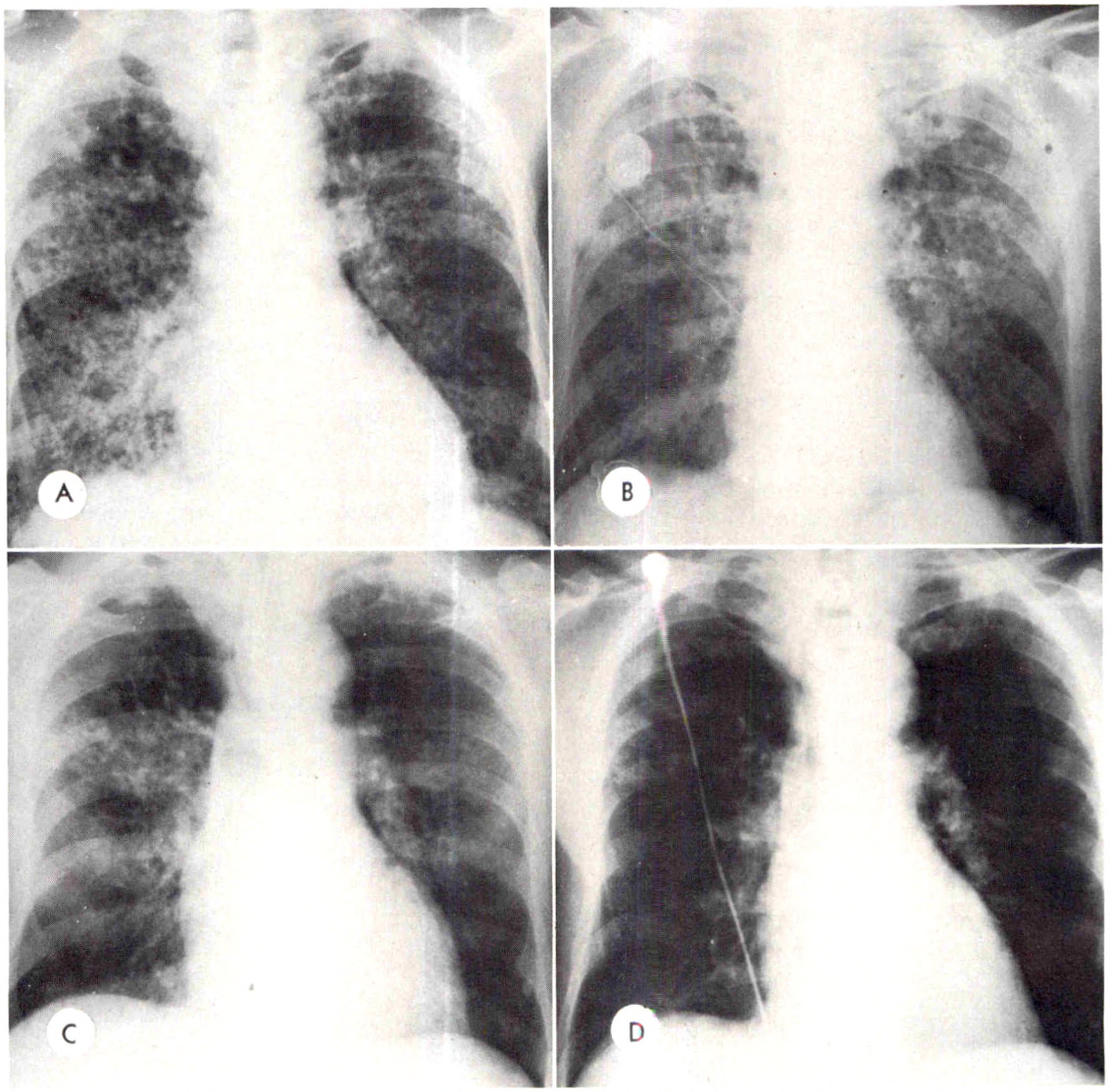


FIG. 5. Case v. (*A*) September 17, 1973; (*B*) September 25, 1973; (*C*) September 28, 1973; and (*D*) October 5, 1973. A series of 4 posteroanterior chest roentgenograms showing a diffuse infiltrate with subsequent clearing.

association of pseudotumor of the orbit with pulmonary involvement. Upper respiratory involvement and facial granulomatous disease may pre-exist or be unassociated with pulmonary or renal disease.[8] A combination of severe lung and kidney involvement in the absence of upper respiratory tract disease was described as "decapitated form."[44] Ear involvement causing deafness was reported by Friedmann and Bauer.[23] Foci of necrotizing vasculitis around both veins and arteries may be found in other tissues as well.[4] Any combination of systemic involvement appears possible, and the diagnosis is made on the basis of the typical pathologic features in the tissues examined.

More recently, Liebow[34] defined several kinds of granulomatosis with angiitis on the basis of histologic structure and other features of the natural history: (1) classical Wegener's granulomatosis; (2) limited angiitis and granulomatosis of the Wegener type; (3) lymphomatoid granulomatosis;

(4) necrotizing "sarcoid" angiitis and granulomatosis; and (5) bronchocentric granulomatosis.

The most frequently described finding on chest roentgenograms has been multiple nodules,[21,32] ranging in size from several mm. to 9 cm., but more often between 2 and 4 cm. Solitary nodules are less common. While some reports mention a predilection for the mid- and lower lung regions,[10,27] others find no zone predilection.[21,26] Central necrosis with ulceration into bronchioles may cause cavitation of some of the nodules,[22,31] and the cavities thus created tend to maintain thick inner walls,[26] although occasionally they may be thin-walled.[27,35] The outline of the nodules may be sharply defined,[41] but is often "shaggy," due to infection or hemorrhage spreading into the alveolar structures surrounding the essentially interstitial granulomatous process characteristic of the disease.[45] In the same way, the original nodule may become unrecognizable, and the lesion may appear as an ill-defined infiltrate.

In an attempt to establish the approximate incidence of the more common findings on chest roentgenograms in W.G., we reviewed 8 series published in Radiology Journals[7,26,27,33,35,36,46,50] after Felson and Braunstein's review of 1958[20] and added their cases. Of the total of 77 cases thus collected, 36 presented with multiple nodules, 22 with localized infiltrates, 14 with solitary nodules, and, in 31, the nodules were cavitated. Cases of Churg and Strauss granulomatosis were included in some of these reports, but not those of periarteritis nodosa.

Several cases with diffuse miliary-type opacities have been variously reported as "generalized infiltrative and nodular densities"[7,13] or "diffuse woolly opacities;"[36] they most likely represent numerous "shaggy" nodules—interstitial nodules with peripheral alveolar involvement—as in our Case v. Pulmonary consolidation as a direct manifestation of W.G. is also uncommon,[26] although it was present in 2 of our 5 cases with pulmonary involvement.

Many other pulmonary or pleural findings have been described in cases with W.G., however, many were possibly due to complications and not to the disease itself: lobar collapse,[20] bronchiectasis,[20] pleural effusion,[19,26,27,28] pulmonary edema,[20] and pneumothorax.[16,26] Hilar masses may simulate bronchogenic carcinomas,[26] while lymph node involvement due to W.G. is unusual.[20]

The correct diagnosis can, at best, be suggested on the basis of the roentgenograms, but few other procedures have proven more helpful short of thoracotomy. Bronchoscopy was nondiagnostic in 16 patients reported by Allen and Moen,[3] while lung biopsy was diagnostic in 1 of their 4 patients biopsied. Two cases of W.G. were diagnosed by percutaneous needle biopsy by Sinner.[45] In our Case 11, a bronchial brushing was negative. With the increasing use of cytologic diagnosis of lung lesions by needle biopsy or bronchial brushing, earlier diagnosis of W.G. may also be facilitated.

The roentgenograms are extremely important in following the patient's clinical course and response to therapy. The disease was almost invariably fatal up to the mid-fifties and in Walton's series[47] of 56 patients, only 18 per cent were alive after 1 year, the average life span being 5 months. The introduction of corticosteroids brought temporary remissions with roentgenologic improvement, but did not significantly alter the course of the disease.[6] In Hollander and Manning's[29] review of 26 patients treated with steroids, 34 per cent survived 1 year and the average life span had increased to 12.5 months. The first reports of complete remission came in the late sixties, with the advent of immunosuppressive therapy. Although the etiology of the disease is still obscure, it seems that a hypersensitivity phenomenon is involved,[17,18] and a variety of cytotoxic agents have had favorable therapeutic responses.[40] Cyclophosphamide[17,18,37] and azathioprine[9,14,24,30] have been most widely used. In 1970, Aldo

et al.[2] reported 80 per cent of their 15 patients alive after 1 year; in 1973 Fauci and Wolff[18] had 12 of 15 patients alive and in complete remission up to 63 months.

Complete remission following treatment with a combination of immunosuppressive and steroid agents was seen in 3 of our 5 patients with pulmonary involvement. The importance of early diagnosis and treatment becomes evident, and the radiologist has an important role to play.

As the variety of roentgenologic findings is recognized, suggesting the diagnosis on the basis of the roentgenograms becomes more difficult.[39] There are certain patterns, however, that have been described more commonly, and these should be well-known to the radiologist, so that together with the clinical findings an early diagnosis can be made.

SUMMARY

Ten patients with Wegener's granulomatosis are reported, 5 of whom had roentgenographic manifestations in the chest. Only 2 patients had pulmonary nodules, the manifestation more commonly described in the literature. Two patients had lobar or segmental consolidation and 1 presented with a diffuse reticulonodular pattern.

The variety of roentgenographic manifestations and the recent developments in the understanding and treatment of necrotizing vasculitis are discussed.

The role of the radiologist in early diagnosis is emphasized in view of the improved prognosis with current methods of treatment.

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REFERENCES

1. AACH, R., and KISSANE, J. Wegener's granulomatosis. *Am. J. Med.*, 1970, *48*, 496–502.
2. ALDO, M. A., BENSON, M. D., COMERFORD, F. R., and COHEN, A. S. Treatment of Wegener's granulomatosis with immunosuppressive agents. *A.M.A. Arch. Int. Med.*, 1970, *126*, 298–305.
3. ALLEN, A. R., and MOEN, C. W. Wegener's granulomatosis. *J. Thoracic & Cardiovasc. Surg.*, 1965, *49*, 388–397.
4. ANDERSON, W. A. D. Pathology. Volume I, and II. C. V. Mosby Company, St. Louis, 1971, pp. 502; 737.
5. ASIN, H. R., WAGENAAR, J. P., and SWIERENGA, J. Mitigated form of Wegener's disease. *Scandinav. J. Resp. Dis.*, 1972, *53*, 367–374.
6. BEIDLEMAN, B. Wegener's granulomatosis: prolonged therapy with large doses of steroids. *J.A.M.A.*, 1963, *186*, 827–830.
7. BISCHOFF, M. E. Noninfectious necrotizing granulomatosis: pulmonary roentgen signs. *Radiology*, 1960, *75*, 752–756.
8. BLATT, I. M. *et al.* Fatal granulomatosis of respiratory tract. *A.M.A. Arch. Otolaryng.*, 1959, *70*, 707–757.
9. BOURONCLE, B. A., SMITH, E. J., and CUPPAGE, F. E. Treatment of Wegener's granulomatosis with imuran. *Am. J. Med.*, 1967, *42*, 314–318.
10. CARRINGTON, C. B., and LIEBOW, A. A. Limited forms of angiitis and granulomatosis of Wegener's type. *Am. J. Med.*, 1966, *41*, 497–527.
11. CASSAN, S. M., COLES, D. T., and HARRISON, E. G., JR. Concept of limited forms of Wegener's granulomatosis. *Am. J. Med.*, 1970, *49*, 366–379.
12. CASSAN, S. M., DIVERTIE, M. B., HOLLENHORST, R. W., and HARRISON, E. G., JR. Pseudotumor of orbit and limited Wegener's granulomatosis. *Ann. Int. Med.*, 1970, *72*, 687–693.
13. CASTLEMAN, B., and MCNEELY, B. U. Case records of MGH. *New England J. Med.*, 1969, *280*, 828–834.
14. CHOY, D. S. J., GOULD, W. J., GEARHART, R. P., and SAUER, J. Remission in Wegener's granulomatosis treated with steroids and azathioprine. *New York J. Med.*, 1969, *69*, 1205–1209.
15. DAVIS, R. W., FETTER, B. F., and YOUNG, W. G., JR. Wegener's granulomatosis: two patients presenting with solitary pulmonary lesions and review of eleven other cases. *Ann. Thoracic Surg.*, 1972, *13*, 427–434.
16. FAHEY, J. L., LEONARD, E., CHURG, J., and GODMAN, G. Wegener's granulomatosis. *Am. J. Med.*, 1954, *17*, 168–179.
17. FAUCI, A. S., WOLFF, S. M., and JOHNSON, J. S. Effect of cyclophosphamide upon immune response in Wegener's granulomatosis. *New England J. Med.*, 1971, *285*, 1493–1496.
18. FAUCI, A. S., and WOLFF, S. M. Wegener's granulomatosis: studies in eighteen patients and review of literature. *Medicine*, 1973, *52*, 535–561.
19. FELDMAN, F., FINK, H., and GRUEZO, Z. Wegener's granulomatosis: report of case in 13-

year-old girl. *A.M.A. Am. J. Dis. Child.*, 1966, *112*, 587–592.
20. Felson, B., and Braunstein, H. Noninfectious necrotizing granulomatosis: Wegener's syndrome, lethal granuloma and allergic angiitis and granulomatosis. *Radiology*, 1958, *70*, 326–334.
21. Fraser, R. G., and Pare, Z. A. P. Diagnosis of Diseases of the Chest. Volume II. W. B. Saunders Company, Philadelphia, 1970, pp. 873–878.
22. Fred, H. L., Lynch, E. C., Greenberg, S. D., and Gonzales-Angulo, A. Patient with Wegener's granulomatosis exhibiting unusual clinical and morphologic features. *Am. J. Med.*, 1964, *37*, 311–319.
23. Friedmann, I., and Bauer, F. Wegener's granulomatosis causing deafness. *J. Laryng. & Otol.*, 1973, *87*, 449–464.
24. Fuller, T., Olsen, N., Block, A., and Cade, J. Wegener's granulomatosis: treatment with heparin in addition to azathioprine and corticosteroids. *Nephron*, 1972, *9*, 225–234.
25. Godman, G. C., and Churg, J. Wegener's granulomatosis: pathology and review of literature. *A.M.A. Arch. Path.*, 1954, *58*, 533–553.
26. Gohel, V. K., Dalinka, M. K., Israel, H. L., and Libshitz, H. I. Radiological manifestations of Wegener's granulomatosis. *Brit. J. Radiol.*, 1973, *46*, 427–432.
27. Gonzalez, L., and Van Ordstrand, H. S. Wegener's granulomatosis. *Radiology*, 1973, *107*, 295–300.
28. Hoch, C. W. Wegener's granulomatosis. *A.M.A. Arch. Otolaryng.*, 1956, *63*, 120–123.
29. Hollander, D., and Manning, R. T. Use of alkylating agents in treatment of Wegener's granulomatosis. *Ann. Int. Med.*, 1967, *67*, 393–401.
30. Israel, H. L., and Patchefsky, A. S. Wegener's granulomatosis of lung: diagnosis and treatment. *Ann. Int. Med.*, 1971, *74*, 881–891.
31. Kornblum, D., and Fienberg, R. Roentgen manifestations of necrotizing granulomatosis and angiitis of lungs. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1955, *74*, 587–592.
32. Leggat, P. O., and Walton, E. W. Wegener's granulomatosis. *Thorax*, 1956, *11*, 94–100.
33. Levin, D. C. Pulmonary abnormalities in necrotizing vasculitides and their rapid response to steroids. *Radiology*, 1970, *97*, 521–526.
34. Liebow, A. A. Pulmonary angiitis and granulomatosis. *Am. Rev. Resp. Dis.*, 1973, *108*, 1–18.
35. Lynch, E. C., Fred, H. L., and Greenberg, S. D. Pulmonary cavitation in Wegener's granulomatosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 521–527.
36. McGregor, M. B. B., and Sandler, G. Wegener's granulomatosis. *Brit. J. Radiol.*, 1964, *37*, 430–439.
37. Novack, S. N., and Pearson, C. M. Cyclophosphamide therapy in Wegener's granulomatosis. *New England J. Med.*, 1971, *284*, 938–942.
38. Patchefsky, A. S., and Israel, H. L. Pulmonary Wegener's granulomatosis. *Ann. Clin. Lab. Sc.*, 1973, *3*, 249–258.
39. Pruszewicz, A., Jaroszewski, F., and Szmeja, A. Radiological lung changes in Wegener's syndrome. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1970, *112*, 204–211.
40. Raitt, J. W. Wegener's granulomatosis: treatment with cytotoxic agents and adrenocorticoids. *Ann. Int. Med.*, 1971, *74*, 344–356.
41. Rogers, J. V., Jr., and Roberto, A. E. Circumscribed pulmonary lesions in periarteritis nodosa and Wegener's granulomatosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 88–93.
42. Roghair, G. D., and Ross, P. Wegener's granulomatosis. *Brit. J. Radiol.*, 1970, *43*, 216–217.
43. Roobaert, N., Courtoy, M., Mockel, J., Bonnyns, M., Rutsaert, J., Flament, J., Ledoux, M., Brihaye, M., and Sternon, J. Granulomatose de Wegener et Granulome Malin Centro-facial. *Semaine Hôp. Paris*, 1973, *49*, 947–953.
44. Schmidt, P., Obiditsch-Mayer, I., Zazgornik, J., Kopsa, H., and Kotzaurek, R. Wegenersche Granulomatose ohne Beteiligung des oberen Respirationstraktes. *Fortschr. Med.*, 1973, *91*, 43–47.
45. Sinner, W. N. Transthoracic needle biopsy of small peripheral malignant lung lesions. *Invest. Radiol.*, 1973, *8*, 305–314.
46. Smith, S. R., Smith, J. W., and Theros, E. G. Wegener's granulomatosis: lymphomatoid variant. *Radiology*, 1971, *98*, 439–444.
47. Walton, E. W. Giant-cell granuloma of respiratory tract: Wegener's granulomatosis. *Brit. M. J.*, 1958, 265–270.
48. Wegener, F. Über generalisierte septische Gefässerkrankungen. *Verh. Deutsch. Ges. Path.*, 1936, *29*, 202–210.
49. Wegener, F. Über eine eigenartige rhinogene Granulomatose mit besonderer Beteiligung des Arteriensystems und der Nieren. *Beitr. path. Anat.*, 1939, *102*, 36–68.
50. Wieners, H., and Hilweg, D. Lungenveränderungen bei der "Pneumogenen Granulomatose" (PG). *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nurlearmedizin*, 1967, *106*, 77–89.
51. Zeek, P. M. Periarteritis nodosa and other forms of necrotizing angiitis. *New England J. Med.*, 1953, *248*, 764–772.

PERCUTANEOUS PULMONARY NEEDLE BIOPSY*

REPORT OF 350 PATIENTS

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PERCUTANEOUS needle aspiration biopsy of the lung has been practiced for many years following the introduction of this procedure by Leyden in 1883[6] and Menetrier in 1886.[10] The procedure has been attended by varying degrees of enthusiasm or condemnation, with objections including possible complications such as pneumothorax, hemorrhage, air embolus, tissue specimen inadequacy, and tumor cell dissemination. Most of these objections have been overcome by major improvements which include the use of: improved needles;[23] pneumothorax catheters;[17] needle guidance instruments;[18] improved cytologic techniques;[4,15,20] and image amplifier fluoroscopy for visual guidance during the biopsy procedure. Some individuals have categorically condemned the procedure or urged extreme caution mainly because of the hazard of disseminating tumor cells along the needle tract. This appears to be without any serious foundation, since only 2 well documented cases of needle tract tumor cell implantation have been described.[2,25] A third case is referred to briefly,[2] but is not documented or described. Two additional cases recently reported[1] are highly questionable, and serious doubts as to their validity have been raised.[12]

Interest in the value of the procedure, predominantly surgical at first, has steadily increased, and more recently reports are to be found with increasing frequency,[9,13,14,19,21,22,26,27] further attesting to its usefulness as a radiologic procedure. Pulmonary needle aspiration biopsy is an appropriate technique for the radiologist because of his familiarity with image-intensified fluoroscopy, depth localization techniques, surgical skills acquired with needle puncture methods for vascular procedures, and experience in the differential diagnosis of pulmonary disease.

The purpose of this paper is to describe the experience with percutaneous pulmonary needle aspiration biopsy performed on 350 patients, exclusively by radiologists, over the past 4 years.

MATERIAL

Included in the group of 350 patients were 257 males, ranging in age from 22 to 85 years, and 93 females, ranging in age from 23 to 86 years. The roentgenograms showed at least 1 localized lesion in the chest, with the smallest area measuring 0.5 cm. in diameter and the largest measuring greater than 10 cm. The majority of the patients were referred for needle biopsy only after many conventional diagnostic methods (bronchoscopy; scalene node biopsy; sputum analysis, including cytology and cultures; skin sensitivity tests; and blood serology examinations) failed to furnish an etiologic diagnosis. Several patients who were considered poor risks for anesthesia, bronchoscopy, or open thoracotomy were included in the group. The biopsies were performed not only by senior members of the staff, but also by several members of the resident staff. All patients were hospitalized for the biopsy procedure.

Indications for needle aspiration biopsy were as follows: (1) medical contraindications to open thoracotomy or a patient's refusal of the thoracotomy; (2) inoperable

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presumptive carcinoma in which tissue diagnosis for preoperative irradiation or chemotherapy was required; (3) confirmation of a suspected benign localized disease; (4) confirmation of metastatic disease; (5) bilateral or other inoperable disease; (6) unresolved pneumonia; (7) confirmation of a bronchogenic carcinoma in a marginal risk patient in whom a positive diagnosis would add weight to the indication for surgery; (8) to obtain living cells for tissue culture, permitting studies of radiation and chemotherapeutic sensitivity;[8] (9) to obtain specimens for the proper identification of bacteria, fungi, or parasites prior to the use of specific chemotherapeutic agents;[7,16] (10) in short, any persistent localized lesion of the lung which could not be diagnosed adequately by conventional methods.

Contraindications to the procedure included: (1) systemic bleeding tendency, except in the most urgent circumstances; (2) suspected vascular lesions such as an arteriovenous malformation or a Rasmussen aneurysm; (3) previous pneumonectomy on either side; (4) far advanced or severe pulmonary hypertension (which would increase the risk of post-biopsy bleeding); (5) advanced pulmonary emphysema, particularly if pulmonary function studies indicated that even a small pneumothorax on one side would be hazardous; (6) inability of the patient to cooperate (except where general anesthesia might be used in extreme circumstances); (7) patient's refusal to have definitive treatment regardless of biopsy results; (8) suspected *Echinococcus* cyst which might disseminate following needle aspiration biopsy.

TECHNIQUE

The location of the lesion is judged by means of appropriate posteroanterior, lateral, oblique, and planigraphic films. These preliminary studies are obtained with the patient recumbent, since the procedure is performed with the patient in this position. By caliper measurements of the patient, film measurements in various projections allowing for magnification, and tomographic measurements of the lesion in relation to the anticipated skin entrance site of the needle, the depth of the center of the lesion can be accurately determined.

The patient is usually kept fasting for 3 to 4 hours, and 50 mg. meperidine hydrochloride (Demerol) is injected intramuscularly one-half hour prior to the procedure. A supine position is maintained for the anterior approach and a prone position for the posterior approach, with the involved side of the lung closest to the radiologist. The lesion is localized under fluoroscopic control, using the tip of a hemostat or a metallic marker on the overlying chest wall. After fluoroscopic confirmation of accurate placement, the marker or tip of the hemostat is pressed into the skin so that an imprint in the appropriate intercostal space remains.

The procedure is performed with strict aseptic technique. The skin is cleansed with a germicidal solution, *e.g.*, tincture of quaternary ammonium antiseptic, 1–500. Two per cent Xylocaine hydrochloride (lidocaine) is used for block anesthesia, and the tissues are infiltrated down to and including the parietal pleura. A small skin and subcutaneous tissue stab incision is made with a No. 11 scalpel blade to facilitate advancing the needle through the chest wall and to facilitate transmission of variations in lung consistency to the palpating fingers without interference from the adjacent surrounding skin. In order to avoid the intercostal vessels, the incision for insertion of the needle is made near the upper border of the lower of the 2 ribs defining the intercostal space.

The biopsy needles are thin-walled and are available in No. 16 and No. 18 gauge sizes, in lengths of 6 inch and 8 inch.[23] It is important to keep the needles constantly sharpened, using a hard oil stone and a carborundum crocus cloth, and to avoid damage to the cutting surface of the needle tips when they are being cleaned prior to sterilization. For small nodules,

it is preferable to employ the No. 18 gauge needle, since large needles tend to push the small nodule to one side rather than to admit direct penetration. The biopsy needle with its contained stylet is inserted directly through the chest wall, the parietal and visceral pleura, and is then advanced directly to the edge of the lesion under fluoroscopic guidance. The patient is allowed to breathe normally, since no advantages have been noted by urging the patient to hold his breath.

When the biopsy needle tip reaches the pulmonary lesion, an increased resistance is often felt. In order to guide the needle tip to the edge of a small lesion, it is advantageous to use a needle holder. A plastic needle holder for advancing the needle tip and manipulating the needle under fluoroscopic control without exposing the radiologist's hands to excessive radiation has been described.[18] A small, well-circumscribed lesion may slide to one side when the needle reaches its surface. If the needle is driven into a lesion with a quick forward thrust, however, a very small lesion can usually be readily transfixed.

By making small, rapid movements with the needle, it can then be readily seen whether or not the needle point has entered a lesion, since the lesion will be displaced by these movements if it has been impaled. Constant superimposition of needle tip and lesion images during respiration makes use of the same principle. If a lesion is fixed or is located near the apex of the hilum, an impaled lesion may not move with respiration or movement of the needle tip. For most lesions, movement of the fluoroscope with the needle in place allows one to use the principle of parallax to confirm the depth of the tip of the needle in relationship to the lesion. Occasionally it may be advantageous to rotate the patient into an oblique position with the needle in place to confirm that the proper depth of needle insertion has been reached. This must be done carefully, since passage of the needle through a heavily-muscled portion of the thorax may cause the needle to change position when exertion during turning causes the muscles to contract. One can also employ a cross-table lateral roentgenogram for further confirmation of the depth of the inserted needle, while the patient is maintained in an immobilized recumbent position. When a lesion is particularly firm, one can often observe fluoroscopically a slight deflection of the needle when the tip reaches the mass. Firm lesions are occasionally more difficult to biopsy than the softer lesions.

Once the needle point has been placed at the desired edge of the lesion or immediately within the lesion, fluoroscopy is discontinued. The stylet is removed from the needle and a 50 cc. glass syringe is immediately attached to the hub. Suction is created by withdrawing the plunger of the syringe with the right hand, while the barrel of the syringe is rotated and advanced with the left hand. The simultaneous advancement and rotation movements cut a sharp core from the lesion. After several rotations and after a sufficient depth is reached (commonly it is preferable to go entirely through the lesion), suction is maintained and the needle is quickly withdrawn. The skin and subcutaneous tissues are gently massaged over the needle opening to avoid air entering the pleural space. Tincture of Collodion and a sterile band-aid are applied over the needle puncture site.

The chest is then examined fluoroscopically during inspiration and expiration, in the recumbent and erect positions, to detect any evidence of pneumothorax. If no evidence of a significant pneumothorax is found on fluoroscopic examination, the patient is permitted to be moved from the table and a posteroanterior erect expiratory "baseline" roentgenogram is obtained. The symptoms of a pneumothorax are explained to the patient. Clinical observation is carried out for 24 hours, and a routine roentgenogram is obtained at the end of this period if no intervening complications have taken place. If a pneumothorax with more than 25 per cent collapse of the lung is noted, or if the patient's prior pul-

monary function studies indicate that even a small pneumothorax may be hazardous, a pneumothorax catheter is immediately inserted and attached to a Heimlich valve.[3,17] The air is immediately removed and the patient is observed with films exposed at 1, 4, and 24 hours. If, after a period of 24 hours, the lung remains completely expanded, the pneumothorax catheter is removed. If, however, there is a continuous air leak as determined by the patient's symptoms of persistent dyspnea, and serial roentgenograms have revealed that the lung has continued to collapse, the pneumothorax catheter is attached to an Emerson pump with continuous suction for an additional period of 24 to 48 hours.

Table I

NEEDLE ASPIRATION LUNG BIOPSY
ANALYSIS OF 410 BIOPSIES IN 350 PATIENTS

44 patients had 2 biopsies
16 patients had 3 biopsies

350 patients—60 lost to follow-up

290 patients had proven diagnosis:
230 patients proven to have malignancy
190 patients had positive needle biopsy, a biopsy success rate of 83 per cent
60 patients proven to have benign disease
44 patients had positive needle biopsy, a biopsy success rate of 73 per cent

234 of 290 patients had positive needle biopsy for malignant or benign disease, a biopsy success rate of 81 per cent

SPECIMEN MATERIAL

The accuracy of cytologic studies of pulmonary aspiration biopsy depends on adequate amounts and proper handling of the tissue material obtained. The cell material within the needle is partially expressed on glass slides to make direct smears, which are immediately fixed in 80 per cent alcohol. The stylet is also inserted into the needle and any remaining tissue is expressed directly into a jar containing an 80 per cent alcohol solution. In addition, a small amount of material is expressed into a tube of serum broth, and routine cultures for bacteria and fungi are obtained on all cases. The syringe and the lumen of the needle are also rinsed with the 80 per cent alcohol solution to collect any tissue particles that may have been sucked into the syringe or may have remained in the lumen of the needle. It is important to be expedient and to take great care in transferring the tissues from the syringe to the slide in order to avoid cellular destruction and degeneration.

All smears are stained by the Papanicolaou method. Tissue fragments are embedded in 3 per cent bacterial agar and are submitted for routine paraffin embedding, and hematoxylin and eosin staining. If sufficient material remains in the preservative alcohol, it is centrifuged and the centrifugate prepared in the same manner as the tissue fragments. If, however, the preservative alcohol is clear, it is filtered through a millipore filter and stained with a Papanicolaou's stain.

RESULTS

The results of 410 biopsy procedures performed on 350 patients are shown in Table 1. Forty-four patients were subjected to 2 biopsies, and 16 patients were biopsied 3 times in an effort to obtain satisfactory specimens.

The correlation of surgical exploration, surgical pathology, postmortem examinations, cultures, clinical course, and roentgenologic findings served as the standard against which the success of the biopsy procedure was evaluated. From a study of available information, 60 of the 350 patients subjected to pulmonary needle biopsy could not be adequately followed to substantiate an accurate diagnosis. Definite and accurate confirmation of the diagnosis was obtained in the remaining 290 patients. One hundred and ninety patients had a positive biopsy for malignancy, while 40 patients were not successfully diagnosed by needle biopsy. Forty-four patients had a positive biopsy for benign disease, while 16 patients were not success-

TABLE II

NEEDLE ASPIRATION LUNG BIOPSY
ANALYSIS OF MALIGNANT NEOPLASMS
PROVEN BY NEEDLE BIOPSY

Total		190
Carcinoma		184
Others		6
Neurofibrosarcoma	1	
Rhabdomyosarcoma	1	
Dermatofibrosarcoma	1	
Fibrosarcoma	1	
Leiomyosarcoma	1	
Giant cell tumor	1	

fully diagnosed by needle biopsy. Two hundred and thirty-four patients out of 290 patients had a positive diagnosis made by needle biopsy, representing a success rate of 81 per cent.

Of the 190 patients with substantiated positive needle biopsies for malignancy, 184 had carcinomas and 6 other lesions (Table II). The carcinomas included well-differentiated squamous cell and adenocarcinomas, as well as undifferentiated anaplastic carcinomas. The other lesions consisted of fibrosarcoma (1), rhabdomyosarcoma (1), dermatofibrosarcoma (1), neurofibrosarcoma (1), leiomyosarcoma (1), and giant cell tumor (1). There were 40 patients in whom a diagnosis was not made by needle biopsy and who were later proven to have malignancy (Table III). Thirty-four had carcinomas, while the remaining 6 had Hodgkin's disease (1), pseudolymphoma (1), metastatic choriocarcinoma (1), leiomyosarcoma (1), and metastatic osteogenic sarcoma (2). In this group there were 18 patients in whom satisfactory biopsies were not obtained at the time of biopsy. Twenty-two tissue specimens were thought to be adequate at the time of biopsy; however, the material was not adequate to allow the pathologist to make a definite diagnosis. Normal lung specimens were classified as "unsatisfactory." The size of the lesion was not a factor in the success of obtaining or failing to obtain a positive biopsy. The lesions of these 22 patients ranged from 1 to 7 cm. in diameter.

TABLE III

NEEDLE ASPIRATION LUNG BIOPSY
ANALYSIS OF PATIENTS WITH PROVEN MALIGNANCIES
IN WHOM A NEEDLE ASPIRATION
BIOPSY WAS NOT SUCCESSFUL

Carcinoma	34
Hodgkin's disease	1
Pseudolymphoma	1
Osteogenic sarcoma	2
Choriocarcinoma	1
Leiomyosarcoma	1

Twenty-two specimens were thought to be adequate at the time of biopsy. However, the material was not adequate to allow the pathologist to make a definite diagnosis. Normal lung specimens were classed as "unsatisfactory."

Forty-four patients had a positive biopsy diagnosis of a benign process (Table IV).

TABLE IV

NEEDLE ASPIRATION LUNG BIOPSY
ANALYSIS OF PROVEN BENIGN LESIONS

Proven by needle biopsy:		44
Granulomatous disease		15
Tuberculosis	6	
Histoplasmosis	2	
Cocci	2	
Sarcoid	2	
Nonspecific	3	
Bronchopneumonia		12
Lung abscess (bacterial)		7
Chronic inflammatory		3
Pleural scar		1
Aspergillosis		1
Amyloidosis		1
Amebiasis		1
Emphysema		2
Pancreatic fluid		1
Benign lesions not proven by needle biopsy, but proven by other methods:		
Granulomas		2
Hamartomas		2
Tuberculosis		4
Cocci		1
Sarcoid		3
M. kattey		1
Fibroxanthoma		1
Lipoid pneumonia		1
Subpleural lipoma		1
Total		16

In this group there were 15 cases of granulomatous disease which included tuberculosis (6), coccidioidomycosis (2), histoplasmosis (2), sarcoid (2), and other nonspecified granulomatous disease (3). There were 12 cases of bacterial bronchopneumonia, 7 cases of bacterial lung abscess, and 3 cases described as "chronic lung disease" of unspecified etiology. The remaining cases included pleural scar (1), amebiasis (1), aspergillosis (1), amyloidosis (1), emphysema (2), and pancreatic fluid (1). Sixteen cases out of 60 benign lesions were unproven by needle biopsy, but were subsequently proven by other means—surgery, postmortem examination, etc. These included granulomas (2), hamartomas (2), *M. tuberculosis* (4), *M. battey* (1), coccidioidomycosis (1), sarcoidosis (3), fibroxanthoma (1), lipoid pneumonia (1), and subpleural lipoma (1).

REPORT OF CASES

Case 1. M.S., 48 year old Negro female. Chest roentgenogram revealed enlargement of the left hilum, with a lesion in the left upper lobe (Fig. 1). Routine work-up, including sputum cytology and cultures, was noncontributory. A brush biopsy following bronchoscopy, a scalene node biopsy, and mediastinoscopy yielded no evidence of any malignant cells. Needle aspiration biopsy proved the lesion to be a typical epidermoid carcinoma of the lung.

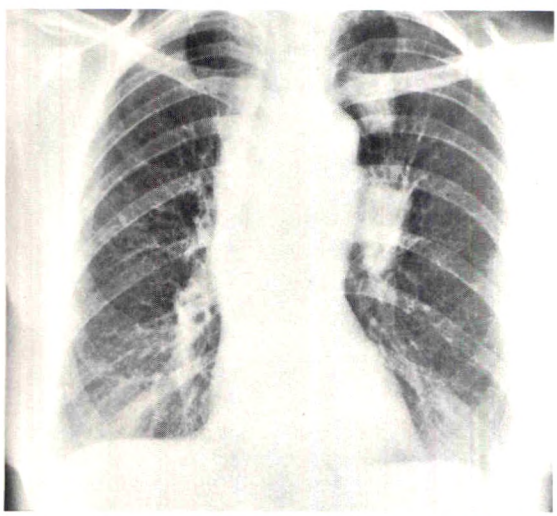


Fig. 1. Case 1. Chest roentgenogram showing enlargement of the left hilum with left upper lobe lesions. Typical epidermoid carcinoma found on needle aspiration biopsy.

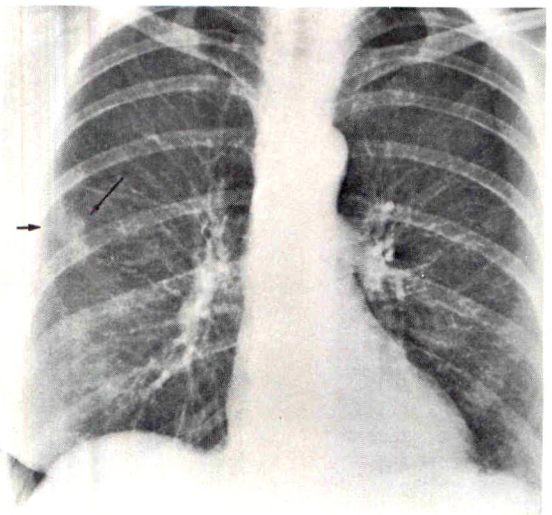


Fig. 2. Case 11. Chest roentgenogram showing peripheral ½ cm. diameter nodule in right mid-lung field (arrows). A typical adenocarcinoma was found on needle aspiration biopsy.

Case 11. P.H., 48 year old Negro female. A 0.5 cm. ill-defined nodule was found on a routine chest roentgenogram (Fig. 2). All laboratory and clinical findings were noncontributory. Needle aspiration biopsy proved this to be an adenocarcinoma.

Case 111. M.M., 68 year old white male. The chest roentgenogram showed a right upper lobe mass (Fig. 3*A*). Needle aspiration biopsy proved the lesion to be a squamous cell carcinoma. A pneumothorax following the biopsy was immediately successfully treated, using a pneumothorax catheter with a Heimlich valve (Fig. 3*B*).

Case 1v. H.G., 32 year old Negro male who was relatively asymptomatic. Diffuse disseminated, ill-defined lesions were noted in both lungs (Fig. 4). Needle aspiration biopsy showed typical sarcoid granuloma.

Case v. C.J., 63 year old white male. Patient had findings of old tuberculosis and bullous emphysema. A rounded opacity was noted within one of the emphysematous areas in the left upper lobe (Fig. 5). Needle aspiration

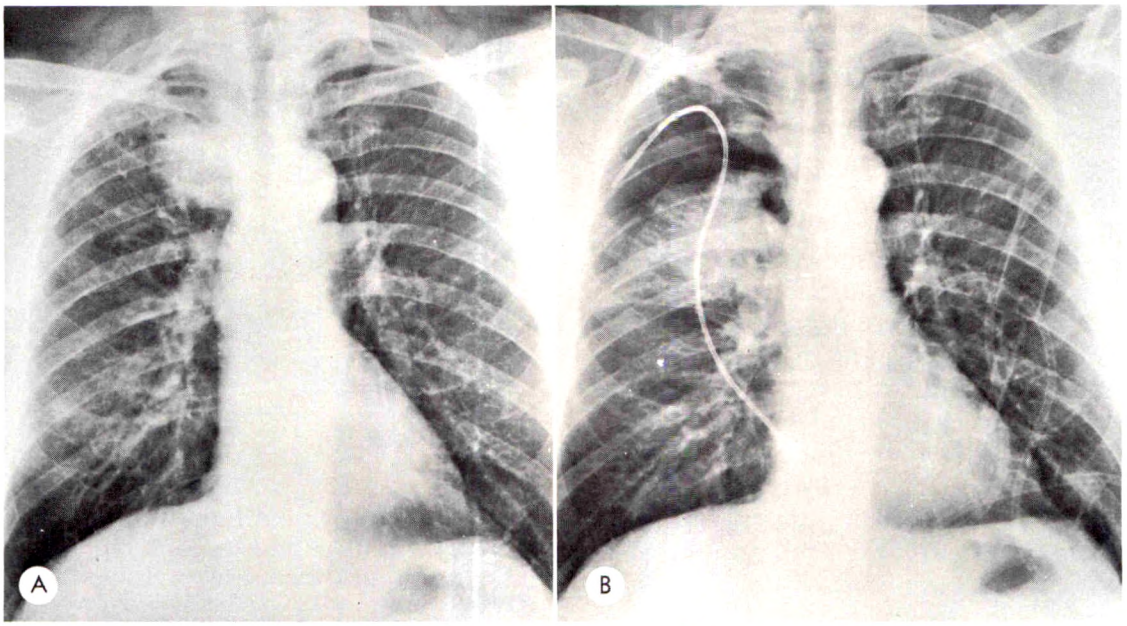


Fig. 3. Case III. (*A*) Chest roentgenogram showing right upper lobe mass. Squamous cell carcinoma was found on needle aspiration biopsy. (*B*) Symptomatic pneumothorax treated immediately with pneumothorax catheter and Heimlich valve.

biopsy showed typical mycelia associated with an aspergilloma.

Case VI. E.G., 35 year old white female. Patient admitted because of dyspnea and fever. The lesion in the right lung field (Fig. 6) failed to respond to antibiotics. Stool cultures were

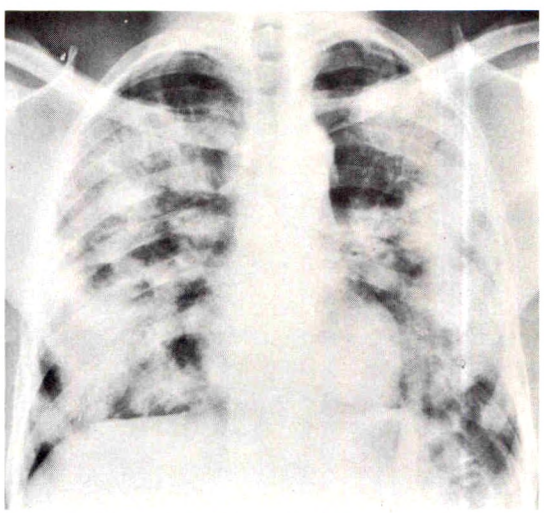


Fig. 4. Case IV. Bilateral diffuse, disseminated, poorly-defined lesions. Typical sarcoid granuloma was found on needle aspiration biopsy.



Fig. 5. Case V. Chest roentgenogram of patient with chronic pulmonary tuberculosis and bullous emphysema, particularly in both upper lobes. There is a mass-like lesion in the cavity in the left upper lobe. Typical mycelia associated with an aspergilloma were found on needle aspiration biopsy.

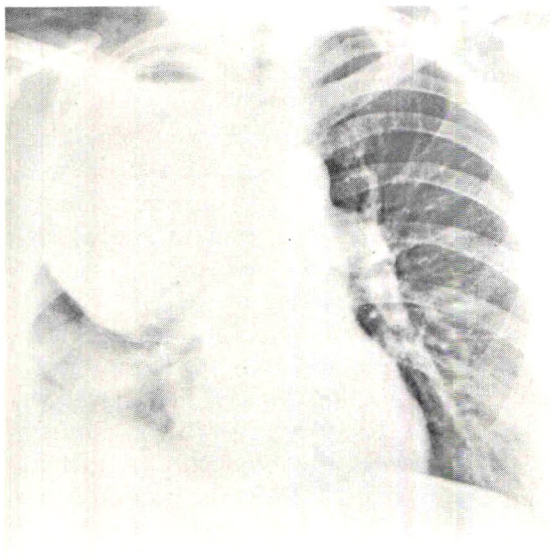


Fig. 6. Case VI. Chest roentgenogram of patient with right upper lobe mass-like lesion and infiltrate in right lower lobe. The fluid removed from the right upper lobe lesion contained trophozoites on wet mount, typical of amebiasis.

negative. Patient was suspected of having amebiasis, since she had visited Mexico several times. Needle aspiration biopsy of the right upper lobe lesion showed trophozoites on wet mount typical of amebiasis.

DISCUSSION

For practical purposes, the pathologic interpretation of a needle aspiration biopsy can be grouped into several categories: (1) the specimen may be interpreted as neoplastic, *i.e.*, a malignancy thus providing a definite diagnosis, and, on more rare occasions, a benign neoplasm may be found; (2) the specimen may be interpreted as providing sufficient information to permit the diagnosis of a benign non-neoplastic process; (3) biopsy specimen may appear adequate; however, if a definite diagnosis cannot be made, *i.e.*, benign or malignant, the biopsy has to be considered "unsatisfactory." Normal lung tissue cannot be accepted as a normal biopsy, but is considered "unsatisfactory." The unsatisfactory biopsy must be repeated, particularly when there is a suspicion of possible malignancy, since experience has shown that the number of unsatisfactory specimens will become satisfactory with second and third biopsy procedures. Of the 44 lesions subjected to 2 biopsy procedures, the diagnosis was established in 20 by the second procedure. Of the 16 patients subjected to 3 biopsies, the diagnosis was made in 9 by the third procedure. Just as multiple sputum cytology specimens improve the yield, so do multiple pulmonary biopsies increase the chances for a correct diagnosis.

When dealing with a persistent, localized pulmonary lesion, the primary concern is to exclude malignancy. However, definite benign diagnosis would provide information to justify further observation before surgery, particularly if the patient is a poor risk. A specific histologic or culture diagnosis was provided in 234 of 290 patients —a success rate of 81 per cent.

Of the cases of proven malignancy, 40 patients were not diagnosed by needle biopsy. In 18 of these patients, the biopsy specimens were not satisfactory at the time of biopsy. In 22 other patients, the specimens were thought to be satisfactory at the time of biopsy; however, the pathologist could not make a definite diagnosis, and these were therefore classified as "unsatisfactory."

Of the benign lesions, 44 out of 60 were diagnosed by needle biopsy—a success rate of 73 per cent. In 16 patients the diagnosis could not be made by needle biopsy because the tissue submitted to the pathologist did not allow a definite diagnosis, and, again, these were classified as "unsatisfactory." Thus, when any biopsy did not supply adequate tissue to make a definite diagnosis of benign or malignant disease, the biopsy was considered "unsatisfactory." Adequate normal lung tissue was considered as "unsatisfactory" and was not classified as an absence of specific benign disease or absence of malignancy.

Table V is an analysis of the complications encountered in 410 needle aspiration procedures performed on 350 patients. One hundred and nine (26.6 per cent) pneumothoraces were observed, and 30 (7.3 per

TABLE V

NEEDLE ASPIRATION LUNG BIOPSY
ANALYSIS OF COMPLICATIONS

(410 biopsy procedures)

	No.	Per Cent
Pneumothorax	109	26.60
Tube required	30	7.30
Subcutaneous emphysema	5	1.25
Hemoptysis—self-limited	4	1.00
Hemothorax	6	1.50
Hematoma	2	.50
Hemorrhage—self-limited focal	2	.50
Seizure	1	.25
Vomiting	1	.24
Hepatic tissue	1	.24
Death (cardiac arrest; hemorrhage—2 days later)	2	.50

cent) required intercostal tube insertions for removal of the air. Most of the pneumothoraces were small, completely asymptomatic, and resolved spontaneously. The 30 cases requiring closed intercostal drainage to re-expand the lung included some patients who were dyspneic at rest due to advanced emphysema, with a minimal pneumothorax resulting in increasing dyspnea. With the others, a large pneumothorax accompanied by dyspnea required the immediate institution of closed intercostal tube drainage to speed re-expansion of the lung. The use of a simple pneumothorax catheter and Heimlich valve described previously allows the radiologist to treat immediately any pneumothorax while the patient is still on the fluoroscopic table, thus preventing any complications that might arise from a pneumothorax. An analysis of the incidence of pneumothoraces in those patients biopsied 2 or 3 times on the same date showed that the incidence of occurrence of pneumothorax was greater. Usually it is more prudent to obtain 1 good biopsy, have it analyzed, and then repeat the procedure on a subsequent date, when necessary. Six patients had a hemothorax, and 1 of these patients required intercostal tube drainage. There were 5 cases of moderate subcutaneous emphysema and 4 cases of hemoptysis which were self-limited. One case died shortly after the needle was inserted, and death was attributed to a pneumothorax followed by a cardiac arrest. A second patient died of pulmonary hemorrhage 2 days after the biopsy procedure. Both patients were extremely poor risks, and it might be well to point out that the general physical condition of the patient must be carefully studied prior to instituting any needle biopsy procedure. Cardiac and pulmonary function studies should be included in this evaluation. Serious pneumothorax complications in debilitated patients can usually be averted by immediately inserting a pneumothorax catheter and evacuating air from the pleural space, particularly if a tension pneumothorax is suspected. A recent case report[11] with a review of the literature describes a death due to aspiration (blood in the tracheobronchial tree) following lung biopsy in a patient with idiopathic interstitial fibrosis. The cause of death was tracheal occlusion secondary to clotted blood. It is emphasized that patients selected must be alert enough to clear their secretions and blood clots. Of the other causes of deaths cited, one was due to tension pneumothorax[5] and another was due to air embolism.[24] There was no evidence of any tumor dissemination along the needle tract in any of our cases. No evidence of any spread of any associated pulmonary infections was encountered. However, it is our policy to routinely place a patient on antibiotics, if a biopsy should yield frank pus or if a pneumothorax has occurred requiring intercostal tube drainage.

The success of needle aspiration of the lung depends not only on the accuracy and adequacy of the biopsy, but equally importantly on the skill and meticulous technique of the cytologist, pathologist, and the bacteriologist. The cytologic technician is present during the procedure, so that the biopsy material can be immediately transferred from the needle to the slides and to the 80 per cent alcohol solution without undue delay causing deterioration of the cellular material.

Stress should be placed upon the fact

that a finding of "no malignant cells" must not be taken as absolutely conclusive proof of the absence of malignancy. Even after repeated "satisfactory" biopsies, if a specific diagnosis is not established and the lesion persists, thoracotomy is mandatory, particularly if the patient is in the 30–70 year group. Conversely, the argument might be advanced against the necessity for a needle biopsy, since those patients who prove to have a bronchogenic carcinoma will undergo resection in any event. Knowledge that a lesion is malignant should prompt routine preoperative scalene node biopsies for metastatic disease, as well as bronchoscopy and mediastinoscopy for mediastinal involvement, in order to determine the extent of spread preoperatively. This is a great aid in the planning of the type of operative procedure prior to open thoracotomy. In addition, the fact remains that preoperative irradiation in some patients may provide the only hope of achieving resectability, while in others the cell type and location or extent of the tumor make irradiation the only reasonable therapeutic choice. With newer tissue culture techniques, it may be possible to obtain cellular material to determine both chemotherapeutic and radiotherapeutic sensitivities of neoplastic cells prior to surgery. Most important, however, is the fact that the diagnostic work-up of a patient can be considerably shortened if a diagnosis is made available by an aspiration biopsy relatively early in the course of the evaluation.

In many cases, the delay in arriving at the proper diagnosis, which is a specific responsibility of the physician in charge, may prove to be as long as the patient's delay in consulting him. For better results to be obtained in treatment of carcinoma of the lung, every diagnostic method should be employed within a short period of time—*i.e.*, one week if possible.

The number of positive biopsies tends to increase with the skill of the operator. This is quite evident in a residency training program, where an increase in positive biopsies parallels the increased proficiency that results from a progressive training experience. Perfection must not be expected, and those who would reserve needle biopsy for those lesions which cannot be diagnosed by any conventional means will often add to the unnecessary delay in the urgent treatment of neoplasm. Needle biopsy should be used earlier and more frequently to shorten the diagnostic interval and to allow more prompt and definitive therapy. This is particularly important because many physicians are reluctant to perform open thoracotomies on nondescript, questionable lesions. A needle aspiration biopsy will more often provide a positive direct diagnosis preoperatively, convincing both the patient and the referring physician that an immediate thoracotomy is necessary.

CONCLUSIONS AND SUMMARY

Percutaneous needle aspiration biopsy of pulmonary lesions is a simple, safe, practical, and acceptable diagnostic procedure. The diagnostic yield of this procedure exceeds that of any other method, other than open thoracotomy. With the use of No. 16 or No. 18 thin-walled needles, image amplifier guidance, and needle guidance instruments, all lesions, either in central or peripheral portions of the lung, are accessible for biopsy.

Four hundred and ten needle aspiration biopsies were carried out on 350 patients. Of 290 confirmed cases, 190 were proven to be malignant by needle biopsy and 44 were proven to be benign. Two hundred and thirty-four patients out of 290 were diagnosed by needle biopsy—a biopsy success rate of 81 per cent. A second or third biopsy will significantly increase the number of satisfactory biopsies. The complications of a pneumothorax are of no consequence and can be immediately handled by the radiologist using a pneumothorax catheter. Other complications of subcutaneous emphysema, hemothorax, hematoma, or hemoptysis are usually self-limited and generally require no treatment. However, 2 deaths occurred out of 410 needle aspirations. Extremely debilitated patients should not be subjected to this procedure.

Needle biopsy should be used earlier and more frequently to shorten the diagnostic interval and to allow more prompt and definitive therapy for any persistent lung lesions which cannot be accurately or promptly diagnosed by conventional means.

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REFERENCES

1. Berger, R. H., Dargan, E. L., and Huang, B. L. Dissemination of cancer cells by needle biopsy of lung. *J. Thoracic & Cardiovasc. Surg.*, 1972, *63*, 430–432.
2. Dutra, F. R., and Geraci, C. L. Needle biopsy of lung *J.A.M.A.*, 1954, *155*, 21–24.
3. Heimlich, H. J. Valve drainage of pleural cavity. *Dis. Chest*, 1968, *53*, 282–287.
4. King, E. B., and Russell, W. M. Needle aspiration biopsy of lung-techniques and cytologic morphology. *Acta cytol.*, 1967, *11*, 319–324.
5. Lauby, V. W., Burnett, W. E., Rosemond, G. P., and Tyson, R. R. Value and risk of pulmonary lesions by needle aspiration: twenty-one years experience, *J. Thoracic & Cardiovasc. Surg.*, 1965, *49*, 159–172.
6. Leyden, H. Über infectiose Pneumonie. *Deutsche med. Wchnschr.*, 1883, *9*, 52–54.
7. Lillehei, J. P., Funke, J. L., Drage, C. W., Sharp, H. L., and Burke, B. A. Pneumocystis Carinii pneumonia: needle-biopsy diagnosis and successful treatment. *J.A.M.A.*, 1968, *206*, 596–600.
8. Lindstrom, R. R., Collins, J. D., and Byfield, J. E. Obtaining viable tumor cells through percutaneous pulmonary needle biopsy. *Radiology*, 1970, *94*, 200–203.
9. Linsk, J. A., *et al.* Diagnosis of intrathoracic tumors by thin needle cytologic aspiration. *Am. J. M. Sc.*, 1972, *263*, 181–195.
10. Menetrier, P. Cancer primitif du poumon. *Bull. Soc. anat.*, 1886, *11*, 643.
11. Meyer, J. E., Ferrucci, J. T., Jr., and Janower, M. L. Fatal complications of percutaneous lung biopsy. *Radiology*, 1970, *96*, 47–48.
12. Naylor, B. Personal communication to the Editor. *J. Thoracic & Cardiovasc. Surg.*, 1972, *64*, 324.
13. Nordenstrom, B. New technique for transthoracic biopsy of lung changes. *Brit. J. Radiol.*, 1965, *38*, 550–553.
14. Rabinov, K., Goldman, H., Rosbash, H., and Simon, M. Role of aspiration biopsy of focal lesions in lung and bone by simple needle and fluoroscopy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 932–938.
15. Sabour, M. S., Osman, L. M., Legolvan, P. C., and Ishak, K. G. Needle biopsy of lung. *Lancet*, 1960, *2*, 182–184.
16. Sappington, S. W., and Favorite, G. O. Lung puncture in lobar pneumonia. *Am. J. M. Sc.*, 1936, *191*, 225–234.
17. Sargent, E. N., and Turner, A. F. Emergency treatment of pneumothorax: simple catheter technique for use in radiology department. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 531–535.
18. Sargent, E. N., and Turner, A. F. Needle holder and guide for pulmonary aspiration biopsy. *Radiology*, 1970, *96*, 346.
19. Sinner, W. N. Transthoracic needle biopsy of small peripheral malignant lung lesions. *Invest. Radiol.*, 1973, *8*, 305–314.
20. Smetana, H. F. Needle biopsy in diagnosis. *Am. J. Clin. Path.*, 1954, *24*, 395–405.
21. Stevens, G. M., Weigen, J. F., and Lillington, G. A. Needle aspiration biopsy of localized pulmonary lesions with amplified fluoroscopic guidance. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 561–571.
22. Thompson, J. E. Aspiration biopsy of localized upper zone lung lesions. *Med. J. Australia*, 1969, *2*, 949–952.
23. Turner, A. F., and Sargent, E. N. Percutaneous pulmonary needle biopsy: improved needle for simple direct method of diagnosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 846–850.
24. Westcott, J. L. Air embolism complicating percutaneous needle biopsy of lung. *Chest*, 1973, *63*, 108–110.
25. Wolinsky, H., and Lischner, M. W. Needle track implantation of tumor after percutaneous lung biopsy. *Ann. Int. Med.*, 1969, *71*, 359–362.
26. Zavala, D. C., and Bedell, G. N. Percutaneous lung biopsy with cutting needle: analysis of 40 cases and comparison with other biopsy techniques. *Am. Rev. Resp. Dis.*, 1972, *106*, 186–193.
27. Zelch, J. V., Lalli, A. F., McCormack, L. J., and Belovich, D. M. Aspiration biopsy in diagnosis of pulmonary nodule. *Chest*, 1973, *63*, 149–152.

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AORTOGRAPHIC ANALYSIS OF AORTIC DISSECTION*

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WITH a more aggressive approach to the medical and surgical management of aortic dissection, angiographic study of the aorta and its branches has become indispensable in the diagnosis and classification of the type of aortic dissection. In typical cases, angiographic diagnosis is not difficult. However, recent experience has shown that precise understanding of the anatomic abnormality may be exceedingly difficult in some patients.

The purpose of this paper is to make a comprehensive analysis of aortograms in aortic dissection. A three dimensional approach to the understanding of the dissection will be emphasized.

DEFINITION

The term aortic dissection is preferred to the commonly used term dissecting aneurysm to clearly distinguish dissection from the usual type of aneurysm and false aneurysm. There is much confusion apparently caused by the misnomer dissecting aneurysm. There are many examples of aortic dissection in which the false lumen is so thin that the term aneurysm cannot be justified. Moreover, the term dissecting aneurysm may be misleading because it indicates that the dissection starts from a pre-existing simple aneurysm.

A schematic depiction of aneurysm, false aneurysm and aortic dissection is shown in Figure 1. In the usual type of aneurysm, all 3 layers of the aortic wall protrude outward from the lumen. A false aneurysm is usually produced by trauma and is essentially a perforation or penetration of the aorta, with the wall of the false aneurysm formed by adventitia or surrounding perivascular tissues. In contrast, an aortic dissection is a separation of the intima and adventitia and usually occurs at the junction between the middle and outer thirds of the media. The dissection may involve a localized area of the aortic circumference or may involve the entire circumference. A three barrel type may also occur, with 1 true lumen and 2 false lumens.

An important anatomic feature is that in dissection, the aortic lumen (the true lumen) is not dilated but compressed by the false lumen. This feature is almost universally found in dissection of the descending thoracic and abdominal aortas. An exception may be in the ascending aorta in cases of Marfan's syndrome or in idiopathic cystic medial necrosis of the ascending aorta. In these cases, the ascending aorta may show aneurysmal dilatation with or without dissection, and the true lumen sometimes remains wider than normal.[7]

CLASSIFICATION

Of several classifications,[3,4] that of DeBakey *et al.*[4] has been widely accepted and has been found useful in the angiographic diagnosis and management of aortic dissection (Fig. 2). Type I is the most common (Fig. 3, *A–E*); the dissection begins in the ascending aorta and extends across the aortic arch and often down to the iliac arteries. In Type II, most commonly seen in Marfan's syndrome, dissection is limited to the ascending aorta. In Type III, the dissection begins from the thoracic aorta distal to the left subclavian artery and extends for a variable distance (Fig. 4, *A–D*).

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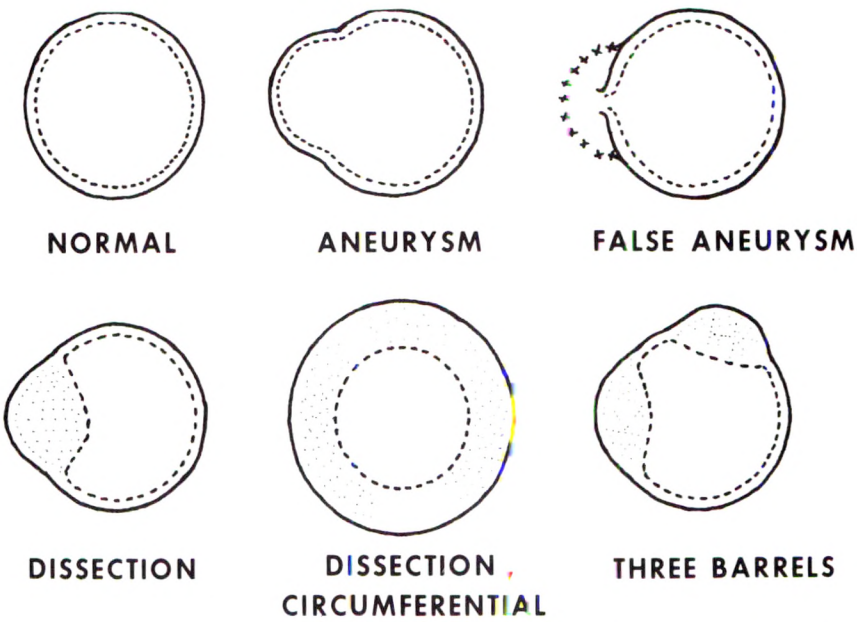


FIG. 1. Schematic depiction of aortic aneurysm, false aneurysm, and dissection. Aorta viewed in cross section. Solid line represents adventitia, broken line intima and dotted area false channel.

TECHNIQUE OF AORTOGRAPHY

We prefer a retrograde femoral approach to aortic catheterization except in cases in which the femoral pulse is absent. In this instance, introduction of the catheter through the brachial or axillary arteries is preferred. Occasionally a combined femoral and brachial approach may be necessary. Venous aortography often proves unsatisfactory because of poor concentration of

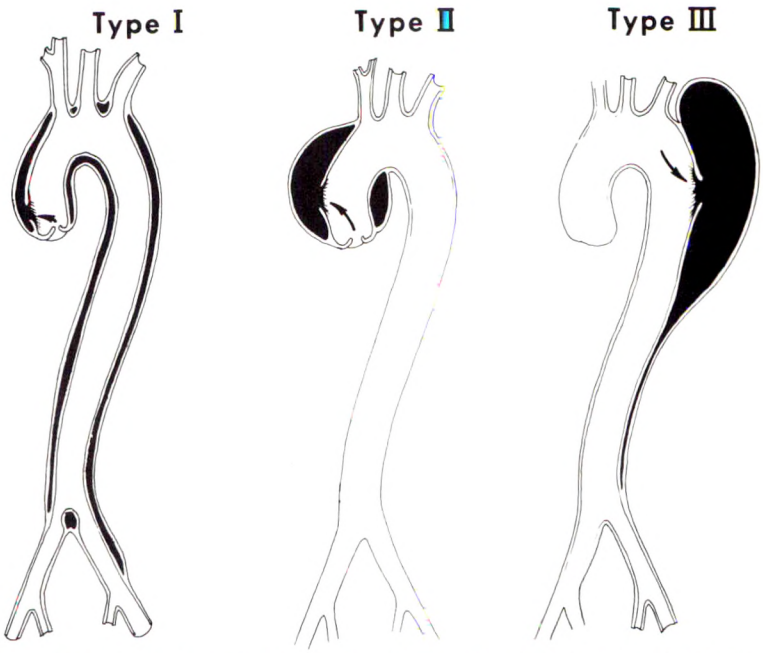


FIG. 2. Types of aortic dissection based upon the classification of DeBakey.

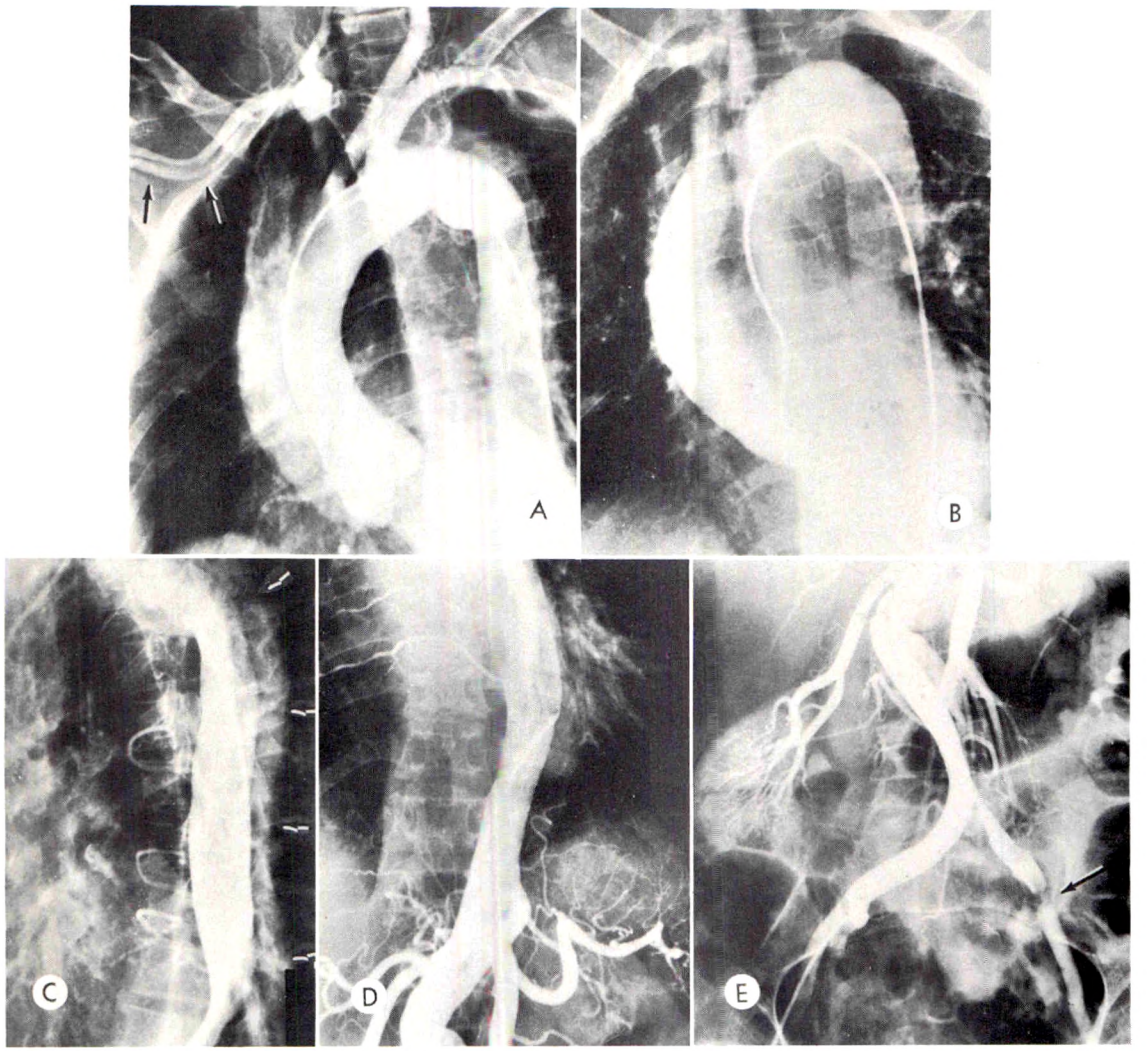


Fig. 3. Type I Aortic Dissection. (*A*) Thoracic aortogram, right posterior oblique projection. Injection into true lumen. Faintly opacified false lumen is located anteriorly and on the right. Dissection extends to the innominate and right subclavian artery (arrows). Marked aortic regurgitation. Note medial position of the catheter in the ascending aorta. (*B*) Injection into false lumen. Innominate artery partially supplied from false channel. (*C*) Descending thoracic aortogram, right posterior oblique projection. Compression of true lumen. False lumen faintly opacified, and located posteriorly and laterally (arrows). (*D*) Thoracic and abdominal aortogram. (*E*) Low abdominal aortogram. Dissection extends to left common iliac artery (arrow). Left renal artery not opacified.

contrast material. Serial films are obtained at the rate of 2 to 3 exposures per second. Cineangiography is not recommended because of relatively poor resolution, particularly in the mediastinal and abdominal areas and because of the limited area of view.

To obtain a complete study, it is important to visualize the aorta from the root of the ascending aorta to the aortic bifurcation and even the iliac arteries. The first injection should be made into the ascending aorta. Biplane anteroposterior and lateral projections are desirable, but we have found that the right posterior oblique projection usually supplies sufficient information. For visualization of the abdominal aorta, an anteroposterior projection is used. Sometimes a lateral view will be needed to provide clear anatomic information. We

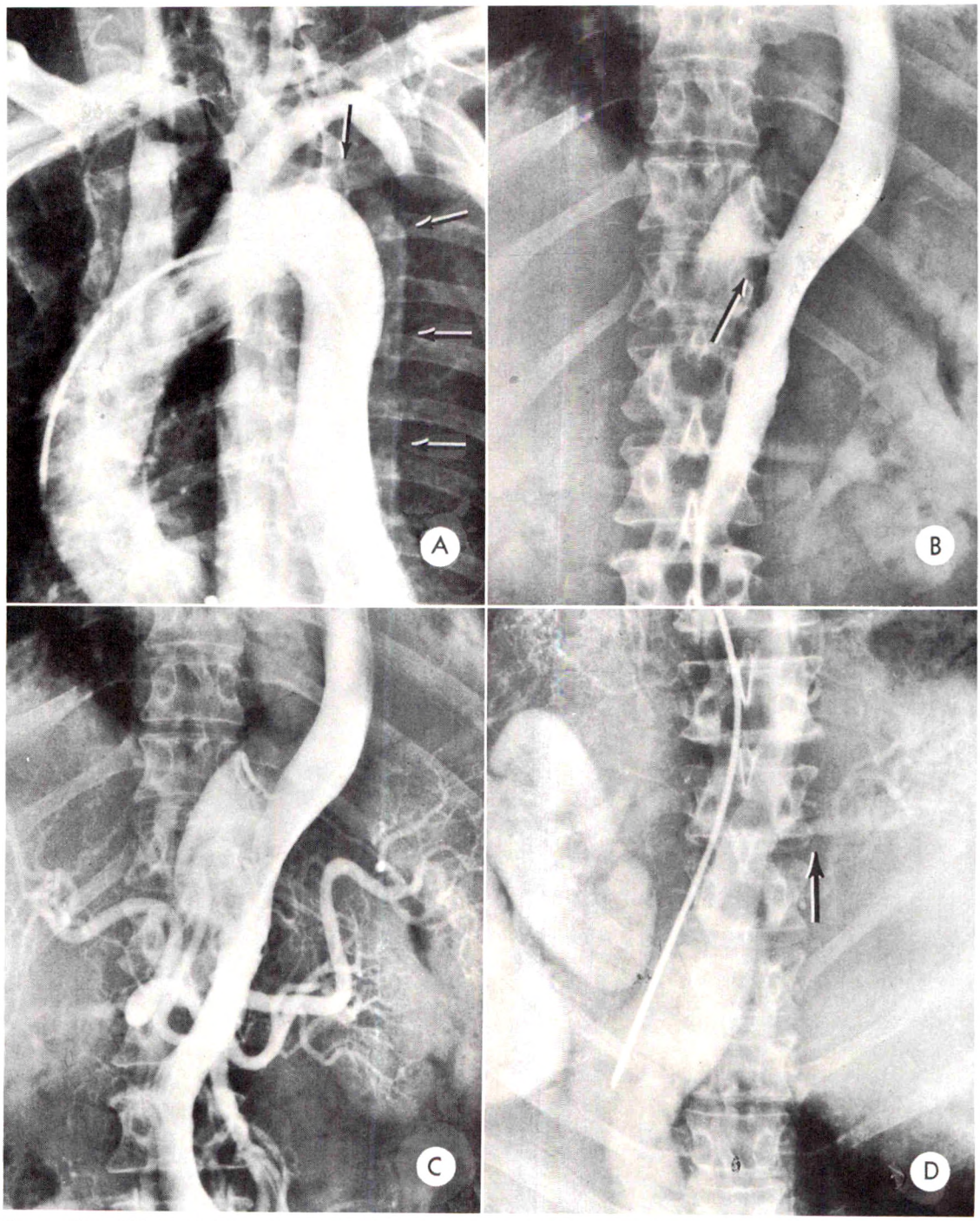


FIG. 4. Type III Aortic Dissection. (*A*) Thoracic aortogram, right posterior oblique projection. False channel is not yet opacified, and is located posteriorly and on the left side (arrows). (*B*) Low thoracic aortogram, anteroposterior projection. False channel has spiraled posteriorly and appears on the right side. Intimal tear is indicated by arrow. (*C*) More contrast material in false channel. Celiac, superior mesenteric and left renal arteries are supplied by true lumen. (*D*) Contrast material in the true lumen has been washed out; false channel is now well opacified and supplies right renal artery (arrow).

have not encountered any serious complications produced in patients with aortic dissection by utilizing retrograde percutaneous femoral angiography.

AORTOGRAPHIC FINDINGS

Aortographic findings are summarized in Table 1. Some of the findings are very reliable and represent aortographic depiction of the actual pathology.

DOUBLE CHANNELS AND LINEAR RADIOLUCENCY

Demonstration of double channels and/or a linear radiolucency within the opacified aorta is the hallmark of aortic dissection. Without these findings, a diagnosis of aortic dissection is subject to error unless there is clear-cut compression of the aortic lumen. It is important to recognize that several projections may be required for the demonstration of these findings. A linear radiolucency, representing the torn or separated intima (plus the inner two-thirds of the media), may be found only when the x-ray beam is tangential to the intima. The probability of such an event is rather small and accounts for the relative infrequency of this finding.

Double channels can be recognized when: (1) a linear radiolucency is visualized separating the 2 channels as described above; (2) the 2 channels are opacified in different density or timing; or (3) catheterization of each lumen is accomplished and contrast material is injected separately.

Aortic dissection may be totally unrecognized in certain projections or when there is simultaneous equal opacification of both channels. *An abnormal catheter position* as described by Cramer and Amplatz,[2] in which the catheter tip curves around the aortic arch and maintains a position on the inner portion of the aorta rather than resting against the outer portion, is a helpful finding. This is particularly true when there is simultaneous opacification of the true and false channels of the ascending aorta and aortic arch. A high degree of opacification of both the true and false channels signifies that there is a re-entry of the false lumen distally accompanied by a high rate of blood flow.

Table I

AORTOGRAPHIC FINDINGS IN AORTIC DISSECTION

I.	Direct Signs
	1. Double channels
	2. Linear radiolucency (torn or separated intima)
	3. Entry
	4. Re-entry
II.	Indirect Signs
	1. Compression of aortic (true) lumen by (false) lumen
	2. Thickening of aortic wall (greater than 5–6 mm., normally 2–3 mm.)
	3. Ulcer-like projection
	4. Abnormal catheter position
III.	Nonspecific Finding
	Aortic regurgitation
IV.	Branch Involvement

ENTRY AND RE-ENTRY

The exact site of entry (the most proximal intimal tear) may be clearly identified. This can be demonstrated by contrast material passing from one lumen to the other, usually from true to false lumen (Fig. 4*C*), by a filling defect caused by unopacified blood passing from the other lumen, or occasionally by the catheter position, which has passed beyond the intimal tear (Fig. 3*A*). Sometimes the exact location of the intimal tear cannot be determined. Fortunately, in the majority of cases, the intimal tear is located in the proximal few centimeters of the ascending aorta in Type I and Type II, and close inspection of the angiogram in this area will often reveal the position of the tear. Similarly, close scrutiny of the aorta in the region of the origin of the left subclavian artery is helpful in identifying Type III dissection. It may be reasonable to consider that the tear is located in the most proximal portion of the demonstrated dissection when it cannot clearly be pointed out. However, it should be recognized that dissection may progress proximally.

The site of re-entry may be single or multiple and is sometimes related to branch

arteries. This finding is described in more detail in the discussion of branch involvement.

Approximately 5 per cent of aortic dissections do not have intimal tears.[9] This supports the theory that the hemorrhage is initiated by ruptured vasa vasorum, rather than intimal tear.

COMPRESSION OF AORTIC (TRUE) LUMEN

This is another characteristic aortographic finding in aortic dissection (Fig. 3*B*). The finding is most dramatic in acute cases where selective injection is made into the narrowed true lumen. As described previously, the true lumen may remain wider than normal in the ascending aorta in cases of Marfan's syndrome or idiopathic medial necrosis of the ascending aorta, and it is sometimes difficult to differentiate between aneurysmal dilatation of the ascending aorta and dissection.[7]

THICKENING OF AORTIC WALL

When the false lumen is not opacified, aortographic findings mentioned above (except for compression of the aortic lumen) will not be found. The false lumen is not opacified when: (1) it is filled with blood clot; (2) the intimal tear is located more distally than the lowermost portion of the obtained film (and the dissection has progressed proximally as well as distally); or (3) there is no intimal tear. In these circumstances, thickening of the aortic wall (normal 2 to 3 mm.) may be the major aortographic finding of aortic dissection. When the thickening of the aortic wall includes a long segment of the aorta, particularly the descending aorta, the diagnosis of aortic dissection can be made with reasonable certainty.[6] However, it should be emphasized that this finding is sometimes subject to error. Aortic wall thickness in the ascending aorta is unreliable as this can be produced by normal mediastinal structure or accumulation of fat.[10]

An ulcer-like projection of the contrast material from an otherwise smooth aortic lumen has been described. Further description of this helpful finding will be made in the Section of Branch Involvement.

AORTIC REGURGITATION

Although the presence of aortic regurgitation is a nonspecific finding, this is an important angiographic observation in association with aortic dissection, both from the prognostic and surgical standpoints. It should be remembered that injection into the false lumen may fail to demonstrate aortic regurgitation. Failure to visualize the typical configuration of the aortic sinuses and coronary arteries when injection is made into the ascending aorta indicates that the catheter is located in the false lumen (Fig. 5*A*).

COURSE OF DISSECTION

The dissections are most often located on the anterolateral aspect on the right side in the ascending aorta, superiorly in the aortic arch and on the posterolateral aspect on the left side in the descending thoracic aorta.[2] The fact that dissection is often located posteriorly and on the left side in the descending and abdominal aortas, thus resulting in higher incidence of involvement of the left renal and iliac arteries, has been emphasized by many authors, notably Siegelman *et al.*[12] A schematic description is given in Figure 6*A*. However, it is also true that in the descending thoracic and abdominal aortas, dissection often spirals posteriorly and appears on the right side (Fig. 6*B*; and 4, *A* and *B*).

Dissection may involve the whole circumference of the aorta. An excellent example of this is shown in Figure 5, *A–C*.

BRANCH INVOLVEMENT

Recognition of branch involvement facilitates clear understanding of the anatomy. Figure 7 is a diagram of various types of branch involvement. In Figure 7*B*, dissection extends into a branch artery. The true lumen of the branch is compressed by dissecting hematoma or false lumen. The true lumen can easily be totally occluded if the false lumen becomes enlarged. Ex-

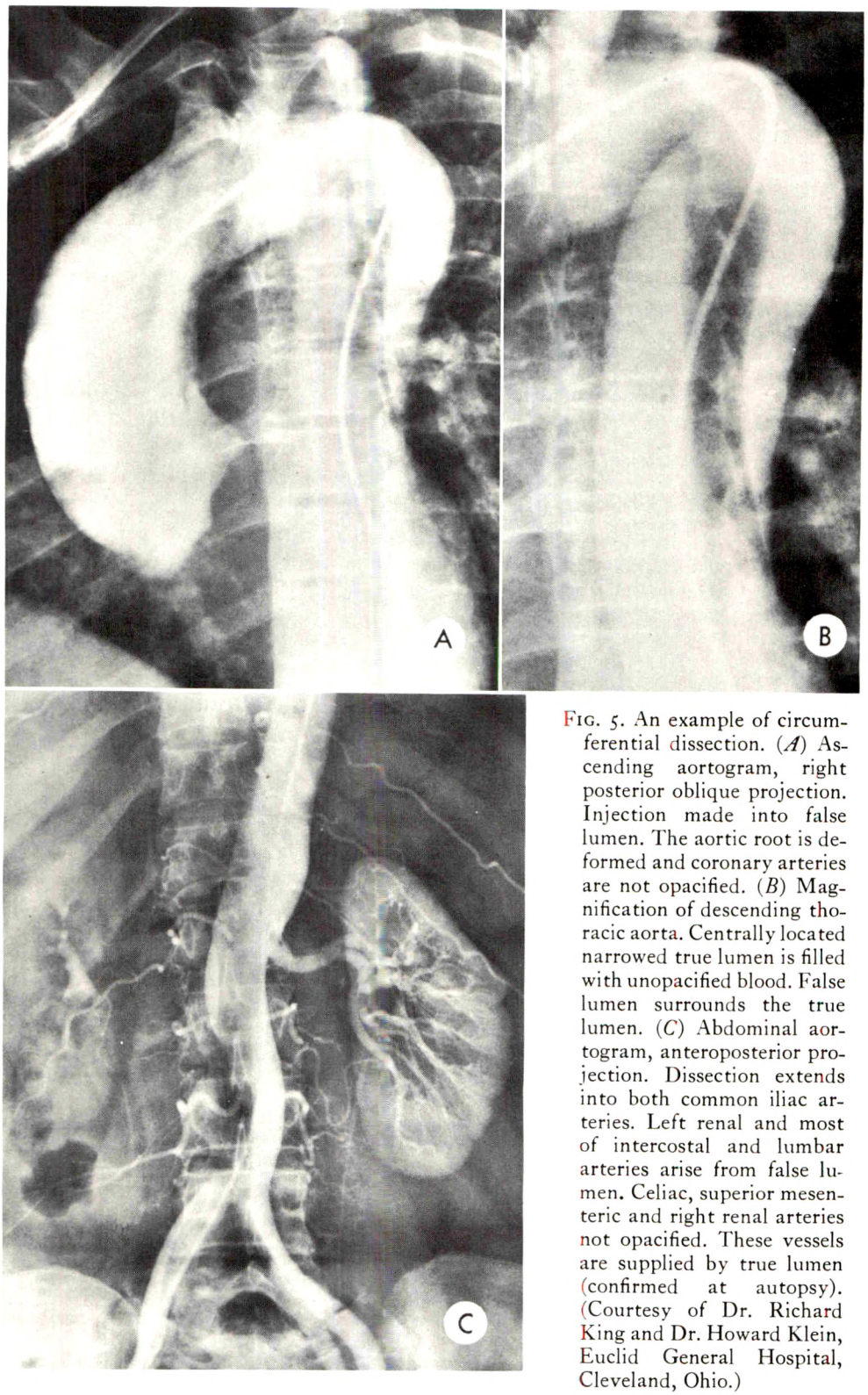


Fig. 5. An example of circumferential dissection. (*A*) Ascending aortogram, right posterior oblique projection. Injection made into false lumen. The aortic root is deformed and coronary arteries are not opacified. (*B*) Magnification of descending thoracic aorta. Centrally located narrowed true lumen is filled with unopacified blood. False lumen surrounds the true lumen. (*C*) Abdominal aortogram, anteroposterior projection. Dissection extends into both common iliac arteries. Left renal and most of intercostal and lumbar arteries arise from false lumen. Celiac, superior mesenteric and right renal arteries not opacified. These vessels are supplied by true lumen (confirmed at autopsy). (Courtesy of Dr. Richard King and Dr. Howard Klein, Euclid General Hospital, Cleveland, Ohio.)

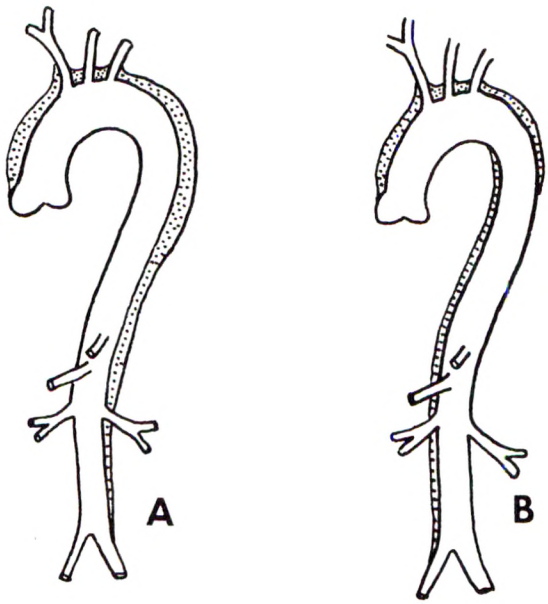


FIG. 6. Common routes of dissection. (*A*) Dissection is commonly located anteriorly on the right side in the ascending aorta, superiorly in the arch, and posteriorly on the left in the descending and abdominal aorta. (*B*) Dissection not infrequently spirals posteriorly in the region of thoracic or abdominal aorta and appears on the right side.

tension of dissection into a branch artery may be associated with distal re-entry (Fig. 7*C*). The branch artery may receive blood supply from both true and false channels. In Figure 7*D*, the intima is incompletely detached at the orifice of a branch artery. Depending upon the degree of the detachment, the branch artery will receive various degrees of blood supply from the false lumen as well as from the true lumen (Fig. 8, *A* and *B*).

Complete detachment of the intima at the orifice of a branch may occur (Fig. 7*E*). A hole may be left in the separated intima, corresponding to the detached orifice of the branch artery. Communication between the true and false channels can occur through the hole or holes (Fig. 9, *A–C*). A similar, although not identical situation is given in Figure 10, showing ulcer-like outpouchings coinciding the orifices of the intercostal arteries. "Ulcer-like projections" from the opacified aortic lumen to the thrombosed false lumen, described by Eyler and Clark[8] and other

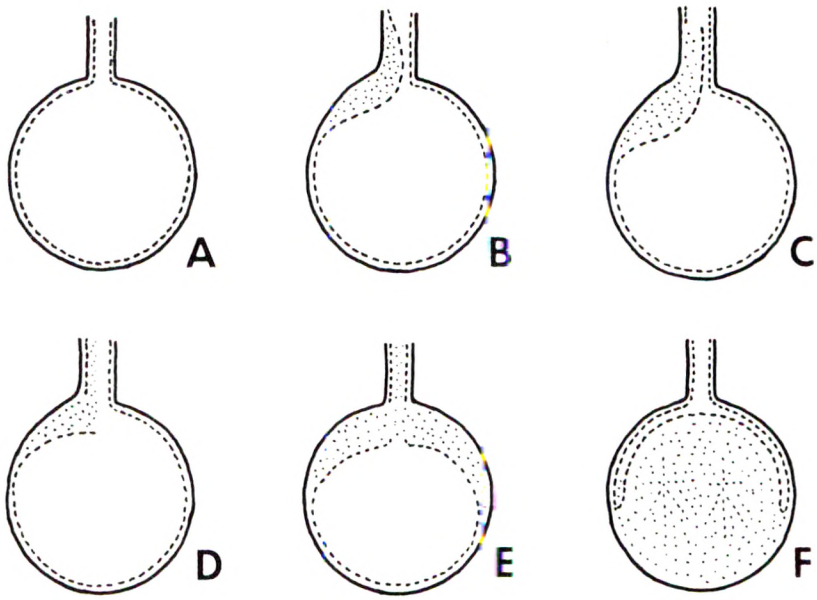


FIG. 7. Diagrams of various types of branch involvement. Aorta viewed in cross section at the orifice of an aortic branch. (*A*) Normal. (*B*) Dissection extends into a branch. True lumen may be compressed or occluded by false lumen. (*C*) Extension of dissection into a branch with distal re-entry. (*D*) Intimal tear at the orifice of a branch. (*E*) Complete detachment of the intima at the orifice of a branch. The branch artery receives blood exclusively from false lumen, unless the intima seals off and occludes the branch. (*F*) No direct involvement of a branch, but the false lumen on the opposite side is approximating the orifice of a branch.

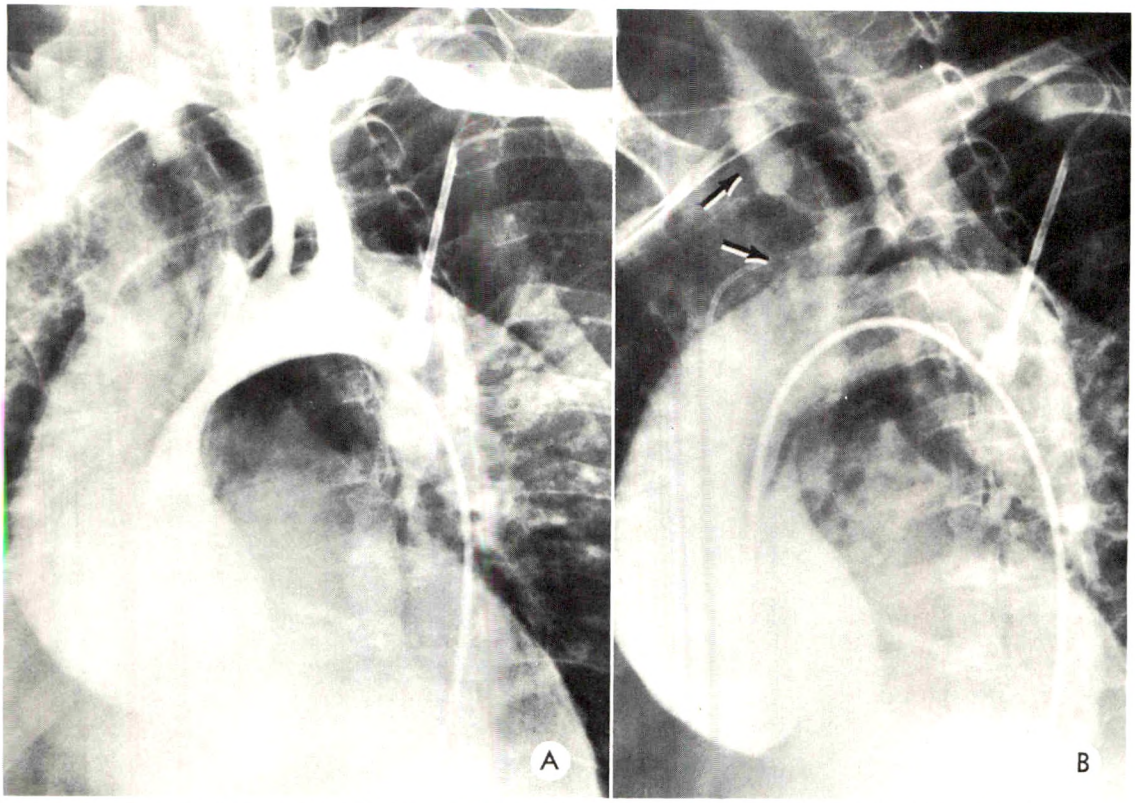


Fig. 8. Partial detachment of the intima at the orifice of an aortic branch (Fig. 7*D*). Thoracic aortogram, right posterior oblique projection. (*A*) Injection into true lumen which is markedly compressed. Left common carotid and subclavian arteries are well opacified from the true lumen. Little flow to the innominate artery. (*B*) Injection into false channel. Good opacification of the innominate artery (arrow), while left common carotid and subclavian arteries are not opacified.

authors,[5,13] may sometimes correspond to the detached or occluded orifices of small branches, in addition to the major intimal tear.

With complete detachment of the intima, the branch artery will receive blood supply exclusively from the false lumen, although the detached intima at the orifice of the branch or developed thrombus may seal off and occlude the vessels. Even small branches such as intercostal and lumbar arteries can arise from the false lumen[14] as in Figure 5*C*. Should the injection of contrast medium be made into the true lumen, the branch will not be visualized.

In Figure 7*F*, there is no direct involvement of a branch artery by the dissection, but the dissection on the opposite side is approximating the orifice of the branch. This type of indirect branch involvement may occur in the abdominal aorta with respect to the relationship between anteriorly located celiac and superior mesenteric arteries and posteriorly located false lumen (Fig. 11, *A* and *B*). Three dimensional depiction is given in Figure 12. This case emphasizes the value of the lateral projection in the abdominal aortic region for clear anatomical assessment and also illustrates the communication between the true lumen and false lumen through the detached orifices of the renal arteries.

Differentiation between the true lumen and false lumen

Table 11 summarizes several points of differentiation between the true lumen and false lumen. These are helpful, but are not universally applicable. As has been pointed out, the intercostal and lumbar arteries

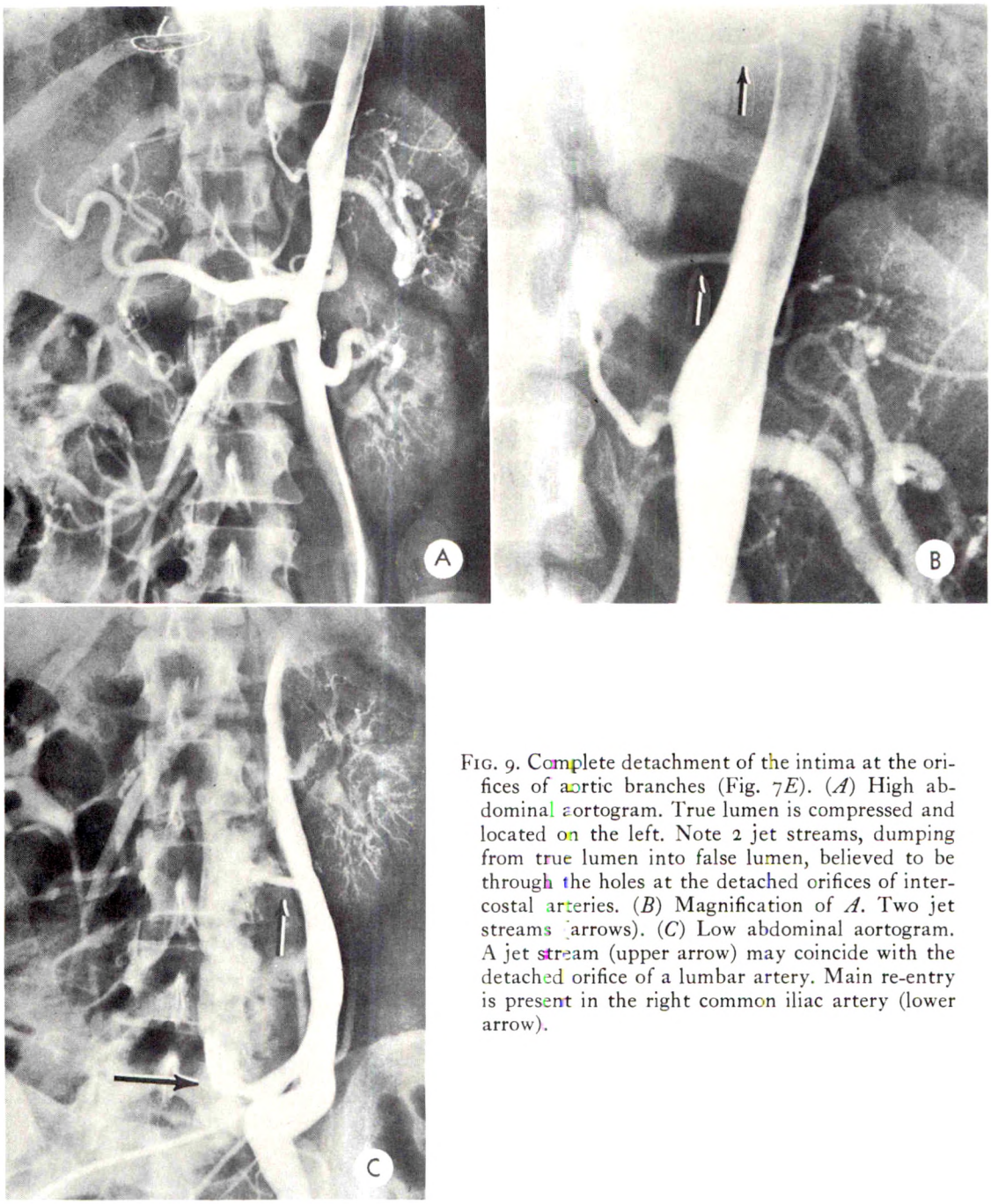


FIG. 9. Complete detachment of the intima at the orifices of aortic branches (Fig. 7*E*). (*A*) High abdominal aortogram. True lumen is compressed and located on the left. Note 2 jet streams, dumping from true lumen into false lumen, believed to be through the holes at the detached orifices of intercostal arteries. (*B*) Magnification of *A*. Two jet streams (arrows). (*C*) Low abdominal aortogram. A jet stream (upper arrow) may coincide with the detached orifice of a lumbar artery. Main re-entry is present in the right common iliac artery (lower arrow).

»»→

FIG. 11. Thin, anteriorly located true lumen compressed by posteriorly located false lumen (Fig. 7*E*). (*A*) Abdominal aortogram, lateral projection. Catheter is in the thin anteriorly located true lumen. False lumen is beginning to be opacified through the communication indicated by arrow. (*B*) Later phase. Celiac and superior mesenteric arteries arise from true lumen. At least one renal artery (arrow) arises from false lumen.

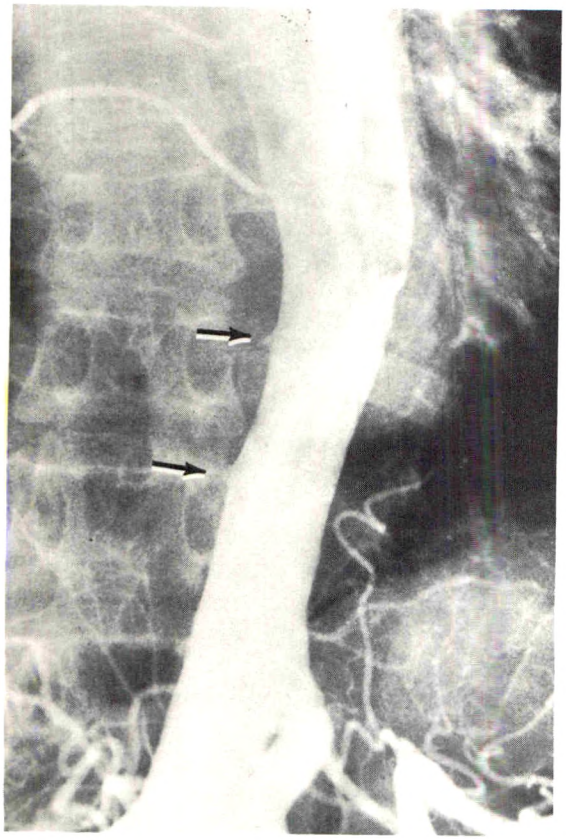


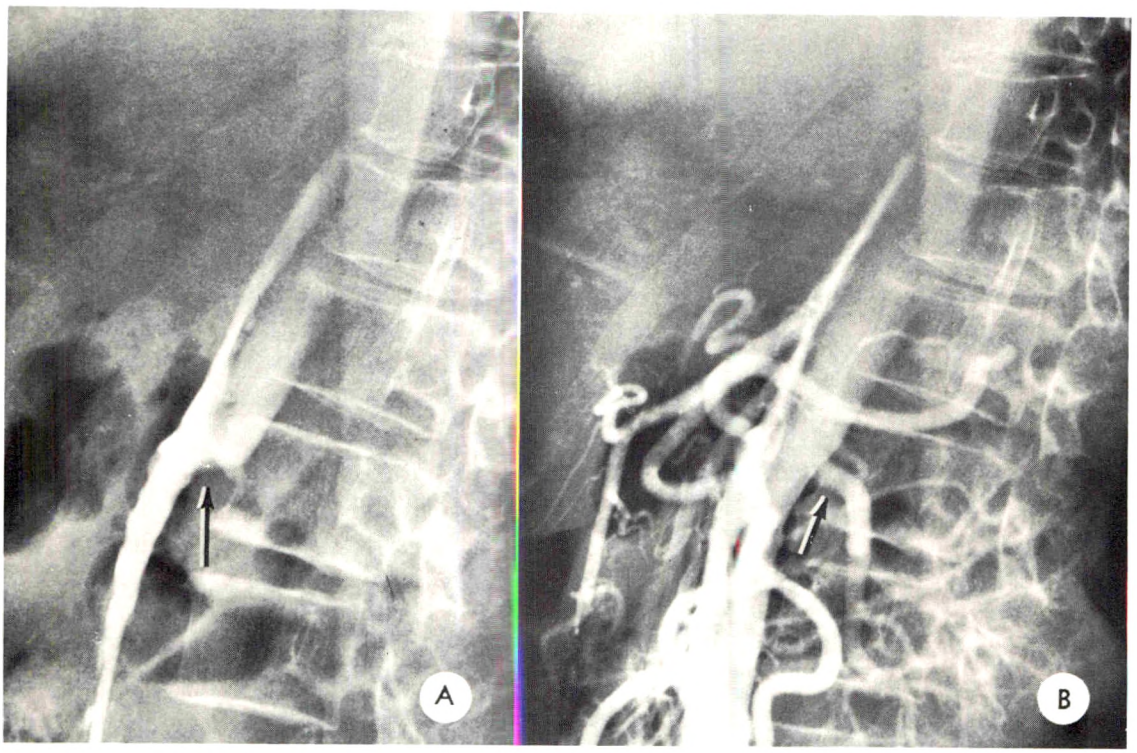
Fig. 10. Ulcer-like projections. Magnification of Figure 3D. The dissection is located mainly on the left side and posteriorly but appears on the right side also. Note 2 projections (arrows), exactly coinciding with the orifices of intercostal arteries. Ulcer-like projections described may sometimes coincide with detached or occluded orifices of aortic branches in addition to main intimal tear.

may arise exclusively from the false lumen. Blood supply of the coronary artery from the false lumen probably never occurs, although it is theoretically possible.

Sometimes, the aortograms are so confusing, especially in the abdominal aortic region, that the differentiation is very difficult or almost impossible. A lateral projection and three dimensional analysis, as is shown in Figure 12, may be helpful in distinguishing the true lumen from false lumen.

Pitfalls and Limitations in Aortographic Diagnosis of Aortic Dissection

Table III summarizes various pitfalls in aortographic diagnosis of aortic dissection.



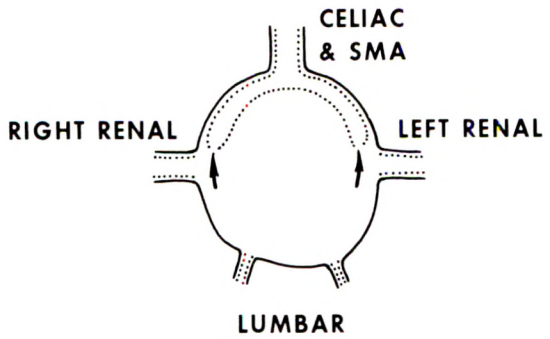


FIG. 12. Schematic explanation of Figure 11. True lumen supplies celiac and superior mesenteric arteries. False lumen supplies renal arteries. Communication between true lumen and false lumen may be through the detached intima at the orifices of renal arteries (arrows).

Simultaneous and equal opacification of both true and false channels has been emphasized.[2,11] Inappropriate projection is another cause of false negative aortograms. As has been previously described, aortic dissection may be totally unrecognized in certain projections (Fig. 13*B*). The illustrated case emphasizes the importance of visualizing not only the thoracic aorta but also the abdominal aorta and sometimes even the iliac arteries.

Acute external rupture may cause false negative aortograms, since the false lumen may not be filled with contrast material. In one of our patients, who had an acute external rupture into the pleural space, aortography failed to demonstrate the dissection. At autopsy, a Type III dissection was demonstrated. It is speculated that the false lumen was not filled with contrast material because of external bleeding, thus causing a false negative aortogram.

TABLE III

PITFALLS IN AORTOGRAPHIC DIAGNOSIS OF AORTIC DISSECTION

I. False Negative
   1. Simultaneous and equal opacification of true and false channels
   2. Inappropriate projection
   3. Acute external rupture
II. False Positive
   1. Thickening of aortic wall (due to clotted aneurysm, mediastinal hematoma, atherosclerosis, neoplasm, accumulation of fat, aortitis)
   2. Layering of contrast material (due to aortic insufficiency or Valsalva effect)

Thickening of the aortic wall is a helpful, but less reliable sign. An example of a false positive finding due to thrombosed aortic aneurysm is shown in Figure 14. The fact that the opacified aortic lumen is not compressed but rather dilated is the point to differentiate aneurysm from aortic dissection. When the opacified aortic lumen is not dilated or compressed, the differentiation between aortic dissection and aortic aneurysm with thrombosis becomes more difficult. While in acute dissection, the true lumen is usually compressed by the false lumen, in chronic dissection with thrombosis of the false lumen, the true lumen more or less regains its original diameter.

Other causes of thickening of the aortic wall such as neoplasm along the aortic wall, mediastinal hematoma, atherosclero-

TABLE II

DIFFERENTIATION BETWEEN TRUE LUMEN AND FALSE LUMEN

	True Lumen	False Lumen
Location		Anterolateral in ascending aorta Superior in aortic arch Posterolateral in descending and abdominal aortas
Pressure	Systemic	Low
Flow	Rapid	Slow
Size of lumen	Often compressed	Often wide
Blood supply	Coronary, intercostal, lumbar	Usually does not supply coronary, intercostal, or lumbar

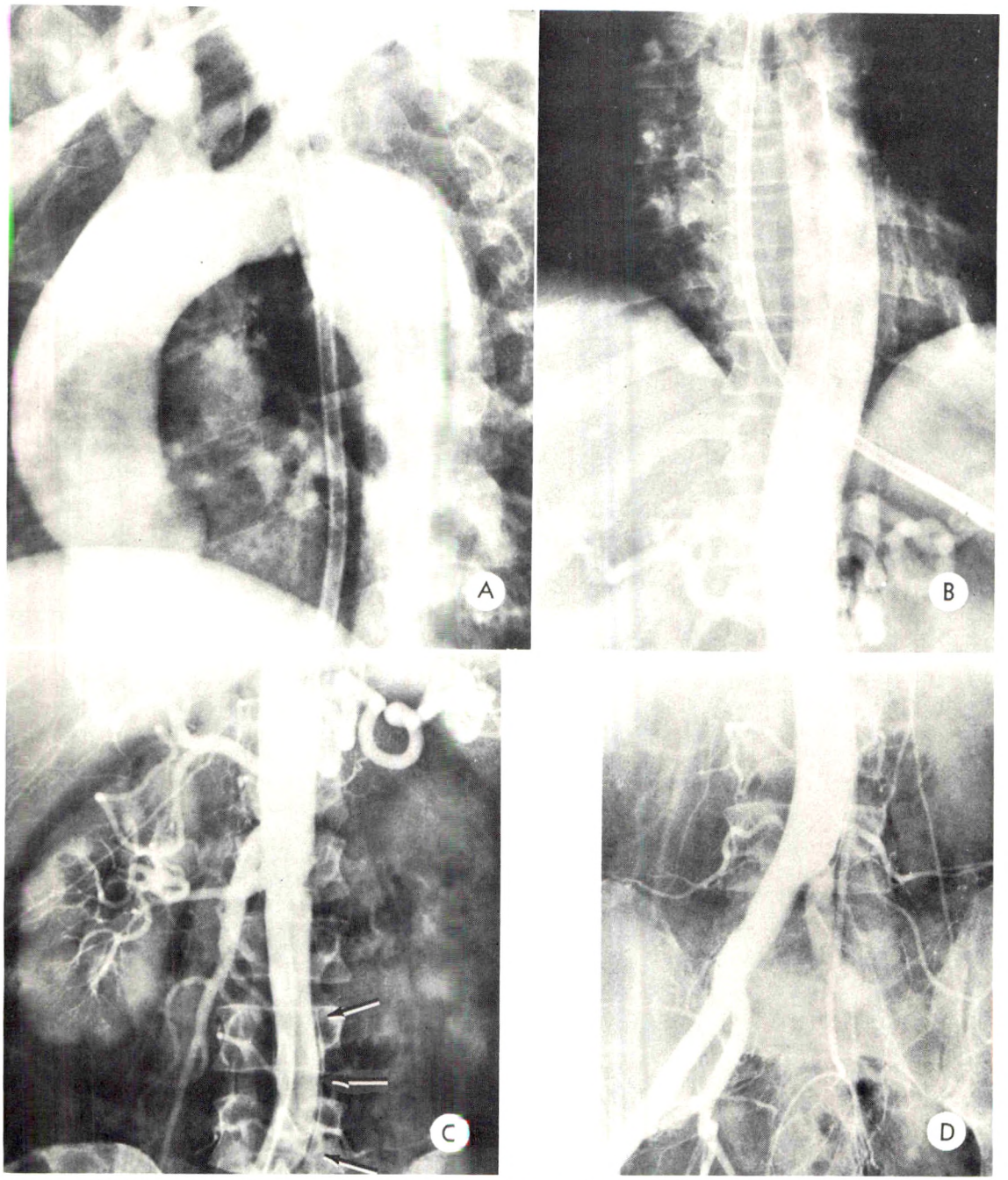


Fig. 13. The importance of proper projection and complete study by additional injection into the abdominal aorta. (*A*) Thoracic aortogram, right posterior oblique projection, showing Type III dissection. (*B*) Descending thoracic aortogram, anteroposterior projection. Pathologic condition is completely hidden. Celiac trunk and its branches well opacified. It is important not to stop the examination at this point. (*C* and *D*) Abdominal aortograms. Complete occlusion of left renal artery. Thin dissection extends to the lower abdominal aorta on the left side (arrows) involving the left common iliac artery.

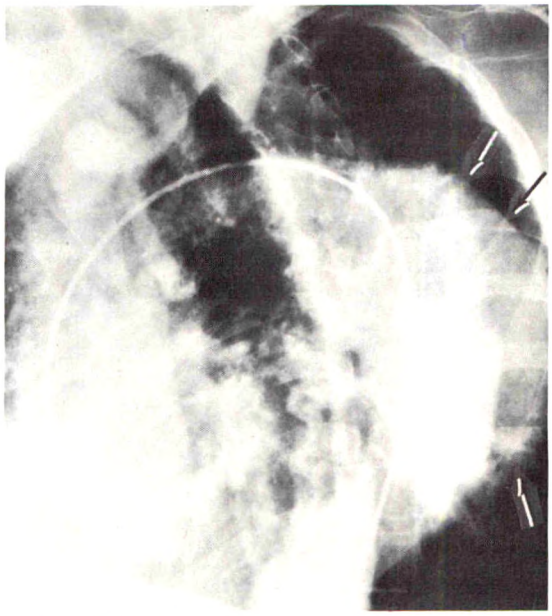


FIG. 14. Thoracic aortic aneurysm with thrombosis proven at surgery, simulating aortic dissection. Thickening of aortic wall (arrows) is due to thrombosis.

sis, accumulation of fat and aortitis must be carefully excluded, although they seldom pose a serious problem in the interpretation of aortograms of good quality.

Layering of contrast material due to aortic insufficiency[11] or Valsalva maneuver[1] has been reported as a cause of false positive aortographic findings.

In some critically ill patients, a precise and complete aortographic study in the chest and abdomen with multiple projections may not be feasible, and the diagnosis may be erroneous.

SUMMARY

The protean features of aortograms in aortic dissection are discussed in detail.

The three dimensional approach to a precise anatomic assessment is emphasized.

Problems in angiographic diagnosis of aortic dissection are also included.

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REFERENCES

1. BARCN, M. G. Dissecting aneurysm of aorta. *Circulation*, 1971, *43*, 933–943.
2. CRAMER, G. G., and AMPLATZ, K. Catheter position: aid in diagnosis of dissecting aneurysm of thoracic aorta. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 836–839.
3. DAILY, P. O., TRUEBLOOD, H. W., STINSON, E. B., WUERFLEIN, R. D., and SHUMWAY, T. E. Management of acute aortic dissections. *Ann. Thoracic Surg.*, 1970, *10*, 237–247.
4. DEBAKEY, M. E., HENLY, W. S., COOLEY, D. A., MORRIS, G. C., JR., CRAWFORD, E. S., and BEALL, A. C., JR. Surgical management of dissecting aneurysms of aorta. *J. Thoracic & Cardiovasc. Surg.*, 1965, *49*, 130–149.
5. DIMSMORE, R. E., ROURKE, J. A., DESANCTIS, R. D., HARTHORNE, J. W., and AUSTEN, W. G. Angiographic findings in dissecting aortic aneurysm. *New England J. Med.*, 1966, *275*, 1152–1175.
6. DIMSMORE, R. E., WILLERSON, J. T., and BUCKLEY, M. J. Dissecting aneurysm of aorta: aortographic features affecting prognosis. *Radiology*, 1972, *105*, 567–572.
7. EISEN, S., and ELLIOTT, L. P. Roentgenology of cystic medial necrosis of ascending aorta. *Radiol. Clin. North America*, 1968, *6*, 437–449.
8. EYLER, W. R., and CLARK, M. D. Dissecting aneurysm of aorta: roentgen manifestations including comparison with other types of aneurysms. *Radiology*, 1965, *85*, 1047–1057.
9. HIRST, A. E., JR., JOHNS, V. J., JR., and KIME, S. W., JR. Dissecting aneurysm of aorta: review of 505 cases. *Medicine*, 1958, *37*, 217–279.
10. PEICE, J. E., JR., GRAY, R. K., and GROLLMAN, J. H., JR. Aortic wall thickness as unreliable sign in diagnosis of dissecting aneurysm of thoracic aorta. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 710–712.
11. SHUFORD, W. H., SYBERS, R. G., and WEENS, H. S. Problems in aortographic diagnosis of dissecting aneurysm of aorta. *New England J. Med.*, 1969, *280*, 225–231.
12. SIEGELMAN, S. S., SPRAYREGEN, S., STRASBERG, Z., ATTAI, L. A., and ROBINSON, G. Aortic dissection and left renal artery. *Radiology*, 1970, *95*, 73–78.
13. STEIN, H. L., and STEINBERG, I. Selective aortography: definitive technique for diagnosis of dissecting aneurysm of aorta. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 333–348.
14. SYME, J. Branch involvement at aortography in dissecting aneurysm: with particular reference to lower intercostal and renal arteries. *Australian Radiol.*, 1972, *16*, 140–147.

Vol. 122, No. 4

CALCIFIED NONSYPHILITIC ANEURYSMS OF THE SINUSES OF VALSALVA

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SINUS of Valsalva aneurysms are an uncommon entity with less than 300 cases reported in the literature prior to 1973.[5,11] These aneurysms may be discrete aneurysms of a single sinus which are usually congenital, or may be due to an aneurysm of the aortic root which extends through the aortic annulus and causes generalized dilatation of all 3 sinuses of Valsalva,[11] which is usually acquired.

Calcified sinus of Valsalva aneurysms are even more unusual.[3] In the past these have usually been associated with syphilis. We report here 2 cases of calcified sinus of Valsalva aneurysms, unassociated with syphilis.

REPORT OF CASES

CASE I. J.S. (#9511), a 53 year old white man, was diagnosed as having subacute bacterial endocarditis in 1944, and was told he had the murmurs of aortic valvular insufficiency and stenosis. He received penicillin therapy for $3\frac{1}{2}$ months.

During the 1950s and 1960s he was hospitalized multiple times for ethanol abuse, but had no cardiac symptomatology. He denied ever having had syphilis or a penile lesion. A VDRL in November 1950 was nonreactive. In 1955 curvilinear calcifications were first noted on a chest roentgenogram. The VDRL was still nonreactive in June 1955.

Because of his murmur and progressive calcification of the aneurysms of the sinuses of Valsalva (Fig. 1, *A–C*) he was treated with penicillin and streptomycin in July 1972, although his cardiac symptoms were unchanged clinically. Physical examination revealed a blood pressure of 120/70 mm. Hg and murmurs of aortic valvular stenosis and insufficiency. The VDRL and the Fluorescent Treponemal Antibody-Absorption test were nonreactive. Roentgenograms of the sacroiliac joints were normal. Cardiac catheterization demonstrated moderate aortic valvular insufficiency and the discrete aneurysms of the sinuses of Valsalva (Fig. 2, *A* and *B*). After a 2 year follow-up, the patient remains asymptomatic.

CASE II. H.J. (#4972), a 53 year old white man, had 3 episodes of acute rheumatic fever in his early thirties and was told he had the murmur of aortic valvular insufficiency. He denied ever having syphilis or a penile lesion. He did well until age 43 at which time he had progressive dyspnea on exertion which was relieved by digitalization.

During September 1972 he was admitted for evaluation of chest pain. Physical examination revealed a wide pulse pressure and the peripheral signs of aortic insufficiency. He had the murmurs of aortic valvular stenosis and aortic valvular insufficiency. There was also an Austin-Flint murmur and a S3 gallop. The neurologic examination was normal.

A routine roentgenogram of the chest during September 1972, demonstrated a large calcified ascending aortic aneurysm (Fig. 3, *A–C*) contiguous with calcified aneurysmal dilatation of the sinuses of Valsalva. There was moderate cardiomegaly with biventricular enlargement. An electrocardiogram showed left axis deviation, left ventricular hypertrophy with a strain pattern, and left bundle branch block. Cholesterol, triglycerides, latex fixation and antinuclear antibody tests were normal. The VDRL and Fluorescent Treponemal Antibody-Absorption tests were nonreactive. Roentgenograms of the sacroiliac joints were normal. Right and left heart catheterization demonstrated normal pressures in all chambers, and normal cardiac index and arteriovenous oxygen difference. There was moderate aortic regurgitation and a markedly dilated aortic root with aneurysmal dilatation of the sinuses of Valsalva and ascending aorta extending to and involving the arch vessels.

Surgery was not attempted. Recent roent-

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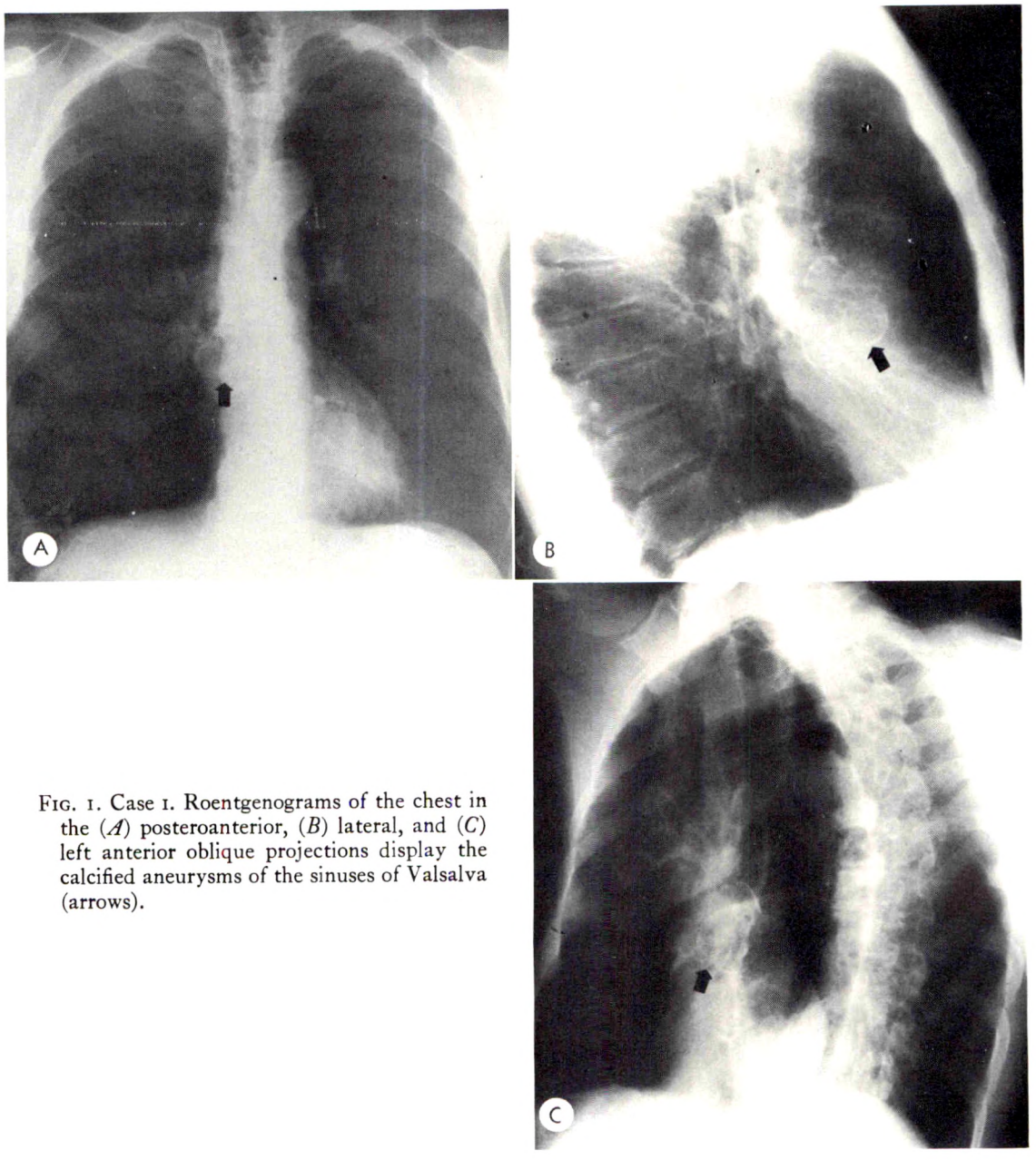


FIG. 1. Case 1. Roentgenograms of the chest in the (*A*) posteroanterior, (*B*) lateral, and (*C*) left anterior oblique projections display the calcified aneurysms of the sinuses of Valsalva (arrows).

genograms of the chest have shown no change in the size of the ascending aortic aneurysm.

DISCUSSION

Aneurysms of the sinus of Valsalva may be congenital or acquired. Several excellent reviews have thoroughly dealt with the congenital sinus of Valsalva aneurysms and their relationships to ventricular septal defects and acquired aortic valvular insufficiency.[13,14,22] Congenital aneurysms of the sinus of Valsalva are usually discrete; *i.e.*, involve a single sinus. They follow a definite pattern in site of origin and direction of development;[14] over 95 per cent of aneurysms originate in the right coronary sinus and the right part of the noncoronary sinus, and project into the right ventricle or right atrium. Edwards *et al.*,[2] have proposed that the fundamental

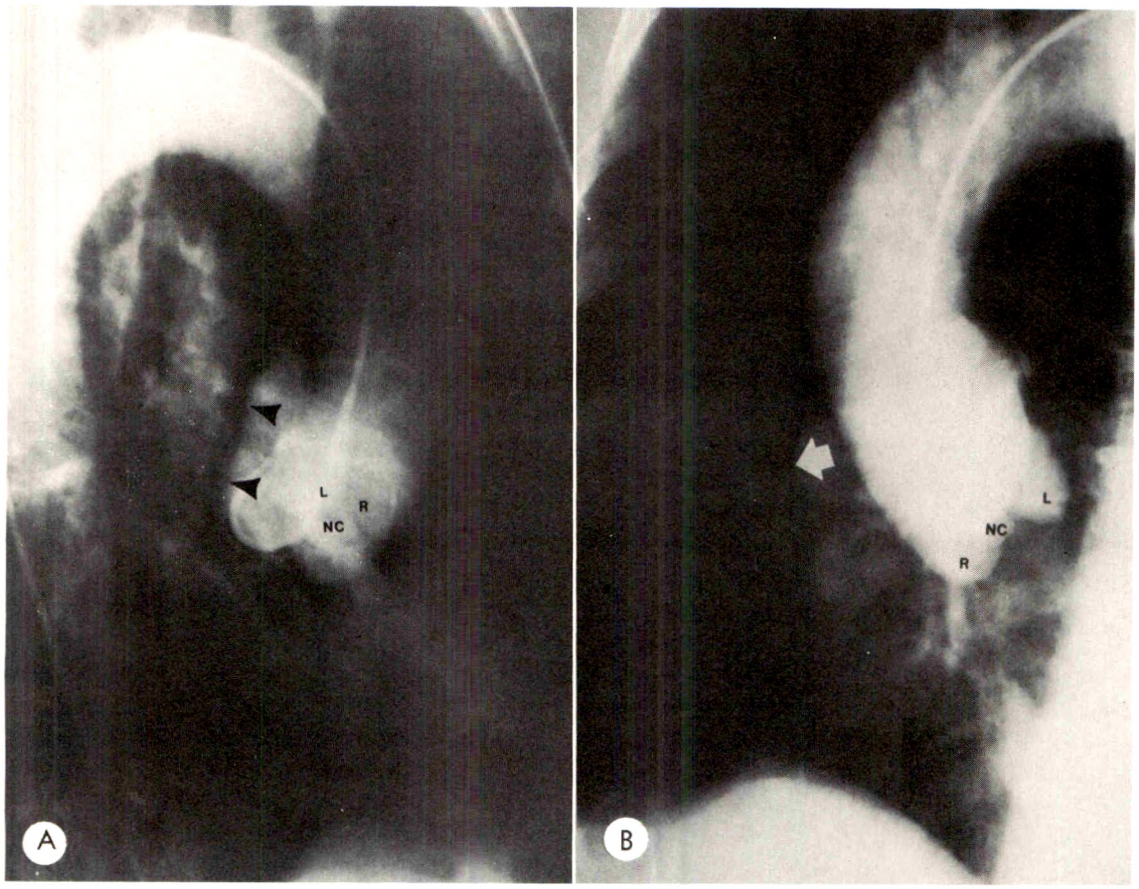


Fig. 2. Case 1. Retrograde aortic root injections in (*A*) lateral and (*B*) left anterior oblique projections demonstrate the sinuses of Valsalva (right [R], left [L] and noncoronary [NC] cusps). There are 2 aneurysmal dilatations extending from them. One (arrowheads) is posterior-inferiorly located between the noncoronary cusp and the left coronary cusp, and is best seen on the lateral projection (*A*). The second, best seen on the left anterior oblique projection (*B*), is higher and is at the level of the junction of the aortic annulus with the right coronary cusp, and does not fill (arrow), probably because it is filled with thrombus.

cause of congenital aneurysms is due to a separation of the aortic media and the annulus of the aortic valve.

Acquired sinus of Valsalva aneurysms may be discrete[9] (localized) or generalized (involving all the sinuses), developing as a consequence of a localized inflammatory process or as a generalized lesion of the aortic root which extends through the aortic annulus and causes dilatation of all 3 sinuses of Valsalva.[11] Generalized dilatation of the sinus of Valsalva is usually acquired secondary to bacterial endocarditis,[1] syphilis,[8,10,15] or atherosclerosis.[6,11] Marfan's syndrome is reponsible for those few generalized sinus of Valsalva aneurysms which arise on a congenital basis.[17,19,21]

The diagnosis of an aneurysm of the sinus of Valsalva on the plain roentgenogram is facilitated by the presence of calcification, which is a relatively rare finding. The majority of those reported in the past have occurred in syphilis.[3,15,16,18] The literature indicates that "when aortic calcification extends into the intracardiac origin of the aorta and outlines the aortic sinuses, the diagnosis of syphilitic aortic sinus aneurysm becomes almost a certainty."[8,12,18,20] Only 2 cases of calcified aneurysm of the sinuses unassociated with syphilis[12,23] have been previously described and both of these were congenital in origin. The current cases indicate that acquired aneurysms of the sinuses of Valsalva may

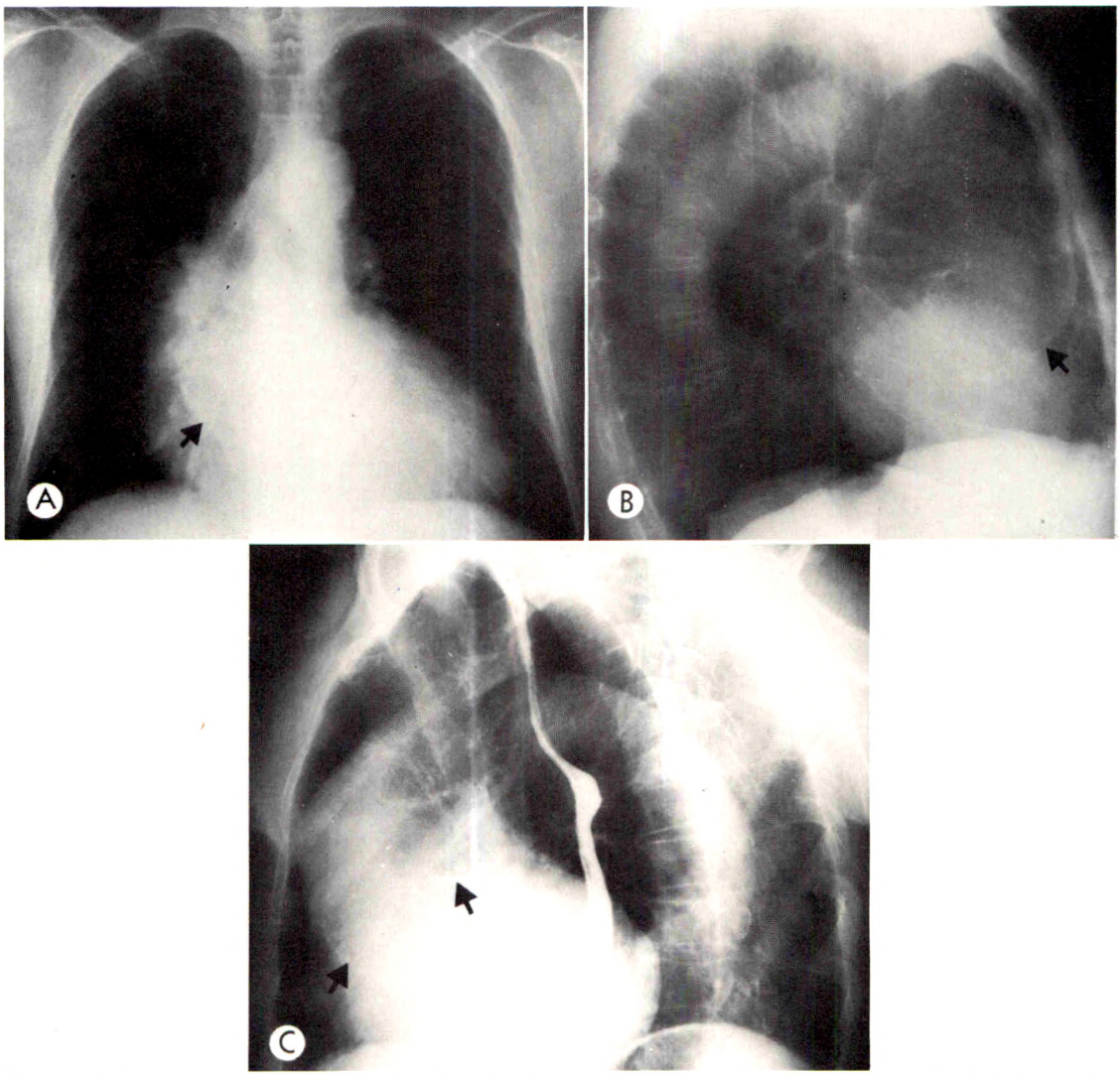


FIG. 3. Case II. Roentgenograms of the chest in the (*A*) posteroanterior, (*B*) lateral and (*C*) left anterior oblique projections demonstrate the large calcified aneurysm of the ascending aorta which extends to and involves the sinuses of Valsalva. Calcification in the dilated sinuses of Valsalva is indicated by arrows.

calcify in the absence of a syphilitic etiology.

The other plain film roentgenographic findings are relatively nonspecific as to etiology and include: (1) a prominent pulmonary arch segment due to enlargement of the sinus of Valsalva to the left;[7] (2) a prominent localized bulge of the lower right cardiac margin;[3] (3) a dilated ascending aorta;[8,17,20] and (4) presence of a "notch sign" between the ascending aorta and the heart noted on the posteroanterior or left anterior oblique views.[15]

Linear calcification of the aorta, which has long been recognized as an important sign of syphilitic aortitis,[16,24] recently has been found not to be a pathognomonic roentgenographic sign of syphilis as previously thought.[16,24] In this study the most common cause of ascending aortic calcification was atherosclerosis.[4] These 2 cases show that nonsyphilitic inflammatory disease may cause calcification of aneurysms of the sinus of Valsalva. With the decrease in cardiovascular syphilis, and the attendant increase in longevity, other causes of

calcification may become relatively more common.

SUMMARY

Calcification of a sinus of Valsalva aneurysm has been considered specific for a syphilitic aortitis.

The current report contains 2 cases of calcified aneurysms of the sinuses of Valsalva due to nonsyphilitic inflammatory disease. These 2 cases along with 2 cases previously reported to be congenital in origin comprise a total of 4 reported cases with calcified aneurysms of the sinuses of Valsalva of nonsyphilitic etiology.

Thus, calcified sinus of Valsalva aneurysms are not specific for syphilitic aortitis, but may rarely be associated with any of the etiologic factors responsible for these aneurysms.

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REFERENCES

1. Bjork, V. O., and Bjork, L. Aneurysm of sinus of Valsalva. *J. Thoracic & Cardiovasc. Surg.*, 1965, *50*, 16–21.
2. Edwards, J. E., Burchell, H. B., and Christensen, N. A. Specimen exhibiting essential lesion in aneurysm of aortic sinus. *Proc. Staff Meet. Mayo Clin.*, 1956, *31*, 407–412.
3. Harris, E. J. Aneurysm of sinus of Valsalva. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1956, *76*, 767–772.
4. Higgins, C. H., and Reinke, R. T. Non-syphilitic etiology of linear calcification of ascending aorta. To be published.
5. Hope, J. A. Treatise on the Heart and Great Vessels. 1840, London.
6. Kawasaki, I. A., and Schultz, F. B. Aneurysm of sinus of Valsalva with rupture into right auricle. *Am. Heart J.*, 1951, *41*, 149–154.
7. McKusick, V. A. Cardiovascular aspects of Marfan's syndrome: heritable disorder of connective tissue. *Circulation*, 1955, *11*, 321–342.
8. Merten, C. W., Finby, N., and Steinberg, I. Ante mortem diagnosis of syphilitic aneurysm of aortic sinuses. *Am. J. Med.*, 1956, *20*, 345–360.
9. Oram, S., and East, T. Rupture of aneurysm of aortic sinus into right side of heart. *Brit. Heart J.*, 1955, *17*, 541–551.
10. Ostrum, H. W., Robinson, B. D., Nichols, C. F., and Widman, B. P. Aneurysm of aortic sinuses or sinus of Valsalva. Am. J. Roentgenol. & Rad. Therapy, 1938, *40*, 828–837.
11. Prian, G. W., and Dietrich, E. B. Sinus of Valsalva abnormalities. *Vasc. Surg.*, 1973, *7*, 155–164.
12. Rowley, J. C., and Holmes, R. B. Sinus of Valsalva aneurysms. *J. Canad. A. Radiologists*, 1965, *16*, 254–261.
13. Sakakibara, S., and Konno, S. Congenital aneurysm of sinus of Valsalva associated with ventricular septal defect. *Am. Heart J.*, 1968, *75*, 595–603.
14. Sakakibara, S., and Konno, S. Congenital aneurysm of sinus of Valsalva: anatomy and classification. *Am. Heart J.*, 1962, *63*, 405–424.
15. Schneider, H. J., and Spitz, H. B. Unruptured aortic sinus aneurysm: plain film diagnosis. *Dis. Chest*, 1968, *53*, 340–348.
16. Shipp, J. C., Crowley, L. V., and Wigh, R. Aortic sinus aneurysm: production of intracardiac calcification and pulmonary artery fistula. *Am. J. Med.*, 1955, *18*, 160–166.
17. Steinberg, I., and Geller, W. Aneurysmal dilatation of aortic sinuses in arachnoidactyly: diagnosis during life in 3 cases. *Ann. Int. Med.*, 1955, *43*, 120–132.
18. Steinberg, I., and Finby, N. Clinical manifestation of unperforated aortic sinus aneurysm. *Circulation*, 1956, *14*, 115–124.
19. Steinberg, I. Dilatation of aortic sinuses in Marfan's syndrome: roentgen findings in 5 new cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *83*, 302–319.
20. Steinberg, I., and Finby, N. Roentgen manifestations of unperforated aortic sinus aneurysms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1957, *77*, 263–273.
21. Symbas, P. N., Raizner, A. E., Tyras, D. H., Hatcher, C. R., Inglesby, T. V., and Baldwin, B. J. Aneurysms of all sinuses of Valsalva in patient with Marfan's syndrome. *Ann. Surg.*, 1971, *174*, 902–907.
22. Taguchi, K., Sasaki, N., Matsuura, Y., and Uemura, R. Surgical correction of aneurysm of sinus of Valsalva. *Am. J. Cardiol.*, 1969, *23*, 180–191.
23. Triano G. J. Calcified congenital left coronary sinus aneurysm. *Radiology*, 1968, *91*, 921–922.
24. Wolkin, A. Significance of calcification in ascending portion of aortic arch. *Radiology*, 1954, *62*, 101–105.

VARICOSE ANEURYSM OF THE THORACIC AORTA WITH AORTO-AZYGOS SYSTEM FISTULA*

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ARTERIOVENOUS fistulas may be congenital or acquired. Congenital arteriovenous fistulas are due to the lack of differentiation of the common primary embryologic anlage into arteries and veins.[6] Acquired arteriovenous fistulas are found most frequently following penetrating injuries from gunshot wounds and stabbings. They have also been reported following angiography, surgery,[10,12,13] automobile accidents[5] and spontaneous rupture of aneurysm.[1]

Although traumatic arteriovenous communications in the extremities are encountered with some frequency,[2,3,6,7,14] central fistulas involving the large vessels of the trunk are rare. Injuries involving trauma severe enough to puncture the aorta frequently preclude survival. Eighty per cent of patients with aortic rupture due to automobile accidents die from the initial trauma.[5] Previously described central arteriovenous fistulas have usually involved the abdominal aorta with communication to the inferior vena cava.[4] Survival of these individuals was the result of retroperitoneal hematoma formation with eventual tamponade of bleeding. Fistulous communication between the aorta and inferior vena cava in these cases may be direct, or *via* an intervening false aneurysm in which case the entity is referred to as a "varicose aneurysm."[14] In addition to ordinary trauma, aortocaval arteriovenous fistulas have been seen following intervertebral disk surgery[10,12] and after the spontaneous perforation of abdominal aortic aneurysm.[1]

A review of 181 arteriovenous fistulas seen at the Mayo Clinic[6] over a 10 year period shows 7 aorta-inferior vena caval fistulas and none involving the thoracic aorta. To our knowledge, a false aneurysm of the thoracic aorta with azygos system fistulization has not yet been previously reported.

REPORT OF A CASE

A 45 year old Puerto Rican man was admitted to the St. Luke's Hospital Center following multiple gunshot wounds. Blood pressure was 80/60 mm. Hg with a pulse rate of 124 per minute. Entry wounds were noted in the neck, both arms and right chest. There were decreased breath sounds at the right lung base. The remainder of the physical examination was within normal limits.

Chest roentgenograms showed a right hemothorax and a bullet in the T 10 vertebral body. Right thoracentesis revealed bright red blood. An intravenous aortography was carried out to determine the integrity of the great vessels (Fig. 1, *A* and *B*). During the arterial phase, (Fig. 1*A*) the descending thoracic aorta appeared intact. However, a roentgenogram obtained late in the series (Fig. 1*B*) showed a rounded collection of contrast material (arrows) overlying the 8th through 10th thoracic vertebrae. This density was not present on the preliminary roentgenogram and was interpreted as extravasated contrast material into the mediastinum.

Because of his stable clinical picture, immediate operative intervention was not carried out. A right tube thoracotomy was performed productive of minimal drainage. The patient improved and made an uneventful recovery with the exception of a residual functional deficit in the left hand due to brachial plexus injury from one of the bullet wounds.

Ten months following his initial discharge, the patient presented in the emergency room with left lower chest and back pain. Physical examination revealed a loud bruit audible over

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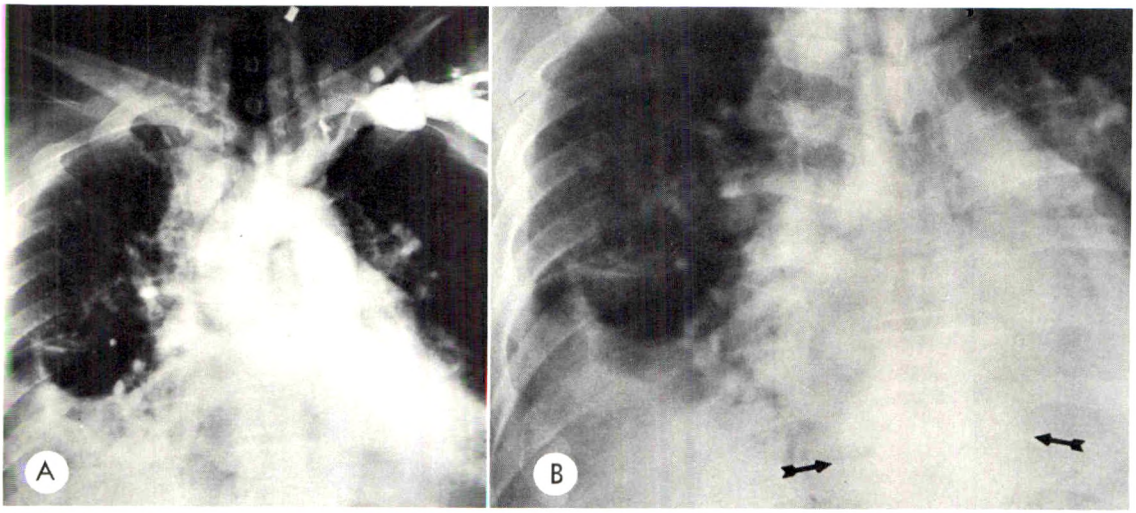


FIG. 1. (*A*) Intravenous angiocardiogram after bullet wound demonstrates the apparent integrity of the thoracic aorta during the arterial phase. Note the fluid in the right hemithorax and a bullet in the plane of the 10th thoracic vertebra. (*B*) A delayed roentgenogram shows a rounded collection of contrast material (arrows) overlying the eighth through tenth thoracic vertebrae representing extravasation into the mediastinum.

the entire back, but maximal over the left lower thorax. There was a left wrist drop. The remainder of the physical examination was unremarkable. The patient was admitted for further evaluation.

A retrograde thoracic aortography was performed *via* the femoral route. Fluoroscopically, there was extensive pulsation of the catheter tip as it reached the T 10 level within the aorta. Thoracic aortography (Fig. 2*A*) demonstrated a large collection of contrast material to the left of the aorta at the T 10 level during the arterial phase. A lateral aortogram (Fig. 2*B*) showed this collection to be located posteriorly. The appearance was compatible with a false aneurysm of the aorta. Additionally, filling of the azygos venous system was seen during the arterial phase with an apparent venous communication with the aortic false aneurysm at the T 10 level. Subtraction manipulation of the roentgenogram (Fig. 2*C*) showed the filled azygos system to better advantage in the frontal aortogram. A roentgenogram from the late arterial phase of the lateral aortogram (Fig. 2*D*) showed the filled azygos vein coursing anteriorly to empty into the superior vena cava.

Operative and Pathologic Findings. The patient was taken to the operating room and a left thoracotomy was performed. A 4 cm. false aneurysm of the thoracic aorta was found with a $\frac{3}{4}$ cm. diameter communication with the aorta. A large venous connection to the false aneurysm was identified, as well as multiple dilated venous channels in the region. Under partial bypass, after ligation of venous connections, the false aneurysm was excised along with a segment of the adjacent aorta and a tubular teflon graft placed end to end to the proximal and distal aortic stumps.

The pathologic specimen showed minimal atheromatous changes on the aortic side (Fig. 3*A*), as well as the communication (probe) into the false aneurysm. Figure 3*B* shows a similar view of the communication into the aorta from the opened aspect of the false aneurysm. The venous connections had been taken down at surgery.

DISCUSSION

In addition to being a very rare clinical entity, the present case is of interest in that it provides an opportunity, *via* sequential angiograms, to study the natural history of the formation of a false aneurysm. The angiocardiograms (Fig. 1, *A* and *B*) obtained immediately after trauma, demonstrate extravasation of contrast material into a mediastinal hematoma. Because surgical intervention was not carried out, this hematoma, adjacent to an apparent aortic tear, went on to false aneurysm for-

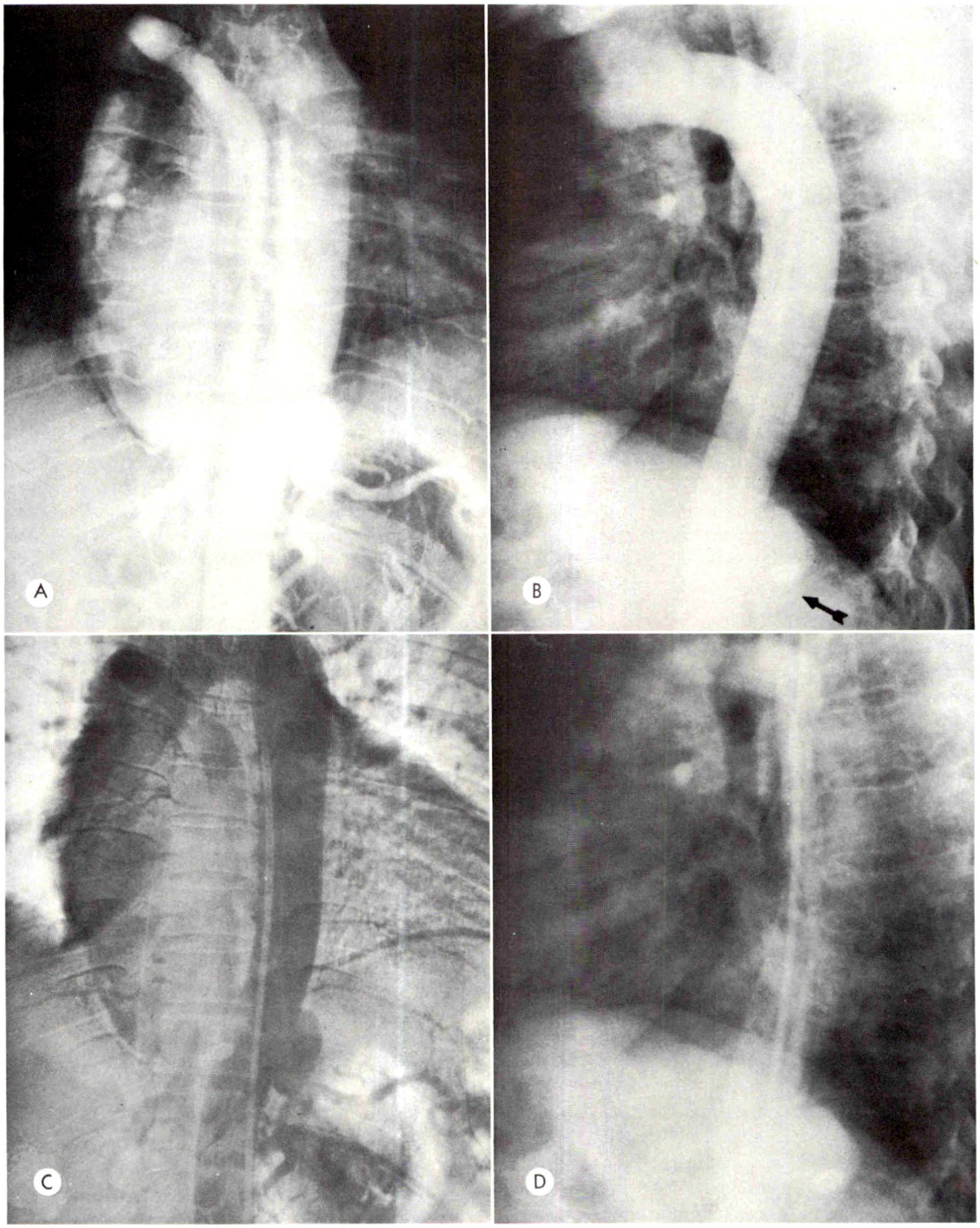


FIG. 2. (*A*) Retrograde thoracic aortogram 10 months after trauma shows a false aneurysm to the left of the aorta, as well as opacification of the azygos venous system and its tributaries during the arterial phase of the study. (*B*) Lateral aortogram demonstrates the false aneurysm to be located posteriorly (arrow). (*C*) Subtraction demonstrates the opacification of the azygos vein and its tributaries to better advantage. (*D*) Later roentgenogram in the lateral projection shows the azygos vein coursing anteriorly to empty into the superior vena cava, while the aorta still contains contrast material.

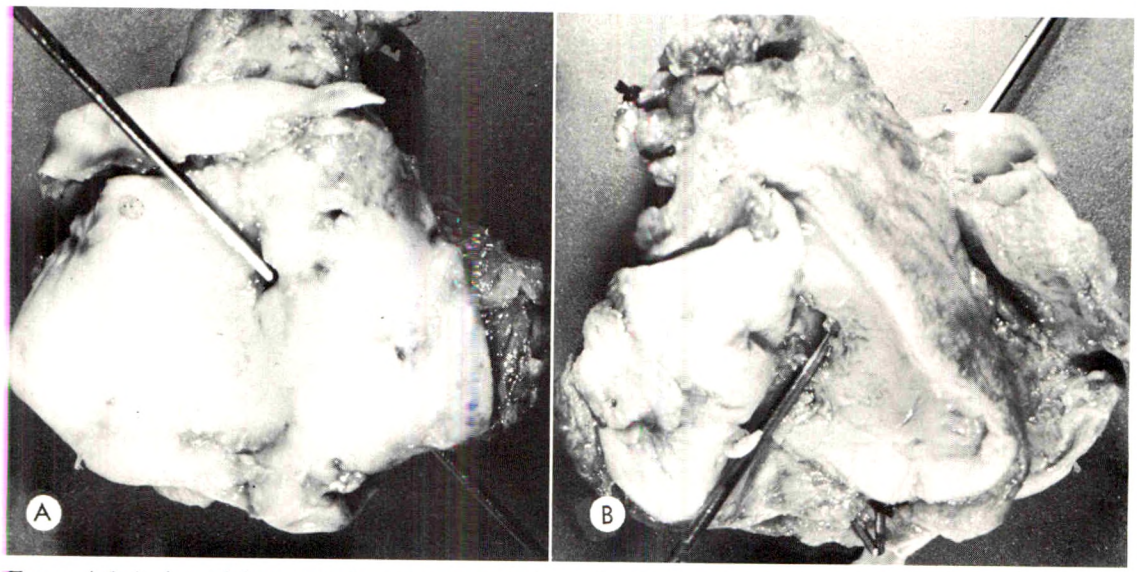


FIG. 3. (*A*) A view of the resected aortic segment shows the communication (probe) into the false aneurysm. Note an intercostal orifice above and to the left of the probe. (*B*) A photograph of the cut section opened surface of the false aneurysm shows its communication to the aorta (probe). The venous connections had been severed at surgery.

mation with venous communications demonstrated in the aortogram obtained after the patient developed left chest pain (Fig. 2, *A* and *B*).

It cannot be directly ascertained from the angiograms at what stage the venous communication occurred. It is most likely that the initial trauma caused venous as well as arterial damage and that the arterial leak, causing a pulsating hematoma, went on to endothelize as a false aneurysm joining the arterial and venous injuries. Although no early venous filling of the azygos system was demonstrated on the initial angiocardiogram, this may have been due to the small blood flow across the communication at that time. Alternatively, the false aneurysm could have spontaneously ruptured into the azygos system after a considerable period of time.

Calcification, commonly seen in varicose aneurysms,[2] was not seen in our case, clearly because the tremendous flow and physiologic disturbances caused by the lesion led to its discovery before it had been present for a year. It is interesting to note that despite the large flow across the central fistula, this patient never experienced symptoms of congestive failure and was not in failure at the time he was admitted because of chest pain.

SUMMARY

A traumatic false aneurysm of the thoracic aorta with fistulous communication to the azygos venous system developed within 1 year of a gunshot wound.

Immediately after injury, angiography had demonstrated a mediastinal hematoma without evidence of venous communication.

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REFERENCES

1. BEALL, A. C., JR., COOLEY, D. A., MORRIS, G. C., JR., and DEBAKEY, M. E. Perforation of arteriosclerotic aneurysms into inferior vena cava. *A.M.A. Arch. Surg.*, 1963, *86*, 809–818.
2. BELL, D., and COCKSHOTT, W. P. Angiography of traumatic arteriovenous fistulae. *Clin. Radiol.*, 1965, *16*, 241–247.
3. BRADHAM, R. R., MCFEE, A. S., and PALMER, J. R., JR. False aneurysm and arteriovenous

fistula. *J. South Carolina M.S.*, 1968, *64*, 313–318.

4. CONN, J. H., FAIN, W. R., and CHAVEZ, C. M. Traumatic aorto-caval fistula. *J. Thoracic & Cardiovasc. Surg.*, 1967, *54*, 880–885.
5. FREED, T. A., NEAL, M. P., JR., and VINIK, M. Roentgenographic findings in extracardiac injury secondary to blunt chest automobile trauma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *104*, 424–432.
6. GOMES, M. M. R., and BERNATZ, P. E. Arteriovenous fistulas: review and ten year experience at Mayo Clinic. *Mayo Clin. Proc.*, 1970, *45*, 81–102.
7. HOLMAN, E. Abnormal arteriovenous communications. *Circulation*, 1965, *32*, 1001–1009.
8. KNOX, W. G., MILLER, R. E., DWYER, E. M., and GUTHRIE, R. Abdominal aortic aneurysm: vena caval fistula. *Ann. Surg.*, 1966, *164*, 947–950.
9. LANG, E. K., TRICHEL, B. E., TURNER, R. W., FONTENOT, R. A., JOHNSON, B., and ST. MARTIN, E. C. Arteriographic assessment of injury resulting from renal trauma: analysis of 72 patients. *J. Urol.*, 1971, *106*, 1–8.
10. SALDINO, R. M., WHITE, A. A., and PALUBINSKAS, A. J. Arteriovenous fistula: complication of lumbar disc surgery. *Clin. Radiol.*, 1965, *16*, 248–250.
11. SIEGELMAN, S. S., ROSENBERG, R. F., and FURMAN, S. Angiographic evaluation of injury to great vessels. *A.M.A. Arch. Surg.*, 1970, *100*, 565–567.
12. STAPLE, T. W., and FRIEDENBERG, M. J. Ilioiliac arteriovenous fistula following intervertebral disc surgery. *Clin. Radiol.*, 1965, *16*, 248–250.
13. STEINBERG, I., and STEIN, H. False aneurysm of ascending aorta following insertion of sutureless aortic valve prosthesis. *South African M. J.*, 1970, *44*, 979–981.
14. TUCKER, W. G., and PERRETT, L. V. Traumatic aneurysms and traumatic arteriovenous fistulae. *Australasian Radiol.*, 1969, *13*, 80–87.

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THE IMPORTANCE OF CORONARY ANGIOGRAPHY IN THE EVALUATION OF PATIENTS WITH AORTIC VALVULAR DISEASE*

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THE condition of the coronary arteries in patients with aortic valvular disease must be known both for the medical management, and in planning aortic valve surgery where these arteries must be cannulated during cardiac bypass.

There have been several reports in the medical literature about the possible protective value of aortic stenosis (AS),[3,7] or aortic insufficiency (AI)[5] against coronary artery disease (CAD).

This study will demonstrate the results of our clinical experience, and in light of it, we will plea that all patients scheduled for aortic valve replacement undergo coronary angiography, as part of their preoperative evaluation.

MATERIAL AND METHOD

Forty-two consecutive patients with AS and/or AI were examined at The New York Hospital. In each case, left heart catheterization followed by coronary angiography and left ventriculography were performed using the Judkins transfemoral technique.[4]

CLASSIFICATION

The status of each patient's aortic valve was evaluated according to the following criteria:

Aortic Stenosis (AS)

1. Mild AS
   a. Aortic valve cross-sectional area larger than 1.5 cm.^{2}, but smaller than normal; and/or,
   b. Peak systolic aortic valve gradient smaller than 30 mm. Hg and normal myocardial contractility, as indicated by the left ventricular end diastolic pressure (LVEDP).*
2. Significant (moderate to severe) AS
   a. Aortic valve cross-sectional area smaller than 1.5 cm.^{2}; and/or,
   b. Peak systolic aortic valve gradient greater than 30 mm. Hg with normal or abnormal myocardial contractility; or,
   c. Peak systolic aortic valve gradient smaller than 30 mm. Hg *but* abnormal myocardial contractility, as indicated by the LVEDP† (there were no patients found in this category).

The role of myocardial contractility is important, since failing hearts will generate less gradient across a stenotic valve than normal hearts.

Aortic Insufficiency (AI)

1. Mild AI—small amount of regurgitation with complete clearing of contrast medium from the left ventricle during each systole.
2. Significant (moderate to severe) AI—large amount of regurgitation with incomplete clearing of contrast medium from the left ventricle during each systole.

* Normal myocardial contractility—LVEDP 12 mm. Hg or less.

† Abnormal myocardial contractility—LVEDP greater than 12 mm. Hg.

* Presented at the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, San Francisco, California, September 24–27, 1974.

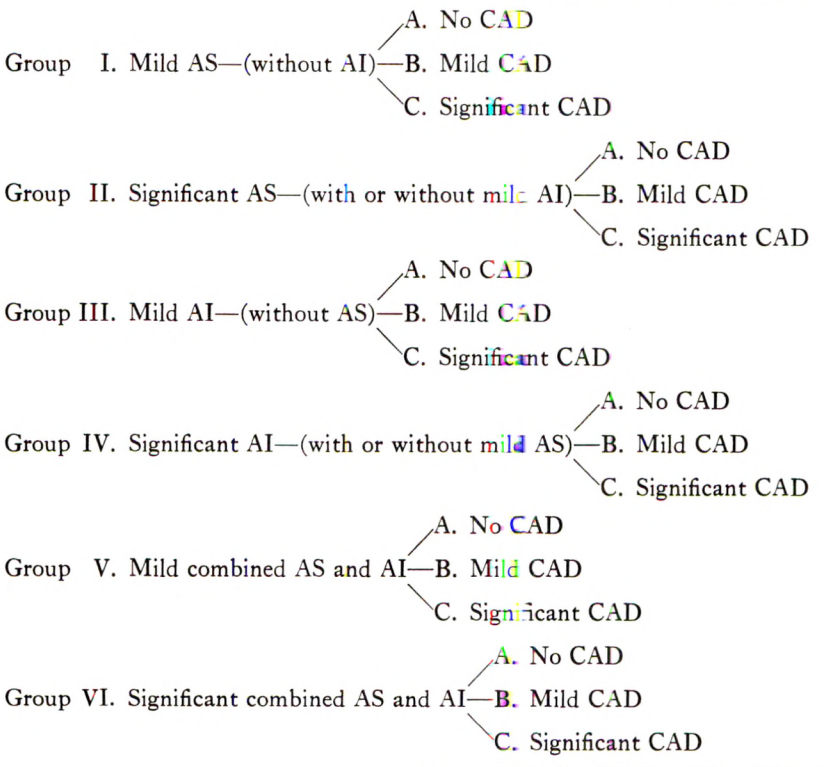
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Coronary Artery Disease (CAD). The degree of CAD was determined in each patient by coronary angiography.

A. No CAD
B. Mild CAD (stenoses less than 50 per cent)
C. Significant CAD (stenoses greater than 50 per cent or complete occlusion).

Patients were then assigned into 6 groups according to the severity and type of aortic valve disease (AVD) and each group was subdivided according to the extent of CAD as follows:



RESULTS

In our series of 42 patients studied for AVD, 17 (39.5 per cent) had significant CAD. Thirty-seven of the 42 (83.3 per cent) patients studied were shown to have hemodynamically significant AVD. Of these 37 patients, 13 (35.1 per cent) had significant CAD. Five patients without hemodynamically significant AVD were not further analyzed since their valvular disease was assumed not to have influenced the development of coronary atherosclerosis and their number is too small to serve as statistically significant controls.

The distribution of significant coronary atherosclerosis was not uniform as statistically expected among the 37 patients with hemodynamically significant AVD.

Fourteen patients with significant AS (Group II) had a 64.3 per cent (9/14) incidence of significant CAD ($p < .01$ using Chi-square analysis) (Fig. 1). Six patients with significant AI (Group IV) had a 33 per cent (2/6) incidence of significant CAD (Fig. 2). Although these figures show a definite trend, the number of patients was not high enough for Chi-square analysis.

Seventeen patients with significant combined AS and AI (Group VI) had only an 11.9 per cent (2/17) incidence of significant CAD ($p < .01$ using Chi-square analysis) (Fig. 3).

If the patients with significant AI (Group IV) and those with combined signif-

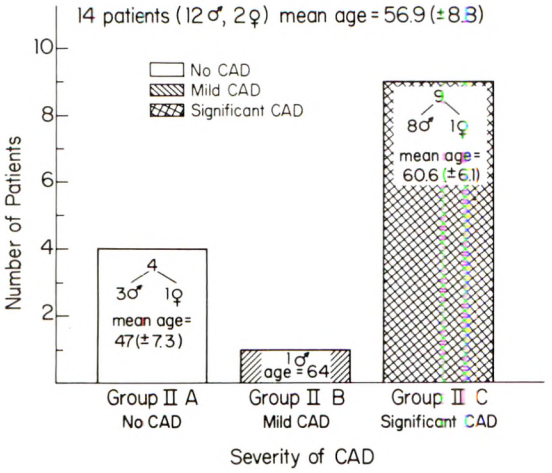


FIG. 1. Group II. Patients with significant aortic stenosis (AS); 64.3 per cent (9/14) of patients with significant AS had significant coronary artery disease (CAD).

icant AS and AI (Group VI) are added (for hemodynamic reasons discussed later), then only 17.4 per cent (4/23) of these patients had significant CAD ($p < .05$ and almost $p < .01$ using Chi-square analysis) (Fig. 4).

The mean age and sexual distribution of patients in all groups with significant AVD was similar:

Significant AS (Group II) mean age

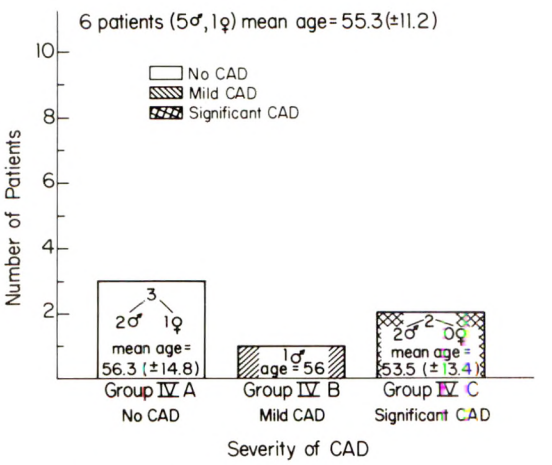


FIG. 2. Group IV. Patients with significant aortic insufficiency (AI). Only 33.3 per cent (2/6) of patients with significant AI had significant coronary artery disease.

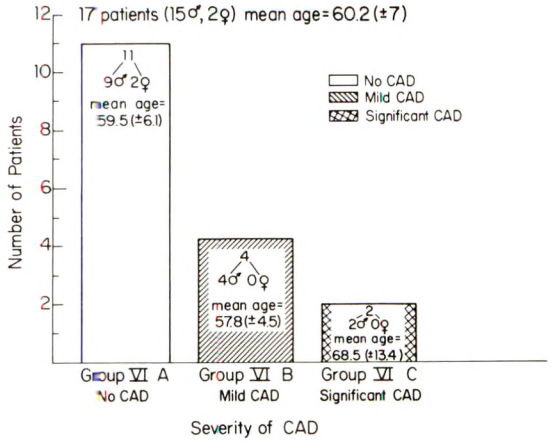


FIG. 3. Group VI. Patients with combined significant aortic stenosis (AS) and aortic insufficiency (AI). Significant AI in combination with AS also appears to have a protective effect against significant coronary artery disease. Only 11.9 per cent (2/17) of these patients had significant coronary artery disease.

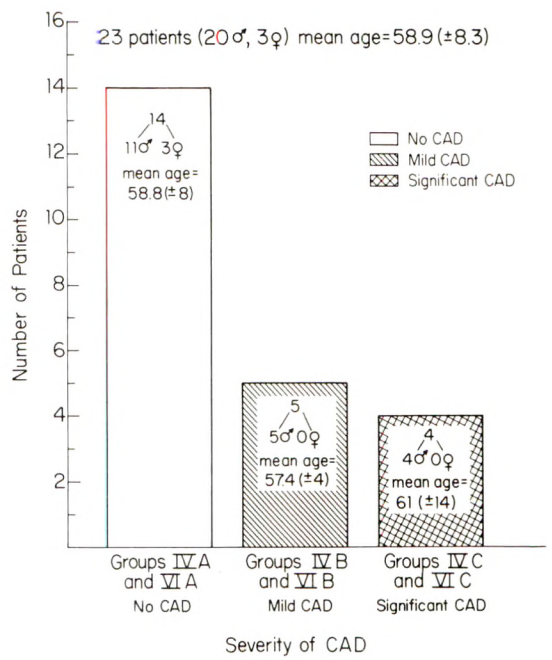


FIG. 4. Groups IV and VI. Patients with significant isolated aortic insufficiency (AI) and significant AI in combination with aortic stenosis (AS). Since significant AI alone or in combination with AS appeared to have a sparing effect on the coronary arteries, these 2 groups were combined, and showed an only 17.4 per cent (4/23) incidence of significant coronary artery disease.

= 56.9 (±8.8) years (14 patients—12 male and 2 female).

Significant AI (Group IV) mean age = 55.3 (±11.2) years (6 patients—5 male and 1 female).

Significant combined AS and AI (Group VI) mean age = 60.2 (±7) years (17 patients—15 male and 2 female).

Significant AI (Group IV) and Significant combined AS and AI (Group VI) mean age = 58.9 (±8.3) years (23 patients—20 male and 3 female).

Angina was present in patients with significant AVD both with and without significant CAD: significant AS (Group II) 6 of 9 patients with, and 2 of 5 patients without significant CAD had angina; significant AI (Group IV) 2 of 2 patients with, and 2 of 4 patients without significant CAD had angina; significant combined AS and AI (Group VI) 0 of 2 patients with, and 4 of 15 patients without significant CAD had angina.

DISCUSSION

Although we attempted to rely on objective data as much as possible, we recognize that a study of live patients presents many pitfalls.

In reviewing the coronary angiograms, it is difficult to quantitate accurately the degree of stenosis. Each coronary artery was imaged in at least 2 different projections perpendicular to one another. In this fashion we tried to reconstruct the lesions in a cross-sectional manner which we feel is more hemodynamically relevant. Nevertheless, under- or over-estimation of the areas of stenoses is possible,[2] and certain lesions could be masked or missed.[8]

The severity of AS was also somewhat difficult to grade with accuracy and reproducibility. The calculated cross-sectional valve area (available in 14 patients) is artificially decreased in the presence of AI. The systolic gradient across the aortic valve is dependent not only upon the cross-sectional area of the valve, but also upon the cardiac output (*i.e.*, myocardial contractility). All patients with AS and an LVEDP of 12 mm. Hg or more generated a peak systolic gradient of at least 30 mm. Hg across their aortic valves (significant AS); therefore, myocardial contractility was not an important factor in the classification of our patients.

The severity of AI is perhaps the most subjective of our data. The opacification of the left ventricle depends in each patient not only on the diastolic open aortic valve cross-sectional area, but also on the position of the catheter,[1] the volume and pressure at which the contrast was injected, and on the functional status of the left ventricle in clearing the regurgitant contrast material.

The lower frequency of significant CAD in patients with significant isolated AI or AI in combination with AS is statistically significant. The mean age (5th decade of life) and sexual distribution of patients was similar in each group as shown above. At least 3 pathophysiologic explanations for this finding appear to be readily possible.

1. It is not true that AI has a protective effect on the coronary arteries, but rather that many patients with significant AI who have significant coexistent CAD, die at a younger age and were somehow excluded from our patient population. In fact, Nakib *et al.*[7] in an analysis of 100 consecutive autopsy hearts of patients with AVD found that 80 per cent (29/36) of patients with AI died in their 4th and 5th decades, and only 20 per cent (7/36) survived into their 6th and 7th decades. On the other hand, only 42 per cent (27/64) patients with AS died in their 4th and 5th decades, and 58 per cent (37/64) survived into their 6th, 7th, 8th and 9th decades. The study of Nakib *et al.*,[7] however, was only based on autopsy material (58 per cent of whom died following aortic valve surgery), and thus does not completely evaluate the natural history of AVD and the distribution of CAD in live patients with AVD.

Our patient population, on whom coronary angiography was performed, includes many in their 3rd and 4th decades.

Since sudden death in AI is not frequent (as it is in advanced AS), we feel that patients (young or old) with AI and coexistent symptoms of significant CAD would have been studied by coronary angiography and heart catheterization. Since they did not appear in our study, we assume that this was due to the very low incidence in the general population of this group of patients, rather than to their elimination by death at a younger age.

2. It is not true that AI protects against significant CAD, but rather that AS predisposes to significant CAD. To evaluate this hypothesis, it is necessary to assess the incidence of significant CAD in the general population in the 5th decade of life. Unfortunately, there are no studies available in live patients. White *et al.*[9] studied 100 consecutive male autopsy hearts of patients in their 5th decade (all causes of death including cardiac), and found an approximately 70 per cent incidence of significant CAD.

Thus, in our population with AS, the incidence of CAD (64.3 per cent) parallels that of the above control group (70 per cent), while patients with AI or AI in combination with AS have a lesser incidence of CAD (33 per cent and 11.9 per cent, respectively).

3. Finally, we are left with the conclusion that AI isolated or in combination with AS either protects against the formation of atherosclerotic plaques or somehow minimizes their effect.

We have observed that in patients with significant AVD, adequate opacification of the coronary arteries (especially the left) is often difficult. This is due to the great dilution of contrast material by the fast flow of large volumes of blood rushing into dilated coronary arteries. The above was observed in patients with AI, AS, and combined AS and AI, and is probably due to myocardial hypertrophy. The increased pulse pressure in AI may contribute an additional increase in flow rate during diastolic coronary artery perfusion. This would be expected to produce an increase in the cross-sectional area of the coronary artery lumen. Thus, equally large eccentric plaques produce a significantly greater degree of hemodynamic obstruction in a normal sized coronary artery than in one with a greater cross-sectional area (Fig. 5).

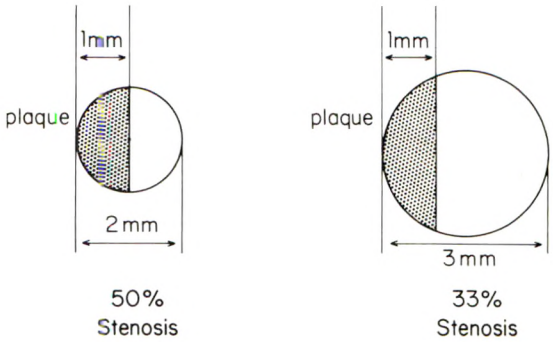


Fig. 5. Equally large intraluminal atheromata produce different degrees of stenosis depending on the diameter of the pre-existent lumen.

The diameter of 6 segments of the coronary artery tree was, therefore, measured and corrected for magnification from films and cine coronary angiography for all 32 male patients with significant AVD in our study, and for a control group of 10 consecutive males without AVD.

The diameter of the main left, proximal and mid left anterior descending coronary arteries, in patients with isolated AI, or AI in combination with AS, and in patients with isolated AS was significantly greater ($p < .005$ to $p < .025$) than in the control group (Fig. 6). The mid circumflex, mid main right and mid posterior descending coronary artery diameters were not statistically different (Fig. 7).

Thus, as expected, both significant AI and significant AS produce enlargement of the coronary arteries. Perhaps the additional increase in diastolic flow in AI due to the increased pulse pressure and regurgitant flow somehow contribute to decreased formation of plaques in addition to the dilatation of the coronary arteries also found in AS.

We feel that this phenomenon, which occurs in both isolated AI and in AI combined with AS, might contribute to the

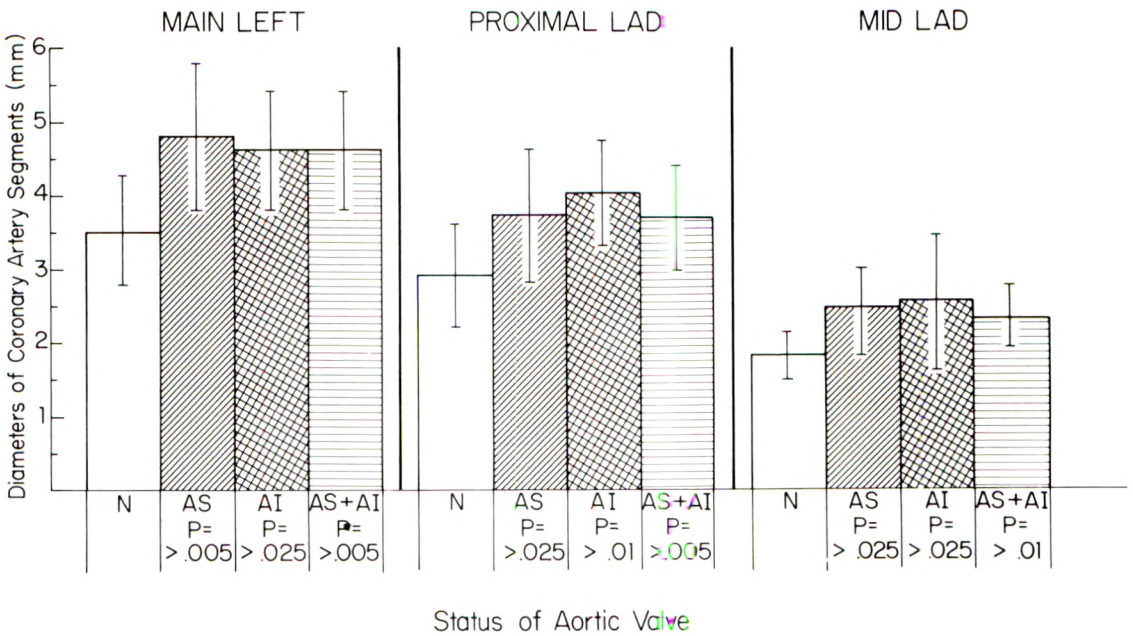


FIG. 6. Aortic valve disease produces a significant increase in the diameter of the proximal branches of the left coronary artery. N = Normal; AS = Aortic Stenosis; AI = Aortic Insufficiency; LAD = Left Anterior Descending Coronary Artery.

lesser incidence of significant CAD in these patients.

Previous reports of the protective value of AS were based on autopsy material, where AS could be assessed but coexistent AI would have been missed.

Our findings closely parallel those of Linhart *et al.*[5] These authors found that 100

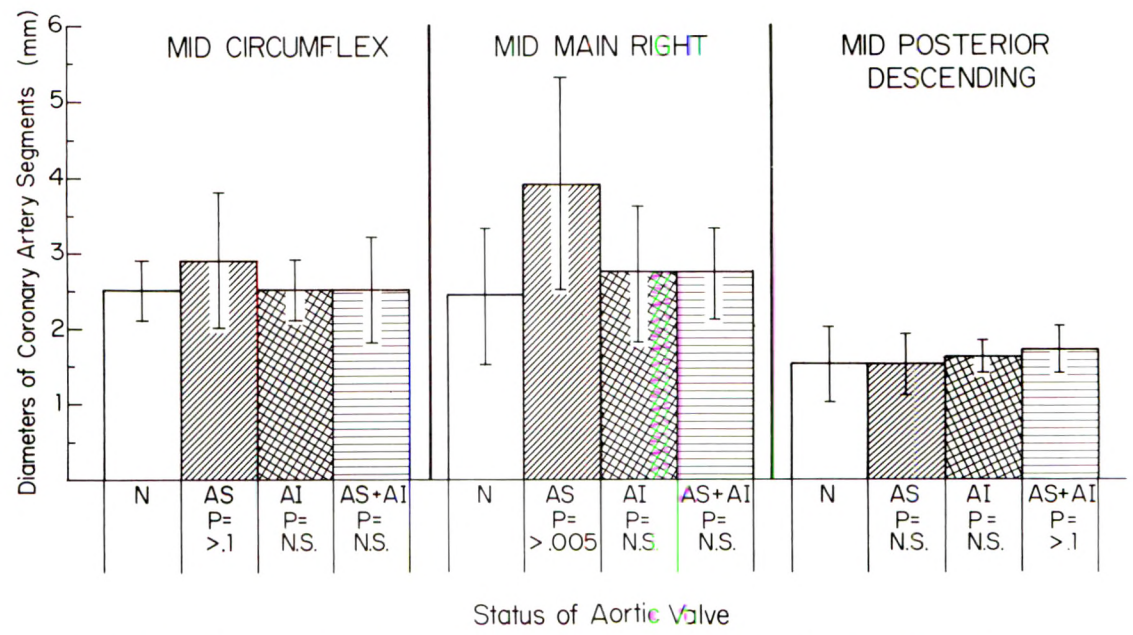


FIG. 7. Patients with and without aortic valve disease have no statistical difference in the diameter of the circumflex and right coronary arteries. N = Normal; AS = Aortic Stenosis; AI = Aortic Insufficiency.

patients evaluated for aortic valve surgery had a 48 per cent incidence of significant CAD. Of these patients, 63 per cent with significant AS and only 30 per cent with significant AI had significant CAD. Linhart *et al.* offer no explanation, and although we also cannot satisfactorily and completely explain our results, we feel that they are significant, and that the pathophysiology needs further investigation.

During aortic valve surgery, the coronary arteries are often cannulated, but satisfactory myocardial perfusion cannot be obtained if severe CAD is present, and, indeed, plaques in the proximal vessel could be dislodged by the cannula. Furthermore, once the aortic valve is replaced, some patients may not be able to be weaned from the pump due to ineffective cardiac function.[6] These patients must have simultaneous coronary artery bypass performed.

The presence or absence of angina was not an accurate indicator of significant CAD present in patients with significant AVD.

Therefore, although our data suggest that patients with AI may be relatively spared, the incidence of significant CAD in significant AVD (especially AS) is so high that all patients considered candidates for aortic valve replacement should have coronary angiography as part of their work-up.

SUMMARY

Forty-two patients with aortic valve disease were evaluated by left heart catheterization and coronary angiography.

Patients with isolated AI or AI in combination with AS have a 17.4 per cent (4/23) incidence of significant CAD, which is significantly less than the 64.3 per cent (9/14) of patients with significant AS. We feel that these findings in AI may be due to a combination of increased diastolic flow and enlarged coronary arteries secondary to cardiac hypertrophy.

Despite the apparent protective value of significant AI against the development of CAD, 40 per cent of our patients with AVD had significant CAD. We feel, therefore, that coronary angiography should be part of the preoperative work-up of candidates for aortic valve surgery.

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REFERENCES

1. Fabian, C. E., and Abrams, C. L. Reflux through normal aortic valves. *Invest. Radiol.*, 1968, *3*, 178–187.
2. Gray, C. R., Hoffman, H. A., Hammond, W. S., Miller, K. L., and Oseasohn, R. O. Correlation of arteriographic and pathologic findings in coronaries in man. *Circulation*, 1962, *26*, 494–499.
3. Honan, M. J., Jr., and Barnes, A. R. Calcaneous aortic stenosis and coronary artery disease. *Am. J. M. Sc.*, 1948, *215*, 451–455.
4. Judkins, M. P. Selective coronary arteriography. I. Percutaneous transfemoral technic. *Radiology*, 1967, *89*, 815–824.
5. Linhart, J. W., de la Torre, A., Ramsey, H., and Wheat, M. W., Jr. Significance of coronary artery disease in aortic valve replacement. *J. Thoracic & Cardiovasc. Surg.*, 1968, *55*, 811–819.
6. Linhart, J. W., and Wheat, M. W., Jr. Myocardial dysfunction following aortic valve replacement: significance of coronary artery disease. *J. Thoracic & Cardiovasc. Surg.*, 1967, *54*, 259–264.
7. Nakib, A., Lillehei, C. W., and Edwards, J. E. Degree of coronary atherosclerosis in aortic valvular disease. *A.M.A. Arch. Path.*, 1965, *80*, 517–520.
8. Sos, T. A., Lee, J. G., Levin, D. C., and Baltaxe, H. A. New lordotic projection for improved visualization of left coronary artery and its branches. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1974, *121*, 575–582.
9. White, N. K., Edwards, J. E., and Dry, T. J. Relationship of degree of coronary atherosclerosis with age in men. *Circulation*, 1950, *1*, 645–654.

PERIRENAL HEMATOMA AS A COMPLICATION OF RENAL INFARCTION IN SICKLE-CELL TRAIT*

A CASE REPORT

By EDWARD A. SICKLES, M.D., *and* MELVYN KOROBKIN, M.D.†
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SPONTANEOUS (nontraumatic) perirenal hematoma can be a complication of a wide range of intrinsic abnormalities of the kidney, the most common of which is neoplasm.[11] Less frequently it may be an extension of a juxtarenal hemorrhage or the result of a systemic (usually cardiovascular) disorder that produces renal infarction and subsequent hemorrhage into the subcapsular and perirenal spaces.[1,9,10] Although several hundred cases of spontaneous perirenal hematoma have been reported in the literature, accurate preoperative diagnosis is seldom made,[12,13] accounting for the high levels of morbidity and mortality for this condition. Recent reports delineating the typical nephrotomographic and angiographic manifestations of perirenal hematoma should greatly facilitate diagnostic evaluation.[3,4,11]

The following case emphasizes the value of an aggressive roentgenographic approach. It is also only the second reported case of perirenal hematoma due to renal infarction of sickle-cell trait.[8] Several roentgenographic and pathologic features of both cases serve to distinguish this type of infarction from the other more common causes of perirenal hematoma.

REPORT OF A CASE

A 48 year old Black man with known sickle-cell trait (hemoglobin AS) was hospitalized for severe pain in the left flank which was nonradiating and steady since its spontaneous and sudden onset 14 hours before admission. There was no history of cardiac dysfunction, hypertension, or renal disease; but blood pressure on admission was 160/115 mm. Hg. Physical examination was unremarkable except for left flank tenderness. Initial hematocrit was 39 per cent and on urinalysis there were 10 to 15 red blood cells per high-power field. A plain film roentgenogram of the abdomen showed extensive pancreatic calcification, aseptic necrosis of the left femoral head, enlargement and blurring of the left renal margin, and absence of the left psoas margin. Excretory urography demonstrated poor concentration of contrast material in the left kidney and a normal-sized nephrogram with a disproportionately small caliber intrarenal collecting system and proximal ureter (Fig. 1). Renal arteriography (Fig. 2, *A* and *B*) performed the same day showed a hypovascular mass in the superolateral cortex of the left kidney with a subcapsular mass impressing the lateral renal margin. No tumor vascularity was identified. Nephrotomograms confirmed the presence of the nonopacified intrarenal mass and also demonstrated outward displacement of Gerota's capsule, indicating a perirenal mass (Fig. 3). Flank pain and hypertension persisted, the hematocrit steadily

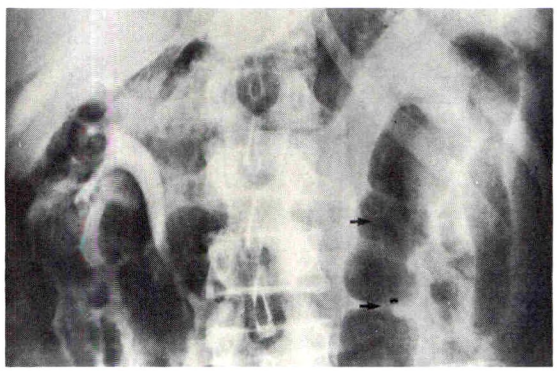


FIG. 1. Excretory urogram showing decreased excretion of contrast material by the left kidney with marked diminution of caliber of the ureter (arrows) and intrarenal collecting system. There is blurring of the margin of the left psoas muscle and extensive pancreatic calcification.

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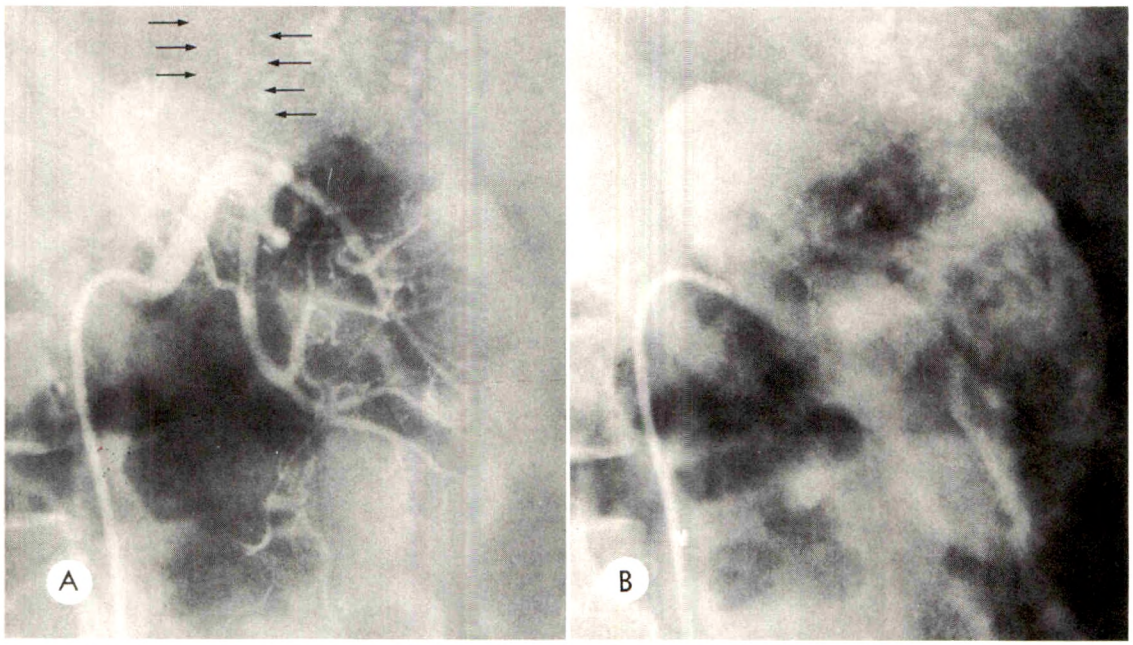


FIG. 2. (*A*) Arterial phase of selective left renal arteriography in left posterior oblique projection, showing capsular arteries (arrows) extending 2 cm. beyond the opacified renal margin. (*B*) Nephrographic phase more clearly showing the opacified renal outline in the same location as the displaced capsular vessels, indicating a subcapsular mass. A subadjacent irregular defect in the renal parenchyma is also seen.

dropped to 30 per cent, and a mass in the left flank became palpable.

The diagnosis of perirenal hematoma was made, with the cause presumed to be spontaneous bleeding from within the left renal lesion and subsequent dissection into the subcapsular and perirenal spaces. It was believed that the intrarenal mass was most likely a neoplasm, but because of the history of sickle-cell trait and documented femoral head infarction, renal cortical infarction was also considered. At laparotomy, a large perirenal hematoma within Gerota's capsule was identified. There was no indication of abdominal or retroperitoneal tumor. Because of uncontrolled bleeding once the renal capsule was entered, nephrectomy was performed. Pathologic examination showed the intrarenal lesion to consist primarily of hemorrhagic necrotic tissue. Surrounding areas of kidney parenchyma showed microscopic foci of ischemic necrosis. Careful study of the renal arterial tree failed to reveal any evidence of thrombus or embolus. No tumor was found. We believe that sickling caused the renal cortical infarction, which then bled into kidney parenchyma, capsule, and Gerota's capsule.

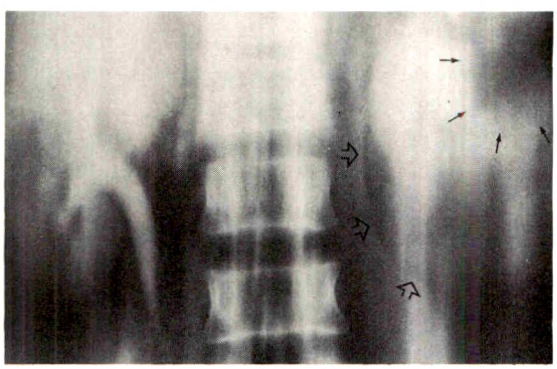


FIG. 3. Nephrotomogram showing the parenchymal defect to be rounded (closed arrows). Medial displacement and bulging of Gerota's capsule (open arrows) by a nonopacifying perirenal mass is also demonstrated.

The patient's recovery was uneventful. The hypertension disappeared immediately following nephrectomy. At the time of discharge from the hospital, the patient had a blood pressure of 130/80 mm. Hg.

DISCUSSION

The diagnosis of spontaneous perirenal hematoma can seldom be made on clinical grounds alone since the presenting signs and symptoms are nonspecific and often

misinterpreted as indicating an intraperitoneal process.[3,12] Accurate preoperative diagnosis is especially difficult in the cases (25 per cent) that demonstrate few if any of the characteristic clinical features.[9,13]

In contrast, the roentgenographic manifestations of perirenal hematoma are easily demonstrated and more specific, making complete roentgenographic evaluation essential to the diagnostic work-up. The supine abdominal roentgenogram usually shows enlargement and indistinctness of the renal outline and absence of the psoas muscle shadow.[1,13] Other, less common, plain film roentgen findings are antalgic scoliosis and elevation of the ipsilateral hemidiaphragm.[9] Excretory urography usually results in nonvisualization of the affected kidney.[3] If function does occur it is almost always delayed and of less magnitude than for the uninvolved side.[5] Despite the enlarged renal outline on plain film roentgenograms, the nephrographic density is of normal size. The kidney is frequently displaced or rotated (in any direction, depending on the location of the major component of the hematoma).[5] The pelvicalyceal system is often elongated and decreased in caliber, and the proximal ureter may be displaced, narrowed, or obstructed by the perirenal mass.[3,5,11] Nephrotomograms, useful only if the affected kidney concentrates the contrast material, may more clearly show compression of opacified parenchyma[3] or, as in our case, demonstrate outward displacement and bulging of Gerota's capsule by a nonopacifying mass. Arteriography is particularly helpful[4,11] because it accurately defines the margins of the renal parenchyma and the capsular vessels. Demonstration of a nonopacifying mass separating capsular vessels from the margin of the parenchyma indicates a subcapsular lesion; a perirenal mass (extracapsular but within Gerota's capsule) is manifested by inward displacement of both the capsular vessels and the opacified renal parenchyma from the renal outline as seen on the plain film roentgenogram.

Nontraumatic perirenal hematoma is known to complicate a variety of conditions, including renal malignancy, nephritis, hydronephrosis, renal artery or aortic aneurysm, periarteritis nodosa, and renal infarction.[1,9,11] Renal infarction most often occurs in patients with underlying cardiac lesions, particularly chronic atrial fibrillation (from rheumatic or atherosclerotic heart disease), left ventricular mural thrombosis (following myocardial infarction), and bacterial endocarditis. The basic cardiac defect leads to formation of a thrombus in the left side of the heart, with subsequent systemic embolization and infarction of the kidney. This type of infarction is, therefore, the result of sudden arterial occlusion. On the other hand, renal infarction in association with sickle-cell trait (or polycythemia) occurs without frank vascular occlusion. The patency of the renal arterial tree has been demonstrated in previously reported cases[2,6,8] as well as in ours, and serves to differentiate infarction caused by capillary blood sludging from the more common embolic type. Embolic renal infarctions are usually not hemorrhagic,[1] whereas those resulting from sludging are more likely to bleed, probably because of accompanying vascular congestion. This may account for the rare occurrence of perirenal hematoma following embolic renal infarction compared to its relatively more frequent association with nonembolic infarctions.

A patient demonstrating the classical clinical features of perirenal hematoma should undergo complete roentgenographic evaluation in an attempt to document and delineate the lesion, unless rapid internal hemorrhage necessitates emergency surgical treatment. In a patient with few if any of the typical signs or symptoms, the possibility of a perirenal hematoma may be suggested only by abnormal findings on plain abdominal roentgenograms or on an excretory urogram. In this case, further evaluation using arteriography (and per-

haps nephrotomography) is imperative to confirm or rule out the diagnosis. Arteriography will also help to exclude many underlying causes of perirenal hematoma; it may, of course, indicate a specific cause. If characteristic roentgenographic features[7] suggest renal infarction, careful examination for underlying cardiac disease should be undertaken. Demonstration of cardiac disease would favor diagnosis of embolic infarction. On the other hand, a history of known sickle-cell trait, or simply a Black patient (especially one with a bone infarction or other manifestations of sickle-cell trait), should alert the radiologist to the possible presence of nonembolic infarction.

Tumor is a prime diagnostic consideration in patients with spontaneous perirenal hematoma, and surgical exploration and subsequent nephrectomy are often performed.[11] Awareness of the association of perirenal hematoma with sickle-cell trait may allow for its preoperative diagnosis; unnecessary nephrectomy may, therefore, be avoided. In sickle-cell trait, as in all systemic diseases, nephrectomy does not remove the underlying defect; with only one remaining kidney the patient would be much more vulnerable to other serious and more common renal complications of sickle-cell trait.

SUMMARY

Accurate preoperative diagnosis of spontaneous perirenal hematoma is often difficult, especially if the clinical presentation is atypical.

Roentgenographic evaluation will demonstrate an enlarged, poorly functioning, or nonfunctioning kidney that may be compressed or displaced by a nonopacifying mass. Nephrotomography can often aid in locating the mass in the perinephric space, but usually renal arteriography is the definitive diagnostic procedure.

Our case of perirenal hematoma due to renal infarction illustrates the features which distinguish the infarction secondary to sickle-cell trait from the much more common embolic type.*

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REFERENCES

1. Amar A. D., and Gray, C. P. Perirenal hematoma following renal infarction. *J. Urol.*, 1963, *89*, 652–655.
2. Femi-Pearse, D., and Odunjo, E. O. Renal cortical infarcts in sickle-cell trait. *Brit. M. J.*, 1963, *3*, 34.
3. Frank, I. N., and Wieche, D. R. Nephrotomographic appearance of spontaneous subcapsular hemorrhage. *Radiology*, 1967, *89*, 477–482.
4. Hare, W. S. C., and Somerset, J. B. Nontraumatic perirenal hematoma: value of arteriography in diagnosis. *J. Urol.*, 1971, *106*, 828–830.
5. Harrow, B. R., and Sloane, J. A. Nontraumatic perirenal hemorrhages. *South. M. J.*, 1962, *57*, 363–369.
6. Kimmelstiel, P. Vascular occlusion and ischemic infarction in sickle cell disease. *Am. J. M. Sc.*, 1948, *216*, 11–19.
7. Lang, E. K., Mertz, J. H. O., and Nourse, M. Renal arteriography in assessment of renal infarction. *J. Urol.*, 1968, *99*, 506–512.
8. Miller, W. A., Peck, D., and Lowman, R. M. Perirenal hematoma in association with renal infarction in sickle-cell trait. *Radiology*, 1969, *92*, 351–352.
9. Ouimet-Oliva, D., Charron, J., and Bélanger, R. Renal infarct with perirenal hematoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 70–76.
10. Polkey, H. J., and Vynalek, W. J. Spontaneous nontraumatic perirenal and renal hematomas: experimental and clinical study. *Arch. Surg.*, 1933, *26*, 196–218.
11. Pollack, H. M., and Popky, G. L. Roentgenographic manifestations of spontaneous renal hemorrhage. *Radiology*, 1974, *110*, 1–6.
12. Stearns, D. B. Spontaneous perirenal hematoma: diagnostic problem. *Am. J. Surg.*, 1954, *88*, 254–260.
13. Uson, A. C., Knappenberger, S. T., and Mellow, M. M. Nontraumatic perirenal hematomas: report based on 7 cases. *J. Urol.*, 1959, *81*, 388–394.

* A recent paper in this Journal (Meyers, M. A., Whalen, J. P., and Evans, J. A. Diagnosis of perirenal and subcapsular masses: anatomic-radiologic correlation, 1974, *121*, 523–538) disputes the validity of displacement of capsular arteries as an aid in differentiating perirenal from subcapsular masses; our case cannot help to resolve this question since both subcapsular and perirenal hematomas were present.

ERRORS IN RENAL VEIN RENIN COLLECTIONS*

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THE measurement of plasma renin activity in the renal veins and inferior vena cava is a safe aid in the diagnosis of renovascular hypertension,[5] but reliable results require strict attention to diet, posture, and technique of collection.[1,2] When the ratio of renal vein renin level on the abnormal side compared to the normal side is 1.4 or greater, an 85 per cent accuracy can be obtained in predicting a successful outcome to surgery for unilateral renal artery stenosis.[1,3,4,6]

In the following 2 patients the importance of a precise location of the collecting venous catheter in the proper draining vein was demonstrated.

REPORT OF CASES

CASE I. J.J. (17-16-35), a 33 year old white female, was admitted for determination of renal vein renins after a 3 year history of hypertension. The onset of systemic hypertension had followed surgery on the right kidney to alleviate hydronephrosis associated with an accessory lower pole artery. A work-up at another hospital just prior to this admission included angiography that showed a surgically amputated accessory right lower pole renal artery and an area of infarction.

The patient was placed on a 10 mEq. sodium diet for 3 days, and selective renin determinations were performed. The results were (Fig. 1): main left renal vein 3,600 ng. per cent; main right renal vein 3,636 ng. per cent; and lower segment of right renal vein 4,916 ng. per cent. Because her hypertension was controlled and she would not accept surgery, she will be followed medically and re-evaluated in 6 months.

CASE II. B.S. (16-62-16), a 59 year old white male, was admitted for evaluation of hypertension which began after an episode of systemic embolization associated with atrial fibrillation. Aortography revealed embolic occlusion of the left renal artery and an absence of function in that kidney. Selective renin determinations were: main left renal vein 2,381 ng. per cent; and main right renal vein 2,034 ng. per cent. Because these values were considered to be possible errors in sampling, repeat determinations were done 3 days later (Fig. 2). They were: left renal vein (distal to adrenal and gonadal veins) 14,815 ng. per cent; left renal vein (proximal to adrenal and gonadal veins) 3138 ng. per cent; and right renal vein 6,571 ng. per cent.

The patient underwent left nephrectomy because of embolic occlusion of the left renal artery. A 6 month checkup at his family

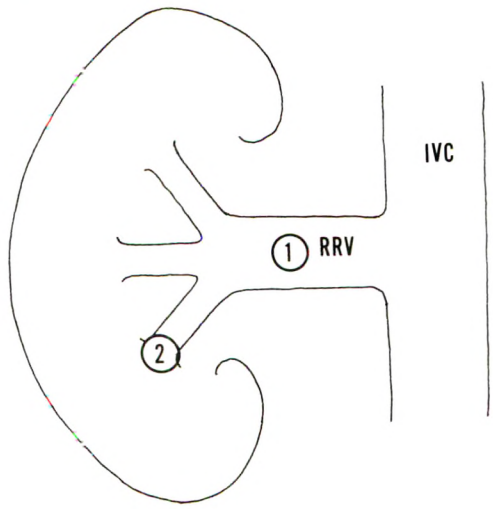


FIG. 1. Case I. Diagram of right kidney. Sampling at Site 1 (main renal vein) resulted in lower renin values (3,636 ng. per cent) than at Site 2 (lower pole segmental vein; 4,916 ng. per cent). This was due to dilution from the middle and upper pole veins at Site 1. IVC = inferior vena cava; RRV = right renal vein.

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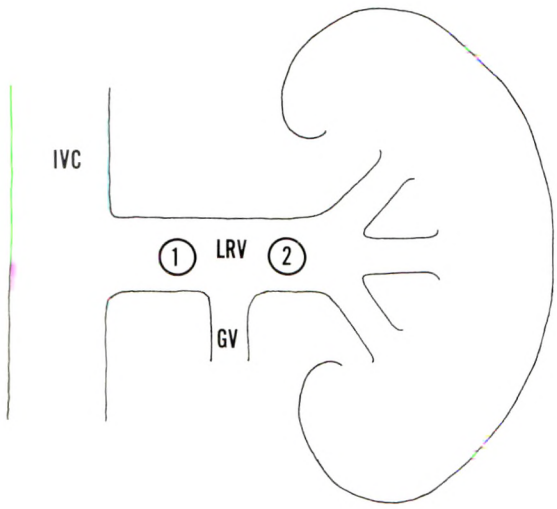


Fig. 2. Case II. Diagram of left kidney. Sampling at Site 1 (proximal renal vein) yielded lower renin values (2,381 ng. per cent) than at Site 2 (distal renal vein; 14,815 ng. per cent). This was due to dilution from the gonadal vein at Site 1. IVC = inferior vena cava; LRV = left renal vein; GV = gonadal vein.

physician's office revealed normal blood pressures.

DISCUSSION

Plasma renin assay has now become an easy, safe, and reliable test in the diagnostic armamentarium for renovascular hypertension.[5] Although usually dependable, misleading values may result from an uncontrolled diet or posture,[2] the administration of antihypertensive medications, an inappropriate sampling technique, or an improperly chosen site. Although some authors suggest the use of 2 catheters, we use a single catheter with one side hole and an end hole. When subselective sampling is necessary, a coaxial catheter system or a controllable-tip guidewire may be helpful. Catheter tip location is tested with 60 per cent Renografin under fluoroscopy and also recorded on videotape. After flushing the catheter with 2–3 ml. of saline, an equal amount of blood is withdrawn and discarded. A second clean syringe is then used to aspirate the sample *slowly*. If aspiration is too vigorous, blood will be drawn from side branches or from the inferior vena cava, retrograde through the renal vein, and into the catheter tip.

The 2 cases discussed here illustrate the need for a thorough understanding of each patient's particular anatomic problem. In Case I, the first sample (Fig. 1) was obtained from the main right renal vein. Because the area of infarction in the lower pole was localized and small in relation to the remainder of the kidney, dilution occurred in the main renal vein or the sample was collected from the portion of the main renal vein "stream" which was derived from the middle and upper poles. Subsequently subselective catheterization of the right lower pole segmental branch revealed renins which were higher than the main vein values in this patient. Analogously, when a localized site of renovascular stenosis or occlusion is present, samples must be obtained from any accessory renal veins.

In Case II, the first set of renin determinations was slightly elevated without a localizing site of abnormality. It was then suspected that wash-out from a large left spermatic vein or other side branches had caused dilution. Repeat sampling distal to the adrenal and spermatic veins yielded a markedly elevated sample (Fig. 2). This case illustrates the need to identify the gonadal vein when catheterizing the left renal vein and the need to aspirate the blood sample slowly, especially when renal blood flow on that side is reduced.

SUMMARY

Two patients in whom renal vein renin determinations were made illustrate 2 of the 3 errors of sampling which may give false-negative results: (1) failure to collect samples from the appropriate tributary or accessory vessel involved in the disease; (2) failure to be selective in the renal vein beyond tributaries from nonrenal sources (*e.g.*, the adrenal vein or the gonadal vein); and (3) failure to withdraw the renal vein

samples slowly (especially in cases of reduced renal blood flow).

Because the presence of positive selective renal vein renins can predict a successful surgical outcome in 85 per cent of the cases of renovascular hypertension, it is essential to collect the venous samples accurately when attempting to distinguish between surgically curable and noncurable forms of hypertension. The described techniques are safe, reliable and easy. The importance of anatomic variations and precise sampling sites is stressed.

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REFERENCES

1. AMSTERDAM, E., COUCH, N., CHRISTLIEB, R., HARRISON, J. H., and CRANE, C. Renal vein renin activity in prognosis of surgery for neovascular hypertension. *Am. J. Med.*, 1969, *47*, 860–868.
2. ASSYKEEN, T. A., CASTELLINO, R. A., LOVE, T. B., and STAMEY, T. A. Peripheral and renal vein renin activity: effects of postural stimulation in normotensive adults. *A.M.A. Arch. Int. Med.*, 1971, *128*, 378–379.
3. BOURGORGINIE, J., KURZ, S., CATANZARO, F., SERIRAT, P. and PERRY, H. M. Renal venous renin in hypertension. *Am. J. Med.*, 1970, *48*, 332–342.
4. ERNST, C. B., BOOKSTEIN, J. J., MONTIE, J., BAUMGARTEL, E., HOOBLER, S. W., and FRY, W. J. Renal vein renin ratios and collateral vessels in renovascular hypertension. *A.M.A. Arch. Surg.*, 1972, *104*, 496–502.
5. PAWSEY, C. G. K., VANDONGEN, R., and GORDON, R. D. Renal venous renin rates in diagnoses of renovascular hypertension: measurement during active secretion of renin. *M. J. Australia*, 1971, *1*, 121–126.
6. STOCKEGT, J. R., NOAKES, C. A., COLLINS, R. D., SCHAMBELAN, M., and BIGLIEN, E. G. Renal vein renin in various forms of renal hypertension. *Lancet*, 1972, *1*, 1194–1197.

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LOCALIZED PULMONARY EDEMA FOLLOWING SURGICALLY CREATED LEFT-TO-RIGHT SHUNTS*

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SURGICAL creation of left-to-right shunts has become an important interim measure in many patients with cyanotic heart disease. The first of these procedures was the subclavian-pulmonary artery anastomosis, introduced by Blalock in 1945[2] as a method of treatment for patients with tetralogy of Fallot. Shortly afterward, the Potts anastomosis (descending aorta to left main pulmonary artery) became available.[7] More recently, in 1962, Waterston constructed an anastomosis of the ascending aorta to the right pulmonary artery, a procedure which has certain technical advantages in those patients for whom definitive surgery is planned.[9]

One of the possible complications following shunt procedures is the development of unilateral pulmonary edema.[5] This complication was initially described by Albers and Nadas in 1967.[1] Subsequently, their clinical and roentgenographic findings were reproduced in experimental animals by Oldham and associates,[6] who verified the existence of preferential flow from the ascending aorta to the right pulmonary artery.

The purpose of this paper is to discuss the roentgenographic aspects of postoperative unilateral pulmonary edema, as well as to describe an even more localized form of congestive change.

CLINICAL MATERIAL

During the past 7 years, 10 patients at the Downstate Medical Center have developed unilateral pulmonary edema following surgical construction of left-to-right shunts.

Figure 1*A* illustrates the roentgen findings in a 4 month old female (L.J.) with a severe tetralogy of Fallot who developed right pulmonary infiltration 4 hours postoperatively. Although this infiltrative pattern could have reflected pulmonary hemorrhage as well as edema, the clinical findings indicated that the patient was in congestive heart failure. Her Waterston shunt was revised approximately 18 hours following the initial procedure and the congestive changes slowly resolved over a 2 week period (Fig. 1*B*).

Unilateral pulmonary edema of this sort is not limited to the Waterston shunt procedure. Figure 2, *A* and *B*, illustrates the findings in a 6 month old female with dextrocardia, common atrium, and pulmonary artery stenosis. Within 24 hours following an anastomosis of the left pulmonary artery to the descending aorta (Potts procedure), she developed roentgen evidence of severe left pulmonary infiltration. Following reduction in the size of the shunt from 5 mm. to 3 mm., her congestive changes resolved over a 1 week period. In the 2 cases described above, pleural effusion was not a major component of the roentgenographic picture, although this has been present in previously reported cases.[1]

Congestive changes generally appear on the side of the anastomotic site, but the anatomy of the lesions may sometimes produce exceptions to this rule. D.P., an 8 week old female with an angiographic diagnosis of single ventricle, pulmonic

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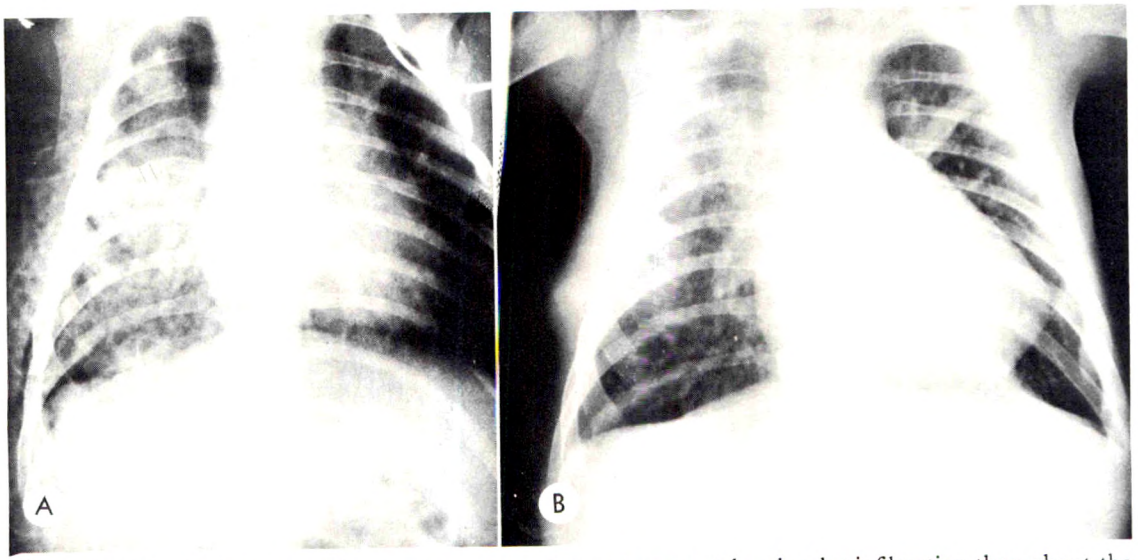


Fig. 1. (*A*) The initial pre-revision roentgenogram demonstrates patchy alveolar infiltration throughout the right lung field several hours after a Waterston shunt. (*B*) This second roentgenogram, obtained 12 days following revision of the Waterston shunt, shows considerable resolution of the parenchymal infiltrate, while some prominence of the right pulmonary arterial tree remains.

valve stenosis, and multiple left peripheral pulmonary artery branch stenoses, had a left-sided Potts anastomosis. Within several hours following the operation, the child began to show signs of congestive heart failure. The chest roentgenogram showed a right-sided infiltrate compatible with pulmonary edema (Fig. 3). Following two revisions of the shunt, the infiltration resolved quickly. Most likely, the multiple left-sided pulmonary artery branch stenoses resulted in a preferential blood flow to the right main pulmonary artery despite the anatomic position of the shunt.

These 3 and 5 additional patients developed unilateral pulmonary edema involving one entire lung, as have all the previously reported cases in the literature. Two of our patients, however, exhibited an even more localized form of pulmonary edema following a Waterston shunt procedure. This was characterized roentgeno-

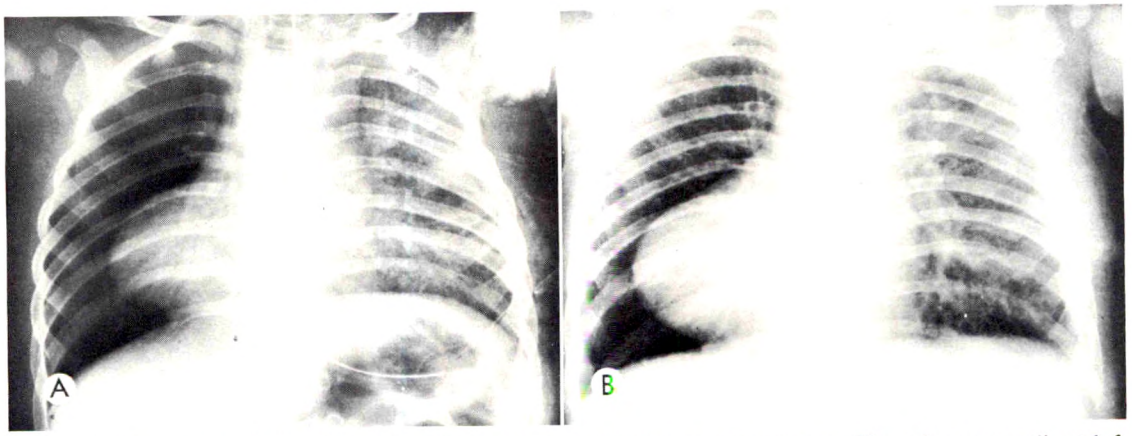


Fig. 2. (*A*) Dextrocardia is evident in this initial postoperative roentgenogram. There is a generalized left lung parenchymal infiltrate 1 day following a Potts anastomosis. (*B*) This roentgenogram, obtained 4 days following shunt revision, demonstrates significant improvement.

graphically by lobar or segmental involvement with bulging of the adjacent interlobar fissures. This phenomenon is a valuable sign allowing localized pulmonary edema to be differentiated from pneumonitis or atelectasis. Outward bowing of the fissures characteristically occurs only in severe necrotizing types of pneumonia. These can be ruled out by the absence of toxic symptoms. In atelectasis, the fissures would bow inward rather than outward. Figure 4, *A* and *B*, illustrates the findings in 1 of these patients. This 2½ year old female (C.L.), previously diagnosed as having tetralogy of Fallot with severe infundibular and valvular pulmonic stenosis, developed signs of congestive heart failure 3 hours after a Waterston shunt. A chest roentgenogram at this time showed a right upper lobe parenchymal infiltrate with caudal bulging of the minor fissure. The diagnosis of localized pulmonary edema was made and the shunt was revised several hours later, at which time the surgeons verified the edematous condition of the right upper lobe. There was rapid clinical improvement thereafter and a roentgenogram 2 days later showed almost complete resolution of the right upper lobe process.

The changes described above were dramatic and developed almost immediately following surgery. Diagnosis of isolated lobar edema can be more difficult when the roentgenographic findings are less obvious and occur somewhat later in the postoperative course. A 10 month old female (T.H.) with tetralogy of Fallot developed a right upper lobe infiltrate with questionable displacement of the minor fissure 4 days following her Waterston shunt. Clinically she had signs of congestive heart failure. The child was both digitalized and placed

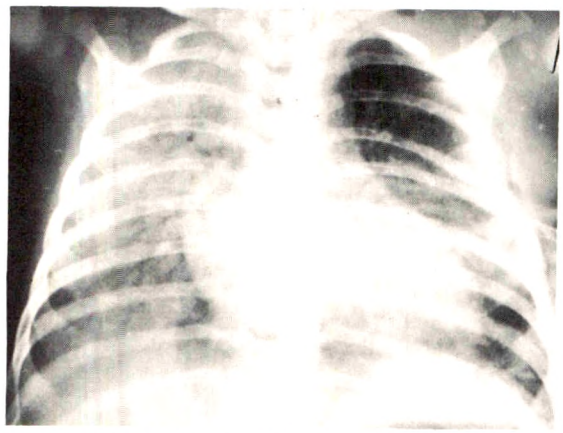


Fig. 3. Diffuse alveolar infiltration is seen throughout the right lung, although the Potts procedure was done on the left. This patient had multiple peripheral stenoses of the left pulmonary artery, which presumably resulted in redirection of the shunt flow to the right.

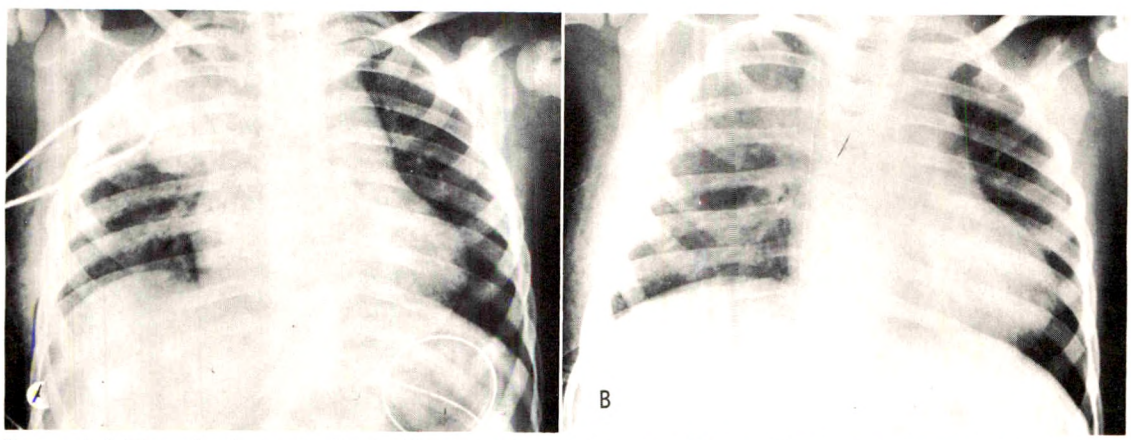


Fig. 4. (*A*) There is almost total opacification of the right upper lobe in this chest roentgenogram obtained 5 hours after the initial Waterston procedure. The distinguishing characteristic in this roentgenographic pattern is the caudal bulging of the minor fissure, reflecting edematous expansion of the right upper lobe. (*B*) Two days following revision of the shunt, the infiltrate has almost completely cleared.

on antibiotics. The right upper lobe infiltrate resolved in 2 days. Three weeks later, the child developed fever without leukocytosis. Parenchymal infiltrate was again noted in the same area. Although slight downward displacement of the minor fissure was evident, the most obvious parenchymal expansion was in the posterior segment of the right upper lobe, reflected by bulging of the major fissure as seen on the lateral roentgenogram (Fig. 5, *A* and *B*). Digitalization was continued without antibiotic therapy and the infiltrate resolved in several days. The patient was discharged 1 week later, still on digitalis.

DISCUSSION

The surgeon who performs Waterston or Potts procedures walks a tightrope between inadequate and overgenerous shunts. Ferencz[3] suggested that the majority of deaths in the early postoperative period resulted from the sudden inflow of a large amount of blood under high pressure, while inadequate shunts accounted for a larger number of deaths among those individuals who survived this period.

It is quite difficult at the time of surgery to measure the effectiveness of the shunt:[4,10] a mere 2 mm. difference in the shunt diameter may spell the difference between inadequate flow and overcirculation. Our surgeons prefer to create a generous shunt and recperate if necessary.[4] They have experienced no increase in mortality with such a policy.

Localized pulmonary edema following shunt procedures probably results from preferential flow of an increased volume of blood at high pressure to the side on which the anastomosis was performed. Somerville *et al.*[8] maintain that this phenomenon is due to kinking at the anastomotic site, with upward displacement of the pulmonary artery. In addition, they describe one individual in whom preferential flow occurred not only to the right lung, but in particular to the pulmonary arterial branches supplying the right upper lobe. An angiogram demonstrated rather severe kinking of the pulmonary artery at the anastomotic site. This provides a reasonable explanation for our more localized pulmonary edema. On the other hand, Waterston *et al.*[10] feel that even when the

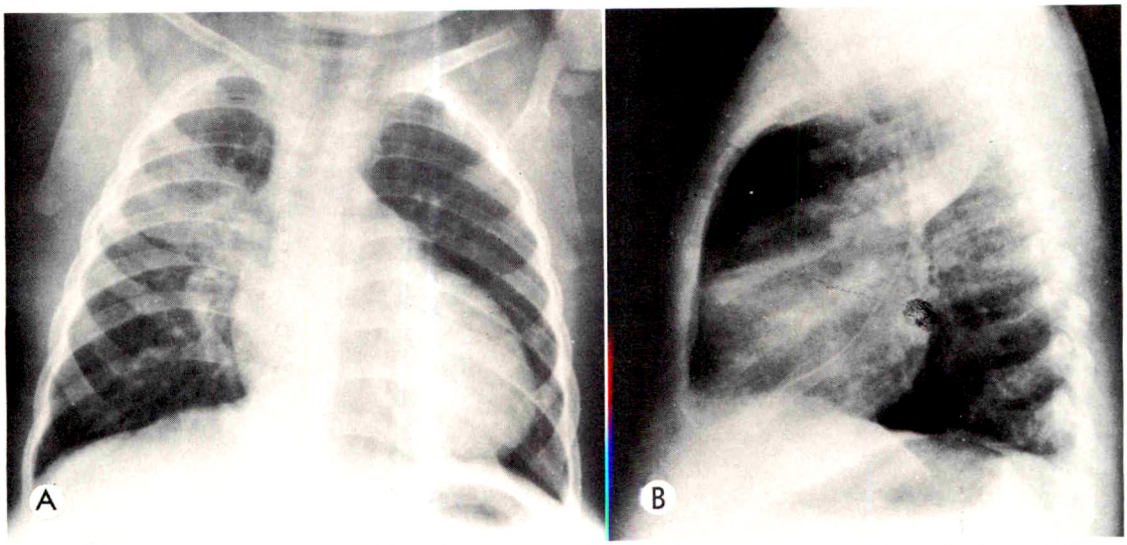


FIG. 5. (*A* and *B*) Parenchymal infiltrate, primarily in the posterior segment of the right upper lobe, is demonstrated in these roentgenograms obtained 3 weeks after a Waterston shunt. Note particularly the posterior displacement of the major fissure seen on the lateral roentgenogram.

shunt is positioned optimally, preferential flow occurs in a certain percentage of cases.

In our experience, postoperative thoracic aortography generally demonstrates this preferential flow to the ipsilateral lung. Selective pulmonary angiography, on the other hand, shows preferential flow to the contralateral lung with little or no flow passing beyond the anastomosis in antegrade fashion. This would seem to confirm some degree of mechanical obstruction in the pulmonary artery due to kinking in the region of the anastomotic site. However, in most cases, withdrawal pressure tracings fail to reveal a gradient between the proximal and distal segments of the involved pulmonary artery.[4] The exact cause of the preferential distribution of flow to one lung or lobe therefore remains unclear.

In any event, the diagnosis of localized pulmonary edema should be strongly considered whenever infiltration is roentgenographically demonstrated in the ipsilateral lung or a portion thereof following surgical creation of left-to-right shunts. Fissure displacement reflecting lobar or segmental expansion has proven a reliable means of differentiating the more localized form of edema from pneumonitis or atelectasis. The lateral chest roentgenogram may be quite helpful (as illustrated in Figure 5*B*) and should be obtained in doubtful cases.

SUMMARY

The roentgen appearance of localized postoperative pulmonary edema in patients who have undergone palliative systemic-pulmonary shunt surgery is discussed.

Unilateral whole lung edema may occur, as well as previously undescribed congestive changes isolated to the right upper lobe in post-Waterston shunt patients.

The presence of a bulging fissure, in addition to parenchymal infiltrate, is important in differentiation of the above from pneumonitis or atelectasis. The cause of this is felt to be preferential distribution of flow, in most cases, from the shunt itself to the ipsilateral lung or one of its lobes or segments.

Prompt recognition of this particular postoperative problem is essential for the successful management of these patients.

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REFERENCES

1. Albers, W., and Nadas, A. S. Unilateral chronic pulmonary edema and pleural effusion after systemic pulmonary arterial shunts. *Am. J. Cardiol.*, 1967, *19*, 861–866.
2. Blalock, A., and Taussig, H. G. Surgical treatment of malformations of heart in which there is pulmonic stenosis or pulmonary atresia. *J.A.M.A.*, 1945, *128*, 189–202.
3. Ferencz, C. Pulmonary vascular bed in tetralogy of Fallot. II. Changes following systemic pulmonary arterial anastomosis. *Bull. Johns Hopkins Hosp.*, 1960, *106*, 100–118.
4. Garzon, A. A., Goldstein, S., Ehrlich, R., Rodriguez-Torres, R., and Karlson, K. E. Technical and hemodynamic considerations of Waterston's shunt. *J. Cardiovasc. Surg.*, 1972, *13*, 240–245.
5. Hipona, F. A., Paredes, S., and Lerona, P. T. Roentgenologic analysis of common postoperative problems in congenital heart disease. *Radiol. Clin. North America*, 1971, *9*, 229–251.
6. Oldham, H. N., Jr., Simpson, L., Jones, R. H., Goodrich, J. K., and Sabiston, D. C., Jr. Differential distribution of pulmonary blood-flow following aortopulmonary anastomosis. *S. Forum*, 1970, *21*, 201–202.
7. Potts, J. W., Smith, S., and Gibson, S. Anastomosis of aorta to pulmonary artery. *J.A.M.A.*, 1946, *132*, 627–632.
8. Somerville, J., Yacoub, M., Ross, D., and Ross, K. Aorta to right pulmonary artery anastomosis (Waterston's operation) for cyanotic heart disease. *Circulation*, 1969, *39*, 593–602.
9. Waterston, D. J. Treatment of Fallot's tetralogy in children under one year of age. *Rozhl. chir.*, 1962, *41*, 181–183.
10. Waterston, D. J., Stark, J., and Ashcroft, K. Ascending aorta to right pulmonary artery shunts: experience with 100 patients. *Surgery*, 1972, *72*, 897–904.

PRINZMETAL'S VARIANT ANGINA PECTORIS*

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ANGINA pectoris (AP) was originally described by Heberden over 2 centuries ago and since that time has become one of the most widely studied afflictions of mankind. The characteristic chest pain in AP is due to ischemia which occurs when myocardial oxygen consumption exceeds the capacity of diseased coronary arteries to deliver oxygen to myocardial cells.[7] Virtually anything which increases cardiac work can precipitate an ischemic attack, including physical activity, emotional upset or excitement, cold weather, hypertensive or hyperthyroid crises and even ingestion of a large meal. The typical electrocardiographic (ECG) abnormalities in patients with AP are displacement of the S-T segment, especially depression, and changes in T wave amplitude and polarity.[1,6]

In 1959, Prinzmetal *et al.*[15] described an unusual or variant form of angina. Although characterized by the same type of chest pain, it differed strikingly from "classical" AP in its mode of onset and the accompanying ECG changes. The pain in Prinzmetal's syndrome developed at rest or with light activity and was not at all associated with any of the precipitating factors listed above. ECG tracings obtained during attacks, instead of showing the familiar S-T segment depression, showed a transient S-T elevation which rapidly reverted to normal after the pain subsided. The authors postulated that the clinical manifestations of variant angina pectoris were produced by spasm of a single stenotic segment of a major coronary artery with resultant temporary occlusion of the vessel. Since the original description in 1959, approximately 115 cases of Prinzmetal's variant angina (PVA) have been reported[12] and many additional aspects of it have come to light. Although the entity has attracted much interest among cardiologists, it has not been previously discussed in the radiology literature. This may be due in part to the fact that many of the reported cases, including Prinzmetal's original group, have not had angiographic documentation of their disease.

The purpose of this paper is to describe the clinical, ECG, hemodynamic and arteriographic aspects of PVA, based upon the previously reported case material and our own experience with 11 patients. Particular emphasis will be placed upon the arteriographic findings and their role in the pathophysiology of the syndrome.

CLINICAL FEATURES

The clinical hallmark of PVA is chest pain which is similar in nature and location to that of classical AP, but which is not induced by physical activity or any of the other known provocative factors. It generally commences at rest or during periods of light activity in individuals who in fact have normal or nearly normal exercise tolerance.[1,12,14,15] The patients frequently report that their pain has a remarkable tendency to occur at the same time of each 24 hour period, especially during sleep or immediately after arising in the morning.[1,12,14,15] The episodes may take the form of a series of cycles or waves of pain,[12,14] each cycle lasting up to several minutes and recurring as many as 40 times during a 2 hour period. Attacks may occur daily for weeks or months, then remit spontaneously for extended intervals.

All the features described above are atypical of AP, which instead tends to be precipitated by physical or emotional exertion, which does not generally exhibit a cyclic pattern and which does not show prolonged spontaneous remissions without

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vigorous medical or surgical therapy or infarction of the affected myocardium. The major clinical similarities between PVA and typical AP are that both respond to the administration of nitrates and that neither is accompanied by elevation of serum enzymes.

In most cases, PVA appears *de novo* in patients with no prior manifestations of coronary artery disease. On occasion, however, it will develop in individuals who have a history of the more common type of effort-induced AP or previous myocardial infarction.

ELECTROCARDIOGRAPHIC CHANGES

The principal ECG manifestation of myocardial ischemia during an attack of AP is depression of the S-T segment.[14,15] By contrast, patients with PVA exhibit S-T segment elevation, a finding which often represents the earliest ECG evidence of overt myocardial infarction rather than mere ischemia.[1,3,12,14,15] A single ECG taken during the height of an attack of PVA may thus be misinterpreted as showing the presence of an infarct. This alteration, however, is only transitory and the S-T segments rapidly revert to their baseline position, when the pain ceases spontaneously or is relieved by nitroglycerin. Electrocardiograms from 3 different patients with PVA are shown in Figures 1–3. The changes can generally be localized to the distribution of either the right or anterior descending coronary arteries. Right coronary lesions produce S-T segment elevations in leads II, III, and a VF, whereas left anterior descending artery lesions produce them in I, aVL and the precordial leads.[3,12,14,15]

Other abnormalities which may coincide with the pain include increase in amplitude of both R and T waves and occasional merging of the R wave, S-T segment and T wave into a broad complex suggesting a monophasic action potential.[12] Serious arrhythmias also occur frequently, the most common of these being premature ventricular contractions, varying degrees of atrioventricular block, ventricular tachycardia and occasional ventricular fibrillation.[3,12,22]

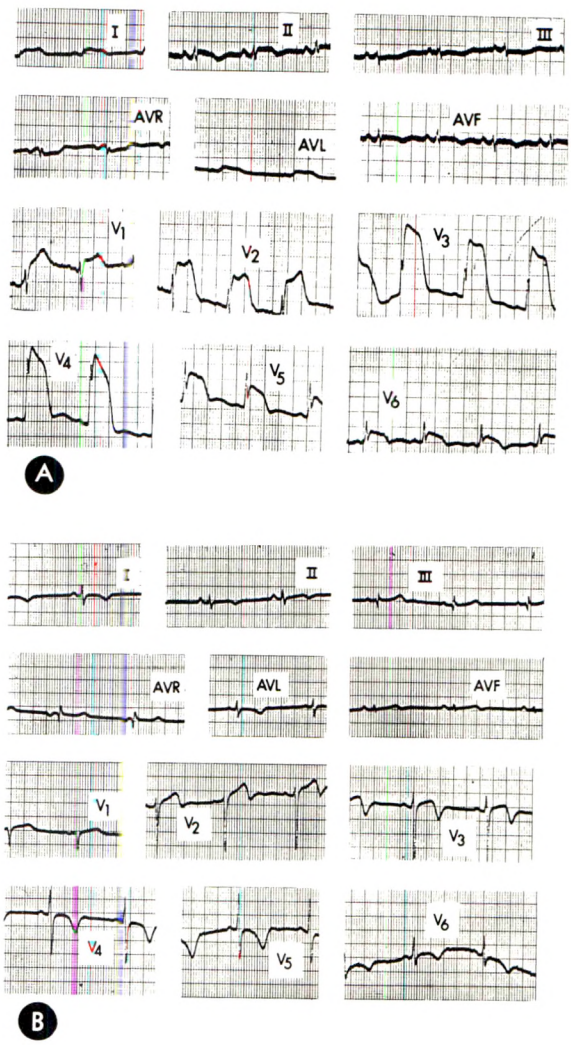


FIG. 1. (*A*) Electrocardiogram (ECG) during an attack of chest pain in a 30 year old male with Prinzmetal's angina. Note S-T segment elevation in leads I, II, aVL and, most strikingly, the precordial leads. These changes suggest a lesion affecting blood supply of the anterior and anterolateral aspects of the left ventricle. Coronary arteriography, shown in Figure 4, *A* and *B*, revealed severe stenosis of the left anterior descending artery. (*B*) After termination of the attack, the previously noted marked S-T segment elevations are no longer present. T wave inversion in leads I, II, aVL and V2-6 is now present and is consistent with myocardial ischemia.

Although the ECG abnormalities in PVA were originally observed during attacks of pain, recent reports have indicated unequivocally that some of these patients

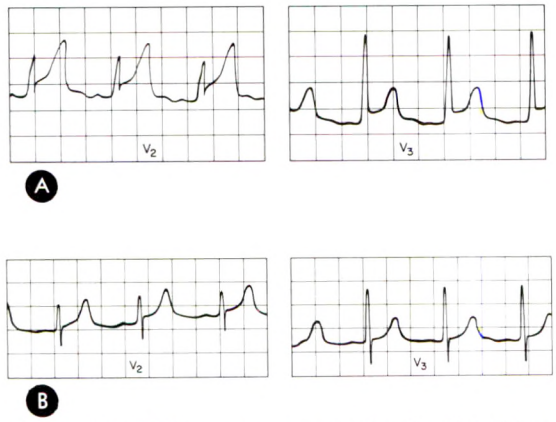


FIG. 2. (*A*) ECG in a 38 year old female with Prinzmetal's angina. During an attack, S-T segments are elevated in leads V2 and V3, suggesting a lesion in the distribution of the left anterior descending artery. Stenosis of this vessel was confirmed at coronary arteriography, as shown in Figure 7. (*B*) Following the anginal episode, the S-T segments have returned to a nearly isoelectric position.

periodically develop transient S-T segment elevations at times when they are completely asymptomatic.[3,9,12,17] This phenomenon was observed in 4 of our patients during extended monitoring in a Coronary Care Unit.

HEMODYNAMIC ASPECTS

As indicated earlier, ischemia occurs when the capacity of the coronary arteries to deliver oxygen is exceeded by myocardial oxygen consumption. The latter is related to the magnitude of systolic wall stress, the interval of time during which the stress is exerted and the inotropic state of the heart.[7] These complex factors are difficult to assess directly, but a reliable index of myocardial oxygen consumption can be simply obtained as the triple product of systolic pressure, heart rate and left ventricular ejection time. Previous studies have indicated that both effort-induced and spontaneous, non-Prinzmetal attacks of AP are generally preceded by elevation of one or more of these 3 determinants, especially systolic pressure and heart rate.[7,16,18,19]

By contrast, Guazzi *et al.*[10] showed that attacks of PVA are *not* preceded by hemodynamic alterations which increase myocardial oxygen consumption. Instead, these alterations occurred simultaneously with the onset of the ECG changes. This fact reinforces the theory that PVA results from a paroxysmal phenomenon such as spasm.

In both AP and PVA, left ventricular function is significantly impaired during the course of the attack itself.[10,18] One patient in our group underwent continuous intracardiac and intra-arterial pressure monitoring as an attack of PVA developed. As in the patients of Guazzi *et al.*, hemodynamic abnormalities did not precede the attack. During the attack, the patient's



FIG. 3. ECGs in a 56 year old female with severe stenosis of the midportion of a dominant right coronary artery, as show in Figure 8. The upper tracing shows S-T segment elevation in lead II during a typical episode of Prinzmetal's angina. The lower tracing demonstrates return of the S-T segment to the baseline following administration of nitroglycerin and relief of pain.

systemic blood pressure rose from 130/80 to 170/120 mm. Hg. Left ventricular end diastolic pressure rose from 12 to 42 mm. of mercury, indicating acute impairment of left ventricular function.

ARTERIOGRAPHIC FINDINGS

There is a relative paucity of information regarding coronary arteriography in patients with PVA. MacAlpin *et al.*[12] discussed the angiographic findings in 19 cases and 18 additional ones have been added by other groups.[2,5,13,21,22] All 11 of our patients have been studied angiographically. An interesting and rather unusual pattern becomes apparent if one analyzes the changes seen in these 48 cases.

Thirty-one individuals with PVA (65 per cent) were found to have severe focal proximal narrowing of a single major coronary artery. The left anterior descending artery was involved in 15 cases, the right coronary artery in 10, the left circumflex artery in 4, and the left main coronary artery in 2. In virtually all of these cases, the distal segments of the arteries were free of significant disease and showed rapid and complete "runoff" of contrast material. Examples of 5 of our patients with these focal lesions are shown in Figures 4–8. Thirteen additional patients (27 per cent) had angiographically normal vessels. The remaining 4 patients (8 per cent) had double or triple vessel disease. Only 2 instances of complete occlusion were found, both occurring in patients with double or triple vessel disease. Collateral circulation was not seen in individuals with normal coronary arteries, or in those with focal, single vessel stenosis, no matter how severe the narrowing. Patients in these 2 categories also demonstrated normal contractility of the left ventricle at ventriculography. The unusual aspect of this distribution of lesions is that there were so many localized narrowings of a single artery and so few instances of multiple vessel disease or total occlusions. In unselected groups of patients with coronary artery disease, the incidence of both multiple vessel disease and total occlusions is considerably greater.[11]

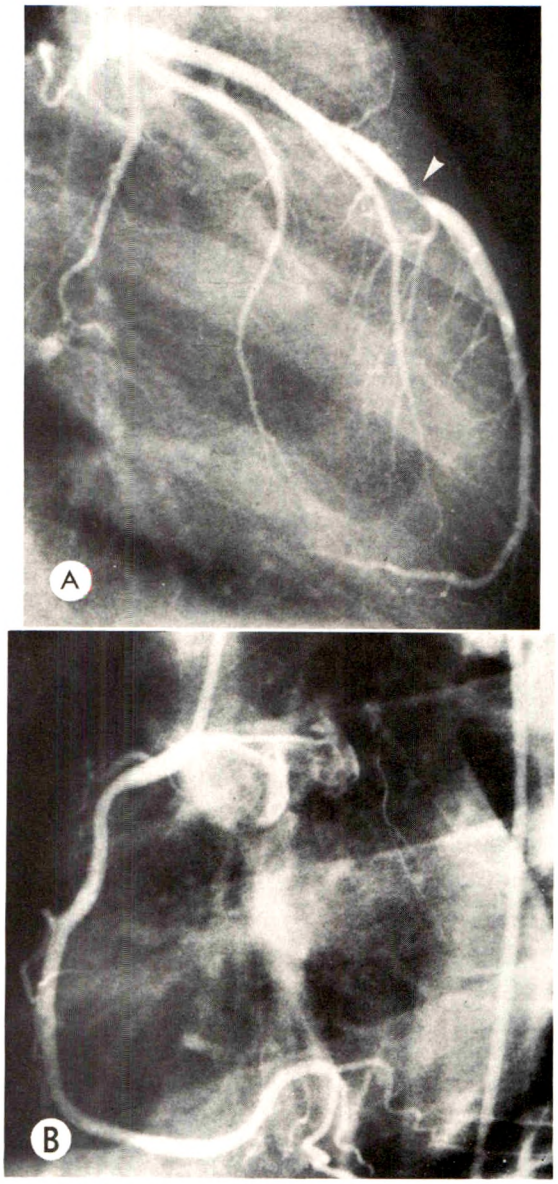


Fig. 4. (*A*) Left coronary arteriogram, right anterior oblique view, in a 30 year old male drug addict with Prinzmetal's angina. There is severe stenosis of the left anterior descending artery (arrowhead). No other lesions are seen. The patient's ECG is shown in Figure 1, *A* and *B*. (*B*) The right coronary artery in this patient is entirely normal.

The most dramatic angiographic aspect of this disease has been the recent demonstration of coronary artery spasm during typical attacks of PVA in a small group of patients.[3,5,13,20] Two such cases were reported by Cheng *et al.*[3] in patients who

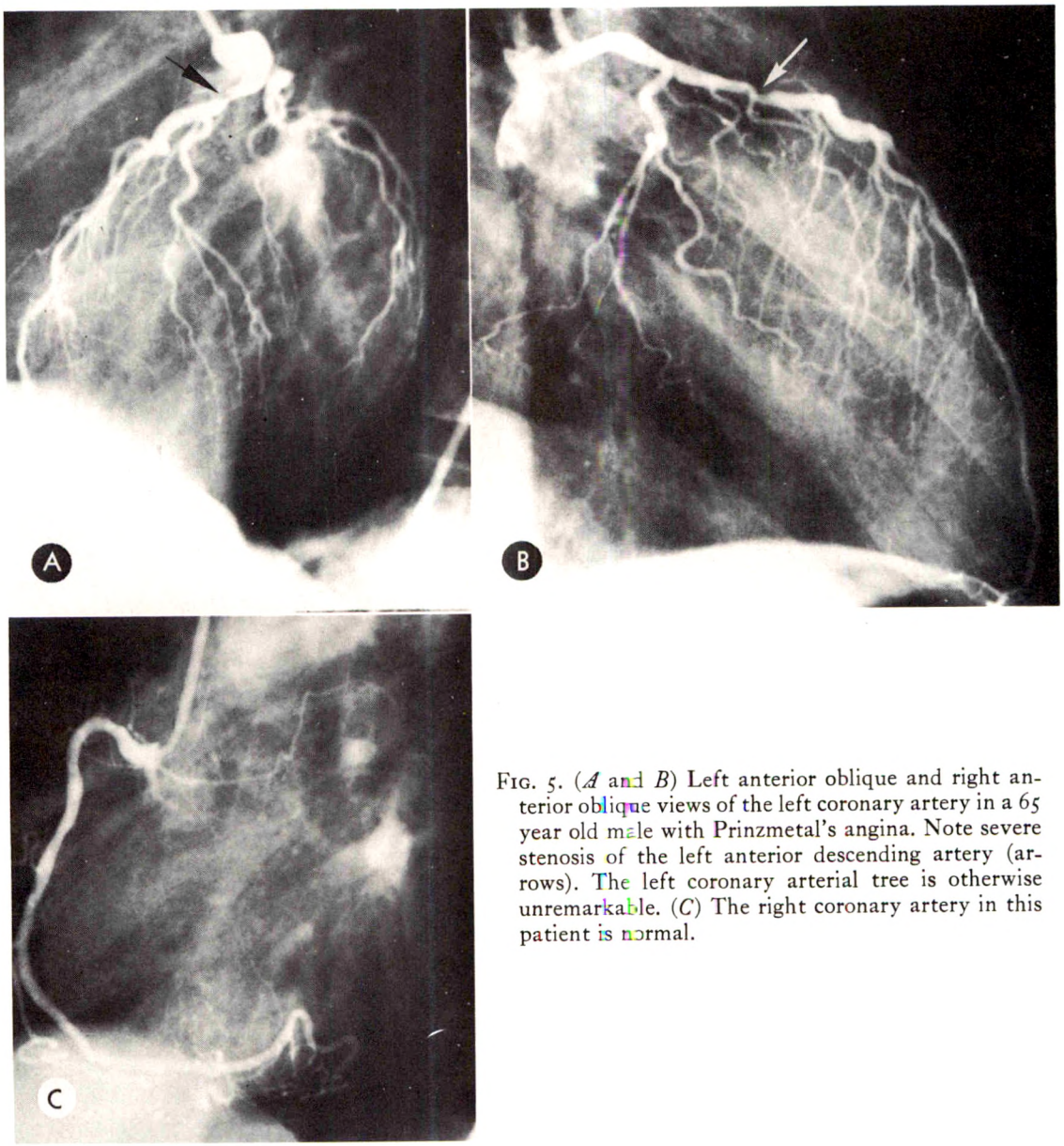


FIG. 5. (*A* and *B*) Left anterior oblique and right anterior oblique views of the left coronary artery in a 65 year old male with Prinzmetal's angina. Note severe stenosis of the left anterior descending artery (arrows). The left coronary arterial tree is otherwise unremarkable. (*C*) The right coronary artery in this patient is normal.

otherwise had normal coronary arteries. Both had typical signs and symptoms of PVA and developed severe chest pain associated with S-T segment elevation during selective coronary arteriography. One patient was seen to have transient severe narrowing of the distal right coronary artery (RCA), while the other had transient narrowing of the proximal RCA just beyond the catheter tip. The narrowed segments rapidly reverted to normal caliber following administration of sublingual nitrates. An especially well-documented case has been presented by Oliva *et al.*,[13] who performed a series of selective arteriograms of the RCA during multiple consecutive transient attacks of PVA in a female patient. During each attack, severe spasm of the RCA was noted, but the site, extent and degree of obstruction varied from one to the next. In between attacks, the RCA appeared entirely normal and no further at-

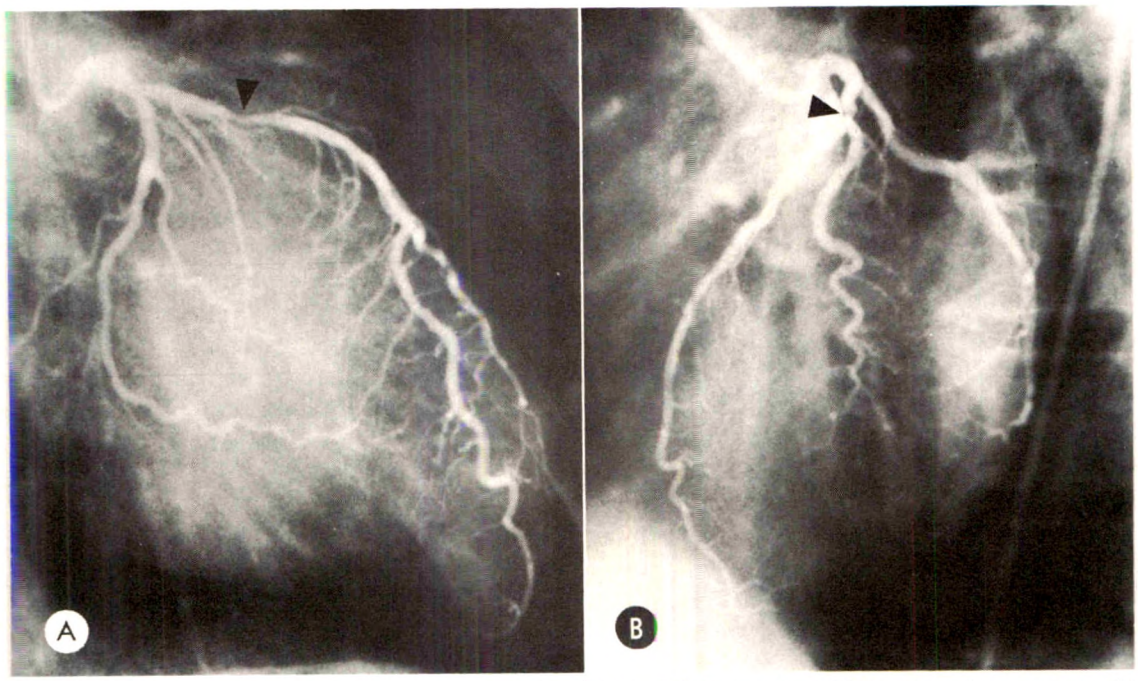


FIG. 6. (*A* and *B*) Left coronary arteriograms in the right and left anterior oblique views in a 72 year old male with Prinzmetal's angina. There is severe proximal stenosis of the left anterior descending artery (arrowheads). Aside from the lesion, both coronary arteries were normal.

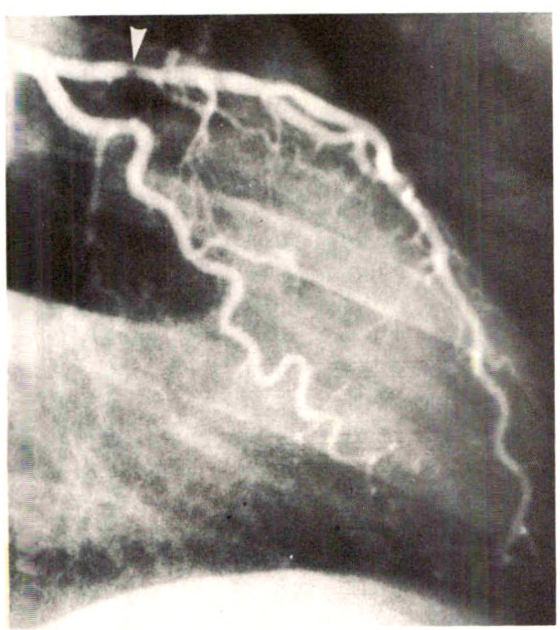


FIG. 7. Left coronary arteriogram, right anterior oblique view. The patient was a 38 year old female, part of whose ECG is shown in Figure 2, *A* and *B*. Severe proximal narrowing of the left anterior descending artery is present (arrowhead). The right coronary artery was unremarkable.

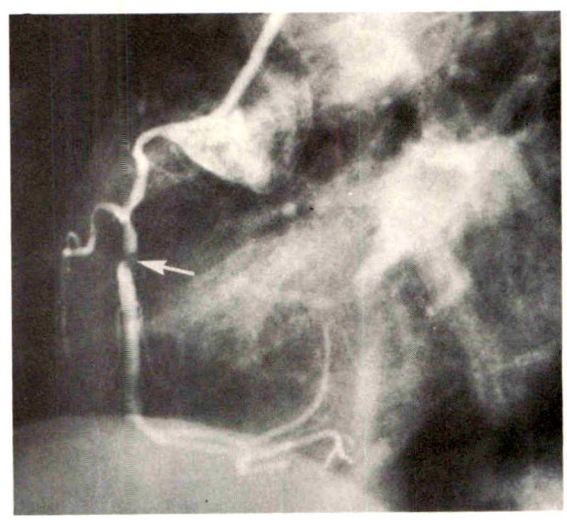


FIG. 8. Right coronary arteriogram, left anterior descending view. The patient was a 56 year old female with typical Prinzmetal's angina, part of whose ECG is shown in Figure 3. Severe stenosis of the mid portion of the right coronary artery is present (arrow). The left coronary arteriogram was normal.

tacks occurred after administration of sublingual nitroglycerin. A fourth case has been reported by Dhurandhar *et al.*,[5] who demonstrated proximal RCA spasm at the site of an atherosclerotic plaque, associated with the onset of chest pain in a patient with the typical clinical features of PVA. The authors, however, did not indicate whether S-T segment elevation occurred during this episode. As in the other cases, the spasm rapidly disappeared after sublingual nitrates. Three additional cases with angiographically-documented spasm have been briefly reported by Ruggeroli *et al.*,[20] but no details of these cases are available as yet. We have not observed this phenomenon in our group of patients, since none of them experienced an attack while arteriography was in progress.

Catheter-induced spasm of the proximal RCA is a common occurrence during selective coronary arteriography, particularly if patients are not premedicated with nitrates. The question might therefore be raised as to whether the above cases were simply examples of catheter-induced spasm during the procedures. This seems unlikely, since catheter-induced spasm is rarely accompanied by significant chest pain or S-T segment elevation.

DISCUSSION

Prinzmetal *et al.*[15] originally hypothesized that their variant form of angina pectoris resulted from episodic spasm of an already-narrowed segment of a major coronary artery, resulting in transient complete obstruction of the vessel. The clinical features of the syndrome amply support the validity of this theory. The development of attacks at rest, lack of reproducibility with exercise, cyclic nature, rapid onset and rapid relief with nitroglycerin, and the frequency of prolonged remissions all bespeak a paroxysmal phenomenon, rather than one related purely to a fixed anatomic obstruction. Furthermore, temporary ligation of a major coronary artery in experimental animals has been found to produce an identical S-T segment elevation, which quickly reverts to normal after release of the ligature.[15] The recent arteriographic demonstration of localized spasm during attacks of PVA has confirmed the role of this mechanism in the genesis of the disease. In addition, it has become quite apparent that significant atherosclerotic disease need not be present, but that spasm alone may account for the symptoms in patients whose coronary arteries are anatomically normal.

The prognosis for patients with PVA appears to be guarded during the early stages. MacAlpin *et al.*[12] report that approximately 20 per cent of patients die suddenly, while another 20 per cent experience myocardial infarction in the area predicted by the distribution of S-T segment elevation. Most of these complications occur during the first 3 months. Beyond this point, symptoms frequently abate and the prognosis improves.

There remains some controversy over the optimal mode of therapy for patients with PVA. Goodin *et al.*,[8] and Silverman and Flamm[21] have stated that bypass surgery is the treatment of choice, whereas Mac Alpin *et al.*,[12] and Oliva *et al.*[13] prefer an initial trial of vigorous medical therapy. In our experience, surgical results have been discouraging. Of 7 patients undergoing saphenous vein bypass procedures, 4 died during or shortly after surgery. A fifth patient had an initially good result, but then suffered an anterior myocardial infarction when his left anterior descending artery bypass thrombosed. The other 2 individuals have had good long-term results. Of the 4 patients treated medically, 1 has had a good long-term result, 1 has had slight improvement, and 2 have been lost to follow-up.

There is certainly some question as to whether coronary bypass can be fully effective against spasm, which can affect the arterial segment distal to the anastomosis as well as the segment proximal to it. Our current recommendations, therefore, coincide with those of MacAlpin *et al.*,[12] *i.e.*, that close observation and medical therapy be instituted, especially during the first 3 months after onset of symptoms. Bypass

surgery should be reserved only for those patients with unremitting pain or uncontrollable arrhythmias.

SUMMARY

The clinical, electrocardiographic, hemodynamic and arteriographic features of Prinzmetal's variant form of angina pectoris are discussed, based upon previously reported case material and our own experience with 11 patients.

Although the chest pain of Prinzmetal's angina results from myocardial ischemia, the syndrome differs from the classical form of angina pectoris in many respects. In particular, Prinzmetal's angina develops at rest or with light activity and is accompanied by S-T segment elevation on the electrocardiogram, while classical angina pectoris is precipitated by increased cardiac work and is frequently accompanied by S-T segment depression.

Arteriography in most cases reveals either focal stenosis of a single major coronary artery or anatomically normal vessels. Only rarely are multiple vessel disease or total obstructions seen.

There is strong clinical evidence suggesting that Prinzmetal's angina is produced by transient paroxysmal spasm of a major coronary artery. This has been verified recently by actual angiographic demonstration of spasm while attacks were in progress.

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REFERENCES

1. Aisner, M. Angina pectoris, 1768–1970. *New England J. Med.*, 1970, *282*, 746–748.
2. Bobba, P., DiGuglielmo, L., Vecchio, C., and Montemartini, C. Coronarographic patterns in Prinzmetal's variant angina. *Acta cardiol.*, 1971, *26*, 568–580.
3. Cheng, T. O., Bashour, T., Kelser, G. A., Jr., Weiss, L., and Bacos, J. Variant angina of Prinzmetal with normal coronary arteriogram: variant of variant. *Circulation*, 1973, *47*, 476–485.
4. Cosby, R. S., Giddings, J. A., See, J. R., and Mayo, M. Variant angina. *Chest*, 1971, *60*, 292 (Abstr.).
5. Dhurandhar, R. W., Watt, D. L., Silver, M. D., Trimble, A. S., and Adelman, A. G. Prinzmetal's variant form of angina with arteriographic evidence of coronary arterial spasm. *Am. J. Cardiol.*, 1972, *30*, 902–905.
6. Ekmekci, A., Toyoshima, H., Kwoczynski, J. K., Nagaya, T., and Prinzmetal, M. Angina pectoris. IV. Clinical and experimental difference between ischemia with S-T elevation and ischemia with S-T depression. *Am. J. Cardiol.*, 1961, *7*, 412–426.
7. Epstein, S. E., Redwood, D. R., Goldstein, R. E., Beiser, G. D., Rosing, D. R., Glancy, D. L., Reis, R. L., and Stinson, E. B. Angina pectoris: pathophysiology, evaluation and treatment. *Ann. Int. Med.*, 1971, *75*, 263–296.
8. Goodin, R. R., Inglesby, T. V., Lansing, A. M., and Wheat, M. W., Jr. Preinfarction angina pectoris: surgical emergency. *J. Thoracic & Cardiovasc. Surg.*, 1973, *66*, 934–942.
9. Guazzi, M., Fiorentini, C., Polese, A., and Magrini, F. Continuous electrocardiographic recording in Prinzmetal's variant angina pectoris: report of 4 cases. *Brit. Heart J.*, 1970, *32*, 611–616.
10. Guazzi, M., Polese, A., Fiorentini, C., Magrini, F., and Bartorelli, C. Left ventricular performance and related haemodynamic changes in Prinzmetal's variant angina pectoris. *Brit. Heart J.*, 1971, *33*, 84–94.
11. Levin, D. C., Sos, T. A., Lee, J. G., and Baltaxe, H. A. Coronary collateral circulation and distal coronary runoff: key factors in preserving myocardial contractility in patients with coronary artery disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 463–473.
12. MacAlpin, R. N., Kattus, A. A., and Alvaro, A. B. Angina pectoris at rest with preservation of exercise capacity: Prinzmetal's variant angina. *Circulation*, 1973, *47*, 946–958.
13. Oliva, P. B., Potts, D. E., and Pluss, R. G. Coronary arterial spasm in Prinzmetal angina: documentation by coronary arteriography. *New England J. Med.*, 1973, *288*, 745–751.
14. Prinzmetal, M., Ekmekci, A., Kennamer, R., Kwoczynski, J. K., Shubin, H., and Toyoshima, H. Variant form of angina pectoris: previously undelineated syndrome. *J.A.M.A.*, 1960, *174*, 1794–1800.
15. Prinzmetal, M., Kennamer, R., Merliss, R., Wada, T., and Bor, N. Angina pectoris. I. Variant form of angina pectoris. *Am. J. Med.*, 1959, *27*, 375–388.
16. Robinson, B. F. Relation of heart rate and systolic blood pressure to onset of pain in angina

pectoris. *Circulation*, 1967, *35*, 1073–1083.
17. ROBINSON, J. Prinzmetal's variant angina pectoris: report of case. *Am. Heart J.*, 1965, *70*, 797–800.
18. ROUGHGARDEN, J. W. Circulatory changes associated with spontaneous angina pectoris. *Am. J. Med.*, 1966, *41*, 947–961
19. ROUGHGARDEN, J. W., and NEWMAN, E. V. Circulatory changes during pain of angina pectoris. 1772–1965—critical review. *Am. J. Med.*, 1966, *41*, 935–946.
20. RUGGEROLI, C. W., COHN, K., and LANGSTON, M. Prinzmetal's variant angina: pathophysiological and clinical kaleidoscope. *Am. J. Cardiol.*, 1974, *33*, 167 (Abstr).
21. SILVERMAN, M. E., and FLAMM, M. D., JR. Variant angina pectoris: anatomic findings and prognostic implications. *Ann. Int. Med.*, 1971, *75*, 339–343.
22. WHITING, R. B., KLEIN, M. D., VANDER VEER, J., and LOWN, B. Variant angina pectoris. *New England J. Med.*, 1970, *282*, 709–712.

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LYMPHOCELES FOLLOWING RENAL TRANSPLANTATION*

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A LYMPHOCELE or lymphocyst is an abnormal encapsulated collection of lymph in the pelvis which occurs in the postoperative period following pelvic surgery. Transsection of the lymphatic channels during the surgical procedure has been shown to result in an outpouring of lymph into the potential or free spaces of the pelvis. Depending upon the size of this collection of fluid and its position within the pelvis, compression of the pelvic organs may occur. This entity has been well documented in the gynecologic literature[3,4,5,12] and in the surgical literature.[19,20]

Only recently have lymphoceles following renal transplantation been recognized as a potentially serious complication.[2,8,9,11,14,16,17] Koehler and Kyaw's[8] report of 4 cases and the 2 cases of Moreau *et al.*[11] have been the only descriptions of this complication of renal transplantation which have appeared in the radiologic literature. For this reason, we reviewed the renal transplant cases at this institution in order to further define the incidence and pathogenesis of this complication.

INCIDENCE

Pelvic lymphoceles following certain gynecologic procedures are common and were reported to occur with an incidence as high as 49 per cent by Mori[12] in 1955. He reported 68 lymphocysts occurring following radical pelvic lymph node dissection for carcinoma of the cervix in 140 patients. Dodd *et al.*[3] in 1970 reported that in a series of 453 patients treated by pelvic lymphadenectomy for carcinoma of the cervix, 131 developed clinically detectable lymphoceles, an incidence of 28.9 per cent. All authors concluded that the frequency of the complication was dependent upon the extent of the surgical interruption of the lymphatic channels of the pelvis. Gray and co-workers[5] found a lower incidence of 16.4 per cent in 55 pelvic lymphadenectomies.

The incidence of lymphocele formation following renal transplantation was reported as 2 per cent of 280 transplant recipients by Schweizer *et al.*[16] Starzl *et al.*[17] found 2 lymphoceles in 216 patients in which the total number of renal transplants was 234; this would be an incidence of less than 1 per cent. Other than these, only scattered cases of this complication following renal transplantation have been reported.[2,8,9,11,14]

MATERIAL

The records of 70 patients, 46 males and 24 females, receiving a total of 88 renal transplants were reviewed. Of these 70 patients, 6 male patients were found to have pelvic lymphoceles, an incidence of 8.6 per cent of the transplant population and 6.8 per cent of the total number of transplants performed. One of these 6 patients suffered loss of his transplanted kidney and another eventually died from pulmonary embolism. Both of these complications resulted from pelvic lymphoceles.

CLINICAL SIGNS AND SYMPTOMS

The clinical presentation of lymphoceles depends on their size and location. Dodd and associates[3] state that the smaller collections tend to regress spontaneously, whereas the larger collections produce more immediate symptoms and severe

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sequelae. The time interval between transplantation and clinical recognition of the lymphocele in our patients varied from 1 month to 17 months. Ipsilateral leg swelling was the most common complaint and finding. Leg pain was variably present. A feeling of fullness in the pelvis both with and without a palpable mass was also reported. Two patients presented with a decrease in renal function as their chief complaint. One patient presenting with a main complaint of leg edema subsequently developed a massive pulmonary embolus that resulted in his death. This embolus was thought to be due directly to compression of the pelvic veins by the lymphocyst with subsequent formation of a thrombus.

Other clinical findings which have been reported include acute anuria,[2] gain in weight, constipation, infection of the lymphocele and fistula formation. Later effects include obstructive uropathy with uremia and bowel obstruction.[3,8,16]

ROENTGENOLOGIC FINDINGS

Plain film roentgenograms of the abdomen will not usually demonstrate any significant abnormality. At times the fascial planes in the pelvis may be distorted by the surgical procedure. A change in the position of phleboliths which is noted on films exposed after the surgical procedure may be significant, but in general plain film roentgen findings are minimal.[3]

Intravenous pyelography is the most useful roentgenologic procedure, giving information about the function of the kidney and the status of the collecting structures and ureter, as well as showing the urinary bladder and any displacement. In addition, intravenous pyelography may permit differentiation of a lymphocele from a urinoma. Obstruction of the ureter with hydroureter and hydronephrosis may or may not be present. An extrinsic mass lesion is almost invariably noted compressing the urinary bladder, and this is the commonest and most characteristic finding (Fig. 1–5). The opacified ureters are commonly displaced medially and anteriorly. Retrograde cystography will show this compression of the urinary bladder to better advantage. In addition, extravasation of urine at the site of the ureteral anastomosis, if present, may be observed (Fig. 4, *A* and *B*).

Direct visualization by aspiration of the lymphatic fluid from the lymphocele and the instillation of a contrast agent into the emptied lymphocyst may reveal its size

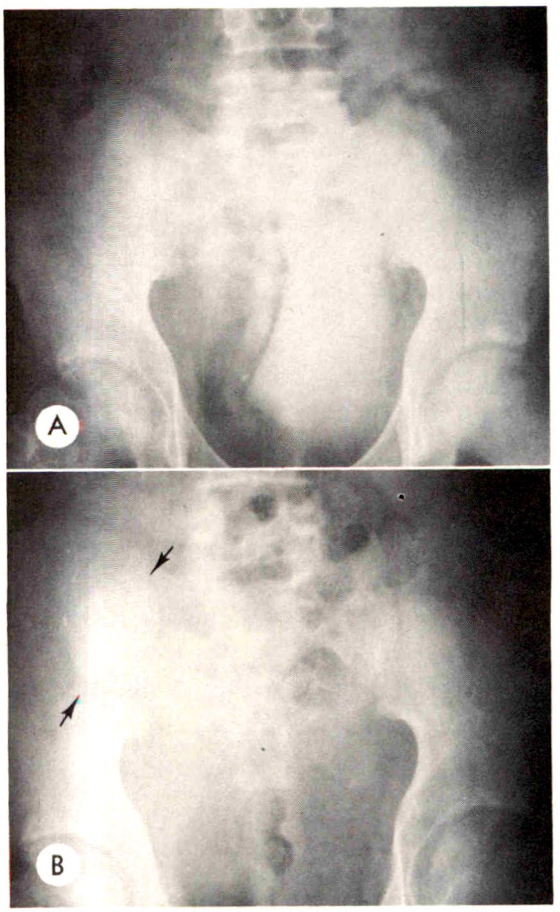


FIG. 1. Eight months after a kidney was transplanted into the right iliac fossa of this 17 year old boy he developed right leg swelling. (*A*) An excretory urogram on June 20, 1972 showed extrinsic compression on the right side of the bladder with poor visualization of the collecting structures of the transplanted kidney. (*B*) An excretory urogram on January 7, 1973 showed a poorly functioning right kidney with a grossly dilated renal pelvis and ureter (arrows). On January 10, 1973 the patient was operated upon and a large lymphocele containing 1,000 cc. of fluid was aspirated and drained externally.

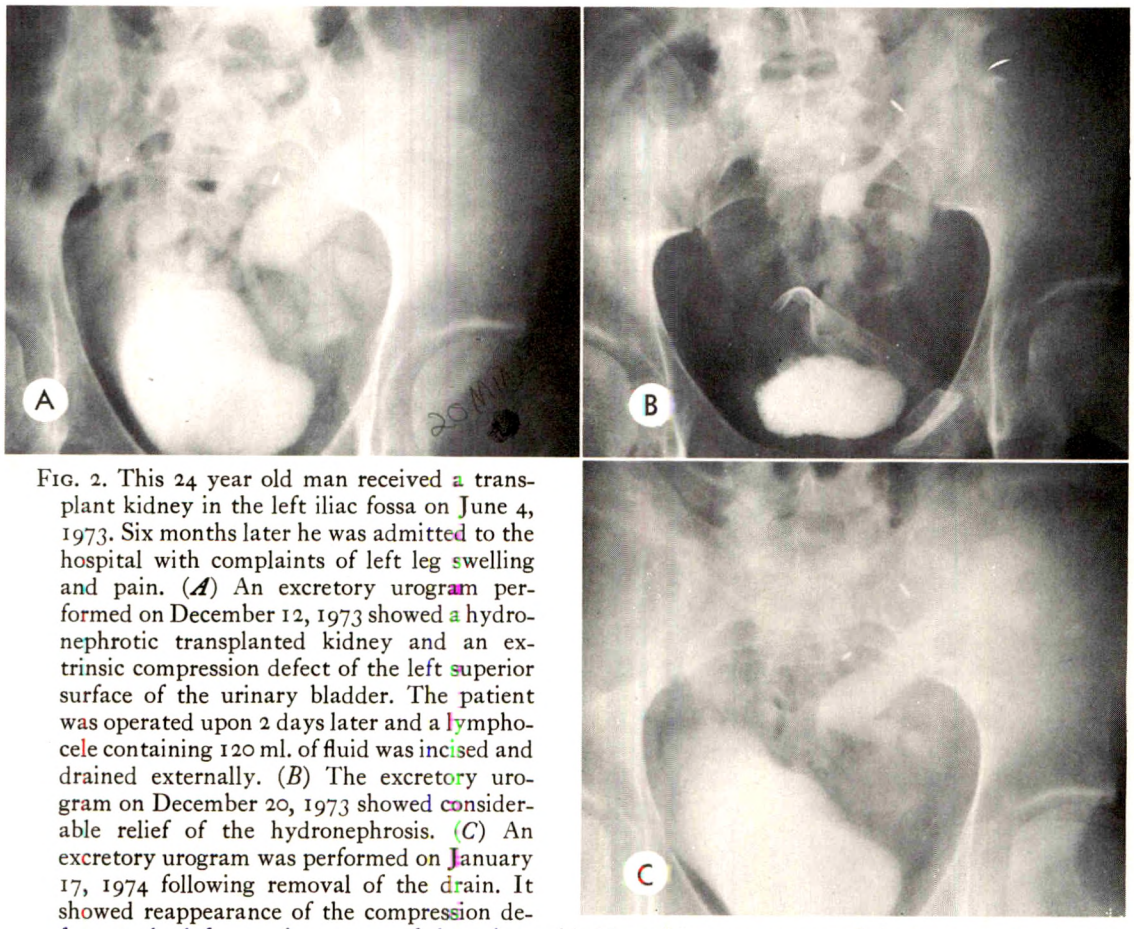


FIG. 2. This 24 year old man received a transplant kidney in the left iliac fossa on June 4, 1973. Six months later he was admitted to the hospital with complaints of left leg swelling and pain. (*A*) An excretory urogram performed on December 12, 1973 showed a hydronephrotic transplanted kidney and an extrinsic compression defect of the left superior surface of the urinary bladder. The patient was operated upon 2 days later and a lymphocele containing 120 ml. of fluid was incised and drained externally. (*B*) The excretory urogram on December 20, 1973 showed considerable relief of the hydronephrosis. (*C*) An excretory urogram was performed on January 17, 1974 following removal of the drain. It showed reappearance of the compression defect on the left superior aspect of the urinary bladder with some return of the hydronephrosis. Recurrence of the lymphocele is a likely explanation.

and position, but it is not a generally recommended procedure (Fig. 5, *A* and *B*). Infection almost invariably results following repeated aspiration.

Venography is not likely to be of much practical value. Lymphoceles do cause compression of the femoral veins and in this manner probably contribute to the edema which is reported to occur in the ipsilateral lower extremity and to stasis of blood and subsequent thrombus formation. Venography may demonstrate the compression and may also demonstrate the clots but would not be useful in determining the etiology.

Lymphangiography is the most positive method of demonstrating lymphoceles but will rarely be needed since the urographic findings are so characteristic.

DISCUSSION

Many lymphatic vessels are transsected during renal transplantation, particularly in the region of the external iliac and hypogastric vessels when the areolar tissue surrounding these vessels is excised. An attempt is made during the surgical procedure to ligate all of the major trunks of the lymphatic vessels which have been transsected. If major trunks are not ligated or fail to contract, lymph from the transsected distal trunks will continue to flow into potential pelvic spaces.[8]

McMaster[10] has defined in more detail

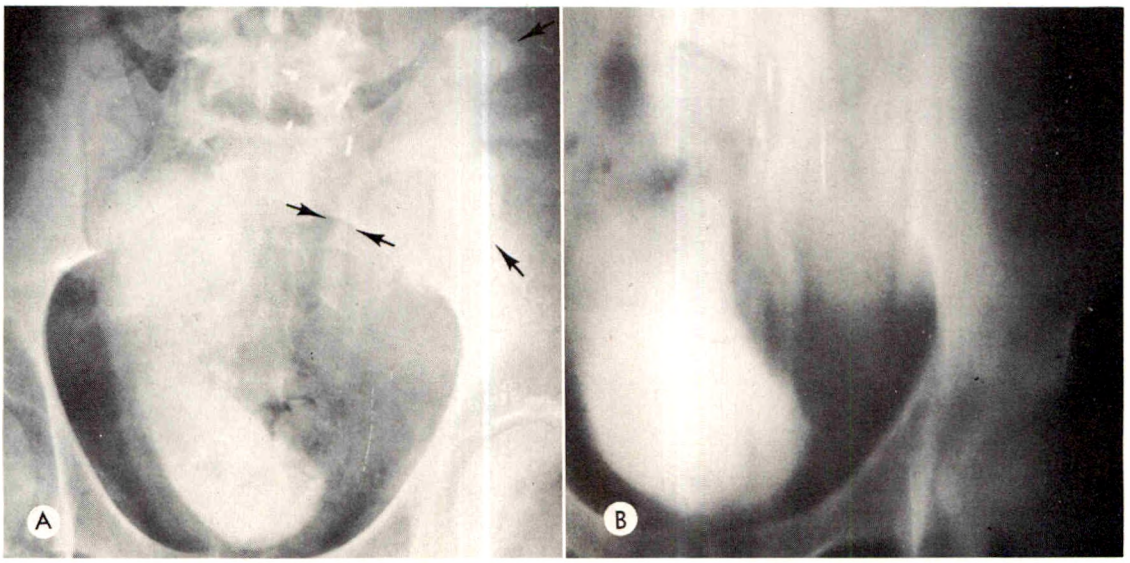


FIG. 3. A kidney was transplanted into the left iliac fossa of this 27 year old man on May 12, 1973. Two months later left leg edema and a pelvic mass were found. (*A*) Excretory urography on August 2, 1973 showed dilatation of the calyces and ureter of the transplanted kidney (arrows). An extrinsic mass compressed the bladder on its left superior aspect. (*B*) A repeat urogram with body section roentgenography 3 days later better demonstrated the defect on the bladder. The patient was operated upon and a lymphocele containing 300 ml. of fluid was incised and drained. The patient suffered massive pulmonary embolism secondary to left saphenous vein, femoral and pelvic vein thrombosis, doubtless related to stasis induced by compression from the lymphocele. He died on August 22, 1973.

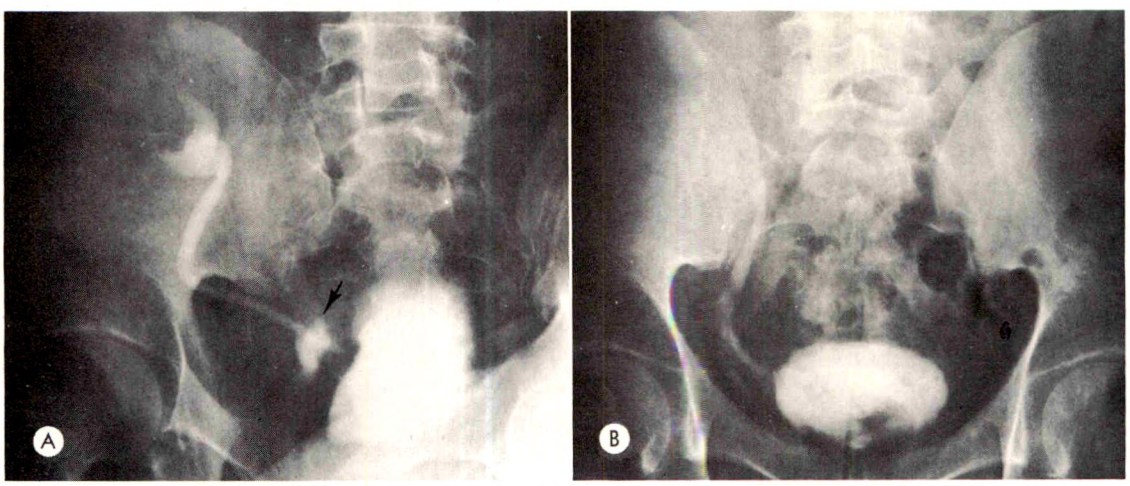


FIG. 4. Two months following transplantation of a kidney into the right iliac fossa of this 28 year old man, leg edema developed, and a pelvic mass was palpable on the right. (*A*) An excretory urogram performed on August 31, 1973 demonstrated a mass deforming and compressing the urinary bladder on the right and displacing it slightly toward the left. A lymphocele was suspected, but because of a small amount of extravasation of contrast material from the distal part of the transplanted ureter (arrow), a urinoma was also thought to be possibly responsible. The patient was operated upon and a small urinoma was found, but the compression of the urinary bladder was produced by a lymphocele which contained 200 ml. of fluid. It was incised and drained. (*B*) An excretory urogram on September 21, 1973 shows return of the bladder to a normal appearance.

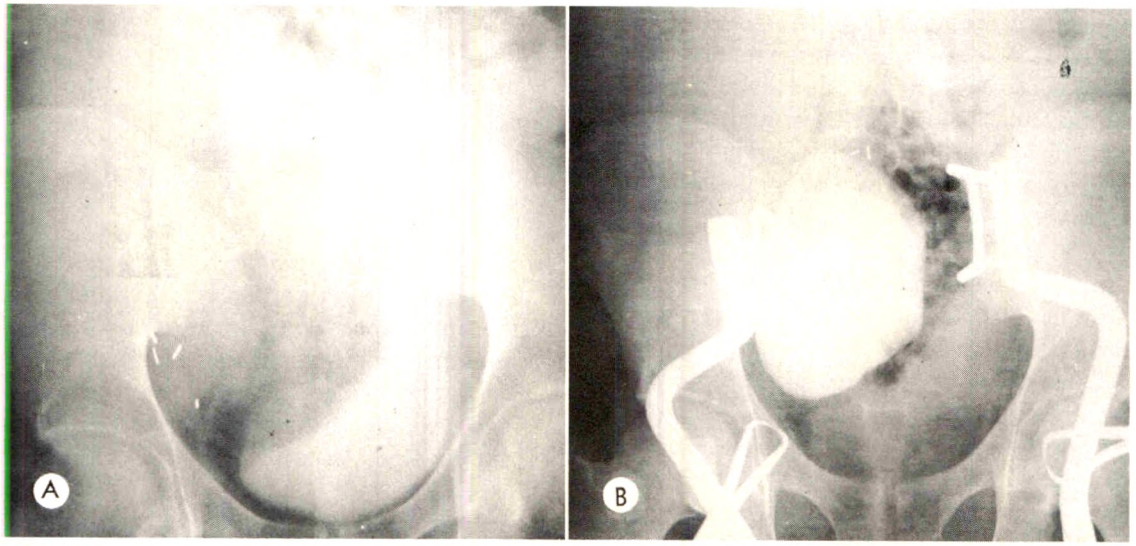


Fig. 5. Renal transplantation was carried out in the right iliac fossa of this 19 year old man on September 13, 1972. An excretory urogram on October 18, 1972 showed a normally functioning transplanted kidney and a normal urinary bladder. (*A*) An excretory urogram on January 21, 1974, 17 months after transplantation, revealed an enlarged right kidney and a large mass impinging on the right superior aspect of the urinary bladder. The patient was operated upon on February 6, 1974 and a 500 ml. lymph-filled cyst was aspirated. (*B*) The lymphocele was injected with contrast material at the time of the operation, displaying its borders, size and position. The lymphocele was treated by marsupialization of the cyst cavity with intraperitoneal drainage.

the occurrences following laceration of lymphatic vessels in experimental animals. Following transsection and the outpouring of lymph, the most proximal end of the lymphatic channels initially remain open and carry lymphatic fluid away from the wound. However, this process is hindered to a certain degree by the presence of fibrin clots within the wound. The lymphatic channels become completely occluded within 48 hours. At this point, two factors determine whether or not the lymphatic fluid will accumulate within the wound: availability of collateral channels and the capability of the lymphatic vessels to regenerate. McMaster[10] was able to demonstrate new lymph vessel formation within 7 to 10 days following injury.

Dodd and associates[3] state that if a long segment of a lymphatic channel is removed, regeneration will not occur, the removal of accumulated lymphatic fluid then being dependent upon the adequacy of the collateral circulation. Pelvic lymphadenectomy is a much more extensive procedure than renal transplantation, and much more lymphatic tissue is removed. Thus it is not surprising that the incidence of lymphocele formation is greater following pelvic lymphadenectomy than after renal transplantation.

In our series of patients, the most common presentation was that of ipsilateral leg swelling occurring in 5 patients, the result of compression of the iliac vessels by the lymphocele. Compression of the iliac veins most likely led to the death of 1 patient because of the subsequent development of thrombophlebitis and pulmonary embolism. Three of our patients complained of a pressure sensation in the pelvis and pain in the ipsilateral leg. One patient presented with decreased renal function.

Diagnosis of lymphocele is best made by excretory urography, which shows the characteristic compression of the urinary bladder and perhaps dilatation and obstruction of the ureter. Compression of the bladder

and displacement of the ureter were noted in all of our patients. The differential diagnosis should include abscess, pelvic hematoma, urinoma, thrombophlebitis and cystitis. This differentiation can usually be readily ascertained by utilizing the clinical and laboratory findings and excretory urography. Other procedures including cystography, femoral venography and ultrasound may be utilized. Lymphangiography will rarely be necessary.

The problems of prevention and treatment of lymphoceles are quite complex. Although ligation of the severed lymphatic channels during the surgical procedure has been suggested as being of utmost importance, it is difficult to demonstrate objectively a lower incidence of lymphocele formation when ligation is done than when it is not.

The treatment of lymphoceles that have already developed has varied. Simple needle aspiration has been attempted with little success. The lymphoceles tend to recur quite rapidly, and, in addition, after repeated aspirations, infection usually supervenes.

External drainage appears to be the initial procedure of choice. Drains can be inserted quickly and have resulted in some cures. Drainage was of benefit in 4 of our 6 patients with lymphoceles. If simple drainage of the cyst is not curative, than marsupialization with drainage into the peritoneal cavity is the next procedure of choice.

Other procedures have included localization of the draining lymph fistula by lymphangiography and then, aided by a colored intralymphatic infusion at the time of wound exploration, direct ligation of the open lymphatic vessel. This technique was first used by Strode and Grininger.[19] Madura *et al.*[9] have suggested that a long trial of conservative management may be indicated, consisting of bed rest, extremity elevation and compression dressings to keep the superficial lymphatic system empty. It is well known that patients may become asymptomatic during hospitalization and bed rest, but following discharge from the hospital and progressive ambulation, the lymphocele may recur.

CONCLUSION AND SUMMARY

The symptom complex of ipsilateral leg swelling, leg pain, pelvic fullness and decreased renal function in a renal transplant recipient coupled with roentgenographic demonstration of an extrinsic mass compressing the urinary bladder and perhaps obstructing or displacing the transplant kidney ureter, is almost diagnostic of a lymphocele.

Impaired renal function, thrombophlebitis and subsequent pulmonary embolism are severe complications of lymphoceles. The treatment of choice appears to be external drainage for primary lymphoceles and intraperitoneal marsupialization for recurrent lymphoceles.

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REFERENCES

1. BYRON, R. C., JR., YONEMOTO, R. H., DAVAJAN, V. *et al.* Lymphocysts: surgical correction and prevention. *Am. J. Obst. & Gynec.*, 1966, *94*, 203–207.
2. DIETHELM, A. G. Anuria secondary to perirenal lymphocele. *South. M. J.*, 1972, *65*, 350–353.
3. DODD, G. D., RUTLEDGE, F., and WALLACE, S. Postoperative pelvic lymphocysts. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 312–323.
4. FERGUSON, J. H., and MAC CLURE, J. G. Lymphocele following lymphadenectomy. *Am. J. Obst. & Gynec.*, 1961, *82*, 783–792.
5. GRAY, M. J., PLENTL, A. A., and TAYLOR, H. C., JR. Lymphocyst: complication of pelvic lymph node dissections. *Am. J. Obst. & Gynec.*, 1958, *75*, 1059–1062.
6. HILL, R. B., JR., DAHRLING, B. E., STARZL, T. E., and RIFKIND, D. Death after transplantation: analysis of sixty cases. *Am. J. Med.*, 1967, *42*, 327–334.
7. INOCENCIO, H. F., PIERCE, J. M., ROSENBERG, J. C., ROSENBERG, B. F., WOLF, P. L., SMALL, M. P., and ING, T. S. Renal allograft with

massive perirenal accumulation of lymph. *Brit. M. J.*, 1969, *3*, 452–453.

8. Koehler, P. R., and Kyaw, M. M. Lymphatic complications following renal transplantations. *Radiology*, 1972, *102*, 539–543.
9. Madura, J. A., Dunbar, J. D., and Cerilli, G. J. Perirenal lymphocele as complication of renal homotransplantation. *Surgery*, 1970, *68*, 310–313.
10. McMaster, P. F. Lymphatic participation in cutaneous phenomena. *The Harvey Lectures*, 1941–42, *37*, 227–268.
11. Moreau, J. F., Leski, M., Beurton, D., Cokier, J., Michel, J. R., Kreis, H., and Lacombe, M. Lymphoceles obstructives après transplantation rénale. *Ann. radiol.*, 1973, *16*, 471–480.
12. Mori, N. Clinical and experimental studies on so-called lymphocyst which develops after radical hysterectomy in cancer of uterine cervix. *J. Jap. Obst. & Gynec. Soc.*, 1955, *2*, 178–203.
13. Parker, J. J., and Schmutzer, K. J. Chylous lymphocyst. *Radiology*, 1971, *98*, 569–670.
14. Pillay, V. K. G., Ing, T. S., Armbruster, K. W., Pierce, J. M., Jr., and Rosenberg, J. C. Lymphocyst following renal transplantation. *Canad. M. A. J.*, 1971, *105*, 1066–1069.
15. Prieto, A. E., Gonzalez, G., Templeton, A. W., Cooper, B. R., and Palacios, E. Lymphatic channel obstruction. *Radiology*, 1971, *113*, 366–375.
16. Schweizer, R. T., Lang-In-Cho, Kountz, S. L., and Belzer, F. O. Lymphoceles following renal transplantation. *A.M.A. Arch. Surg.*, 1972, *104*, 342–345.
17. Starzl, T. E., Groth, C. G., Putnam, C. W., Penn, I., Halgrimson, C. G., Flatmark, A., Gecelter, L., Brettschneider, L., and Stonington, O. G. Urological complication in 216 human recipients of renal transplants. *Ann. Surg.*, 1970, *172*, 1–22.
18. Steinberg, A. O., Madayag, M. A., Bosniak, M. A., and Morales, P. A. Demonstration of two unusually large pelvic lymphocysts by lymphangiography. *J. Urol.*, 1973, *109*, 477–478.
19. Strode, J. E., and Grininger, D. R. Lymphatic fistula of lower extremity: lymphangiography as aid in diagnosis and treatment. *Am. J. Surg.*, 1968, *116*, 135–136.
20. Weingold, A. B., Olivo, E., and Marino, J. Pelvic lymphocyst: diagnosis and management. *A.M.A. Arch. Surg.*, 1967, *95*, 304–307.

ANGIOGRAPHIC FINDINGS IN BRONCHIAL ADENOMA*

(CARCINOID TYPE)

By KAMAL MOJAB, M.D., WALTER BARKER, M.D., JUSTO RODRIGUEZ, M.D., *and* MARCUS RAMOS, M.D.
CHICAGO, ILLINOIS

BRONCHIAL adenomas comprise 2 to 4 per cent of all primary pulmonary neoplasms. The tumors usually occur in patients between the ages of 20 and 40 years and have no apparent predilection for males. Most of these lesions are found in and about the main bronchi. Because of their significant vascularity, such adenomas may be differentiated from avascular lesions angiographically. Carcinoid adenomas are usually supplied by enlarged bronchial arteries which may appear aberrant: as such, they can be confused with pulmonary sequestrations. Although pulmonary sequestration has an aberrant systemic arterial blood supply, it usually arises from the intrathoracic or intra-abdominal aorta and not from bronchial arborization.

REPORT OF A CASE

A 27 year old Black male was admitted to the Thoracic Surgical Service at Cook County Hospital because of a recurrent history of cough, expectoration of mucopurulent sputum, brisk hemoptyses and low-grade fever. Physical examination was within normal limits, except for scattered rhonchi on the right. Sputum cytology was negative for malignant cells and culture failed to yield any significant pathology.

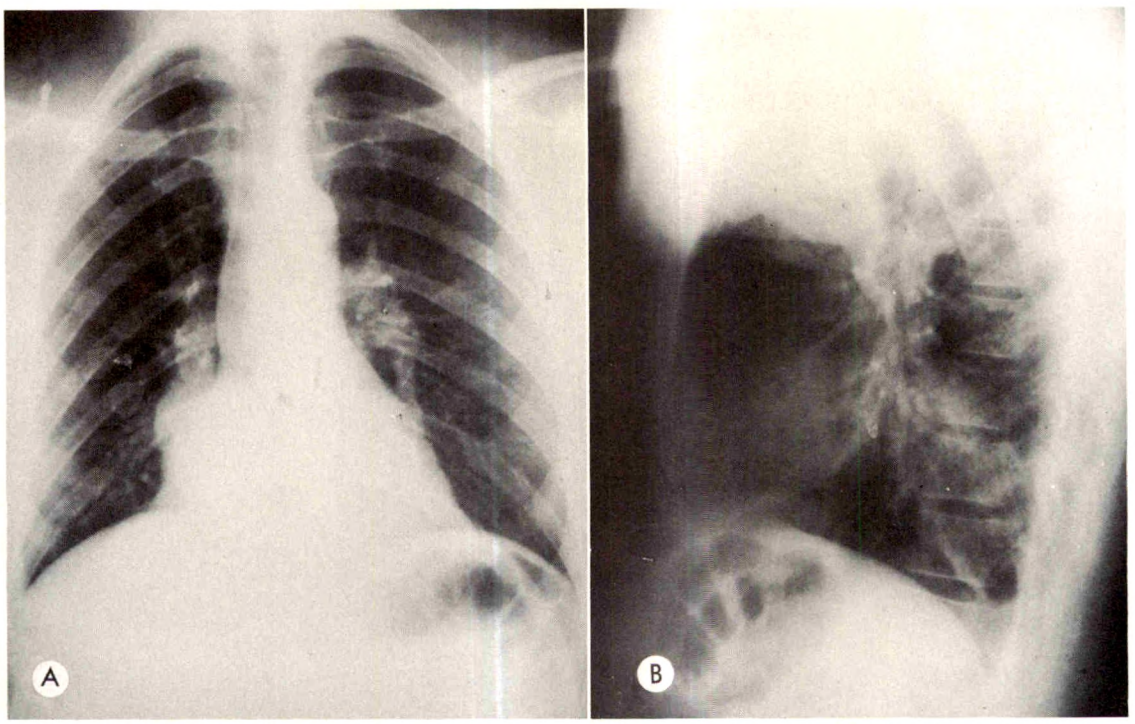


FIG. 1. (*A*) Posteroanterior and (*B*) lateral roentgenograms of the chest reveal a homogeneous mass located in the posterior basal segment of the right lower lobe.

* From the Department of Diagnostic Radiology and Thoracic Surgery, Cook County Hospital, Chicago, Illinois.

Posteroanterior and lateral chest roentgenograms revealed a homogeneous mass located in the posterior basal segment of the right lower lobe (Fig. 1, *A* and *B*). Tomogram revealed the well-delineated non-calcified nature of the lesion and, in addition, indicated a bronchial cut-off at the distal bronchus intermedius (Fig. 2). Bronchograms confirmed the obstruction of the bronchus (Fig. 3, *A* and *B*). Bronchoscopy was performed and a smooth, tan, rounded obstructing mass located at the distal end of the bronchus intermedius was observed. On the basis of the patient's history and plain chest roentgenograms, the diagnosis of pulmonary sequestration was considered.

Aortic angiography was performed which revealed a relatively large artery exiting from the right side of the aorta at the level of T-5 (Fig. 4). Selective catheterization of the bronchial artery was performed and, as seen in Figure 5, it extends through the lower lung to supply a highly vascular mass. Abnormal beaded vessels distal to the tumor, due to pneumonitis and delineation of the lesion, are best seen in the subtraction films (Fig. 6, *A* and *B*). Exploration revealed a large 3×4 cm. mass located in the posterior basal segment of the right lower lobe. Right lower lobectomy and mediastinal dissection were performed. Histopathology revealed a bronchial adenoma of the carcinoid type and pneumonitis distal to the lesion (Fig. 7, *A* and *B*; and 8, *A* and *B*). Hilar and mediastinal lymph nodes were negative for metastatic involvement.



FIG. 2. Tomogram of the right chest reveals the well delineated noncalcified nature of the lesion and obstruction of the corresponding bronchus.

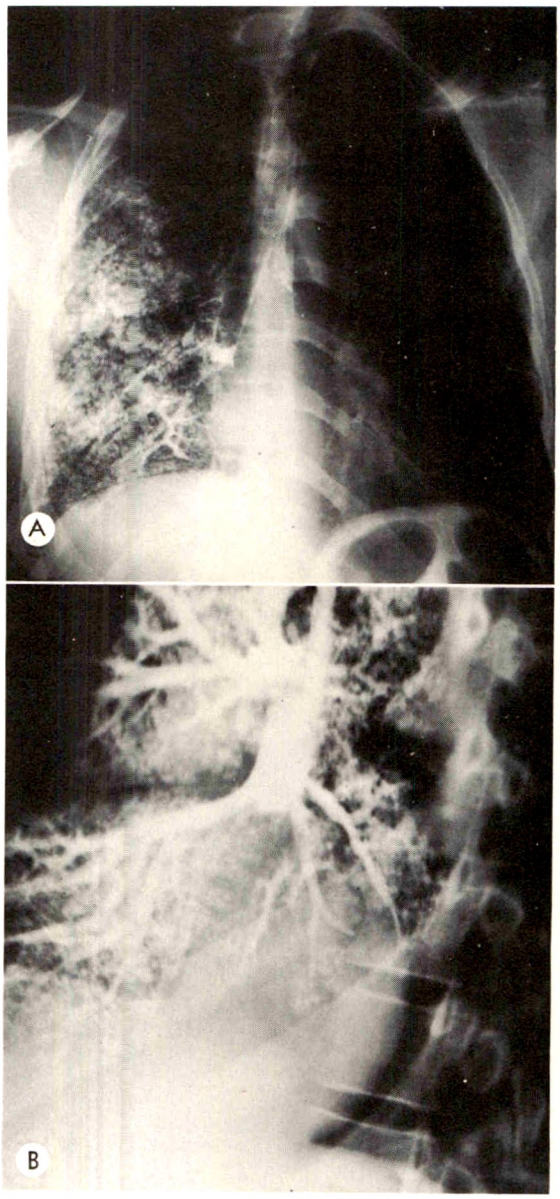


FIG. 3 (*A* and *B*) Bronchograms confirm the obstruction of the bronchus.

DISCUSSION

Bronchial adenomas arise from mucous glands and are usually located centrally,

FIG. 6. (*A* and *B*) Subtraction films demonstrate normal vessels with delineation of the wall. The beaded vessels distal to the tumor are due to pneumonitis.

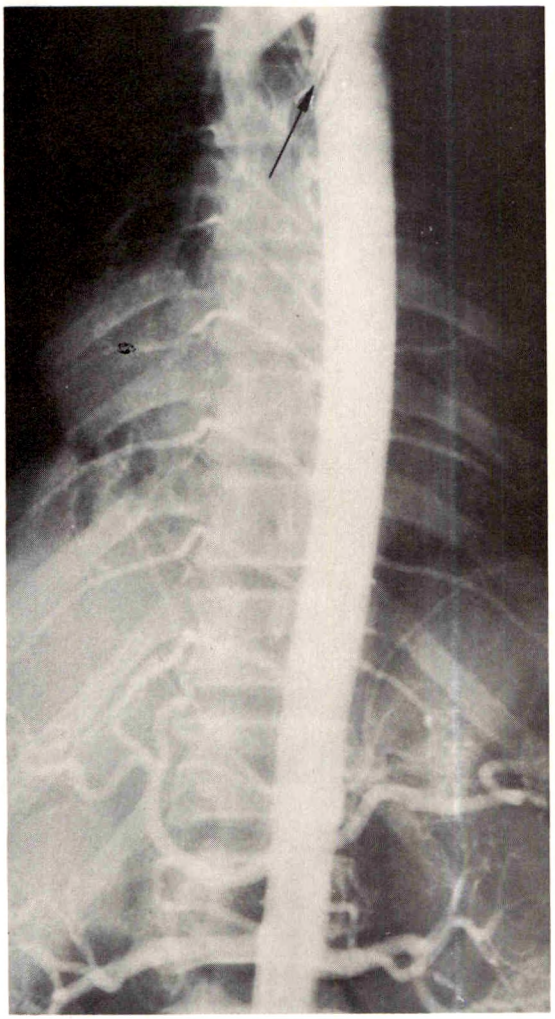


FIG. 4. Aortic angiogram demonstrates a relatively large bronchial artery exiting from the right side of the descending aorta at the level of T-5.

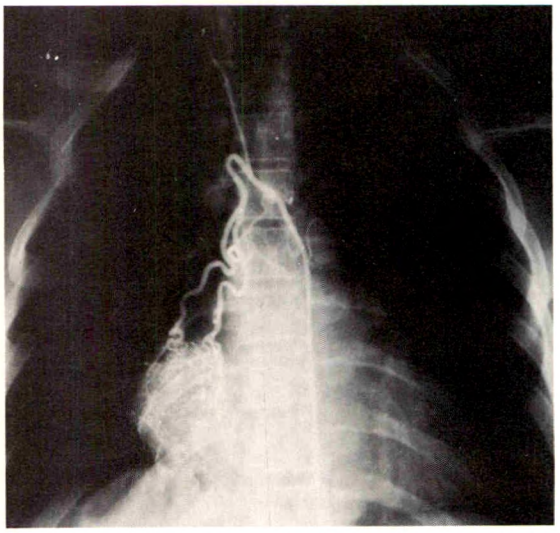


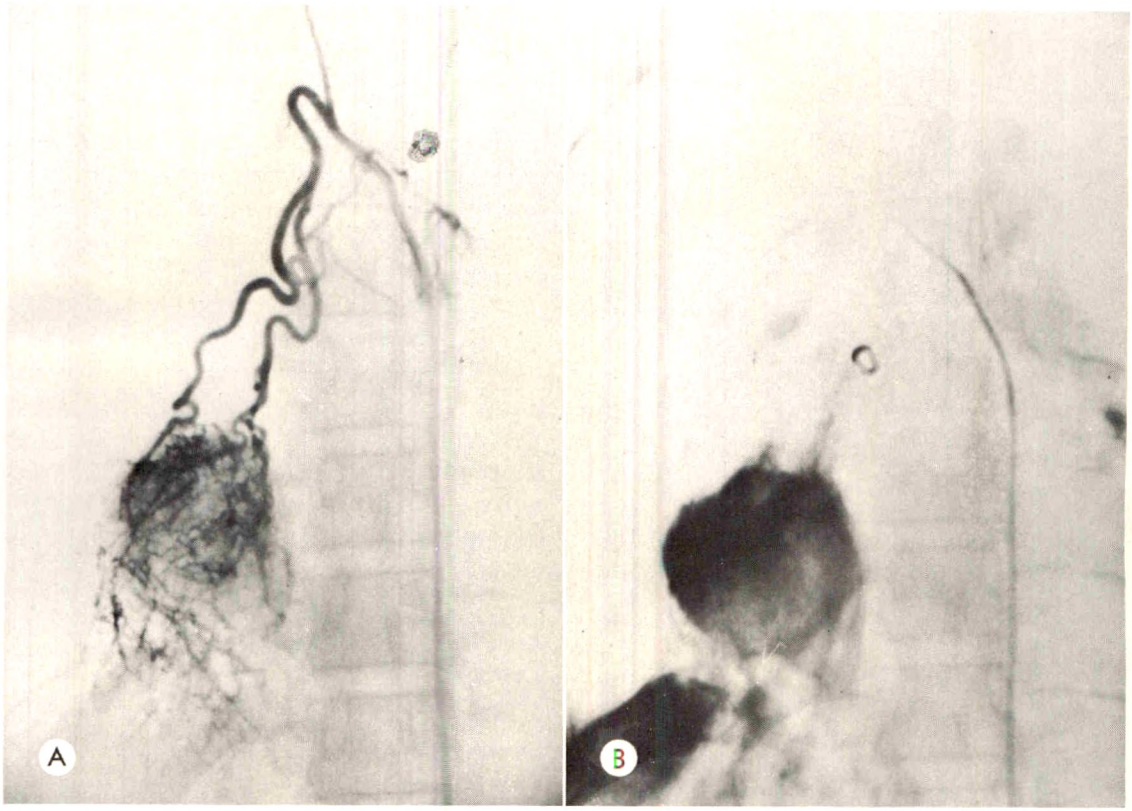
FIG. 5. Selective catheterization angiogram of the bronchial artery reveals extension of this bifid artery to the mass and surrounding parenchyma.

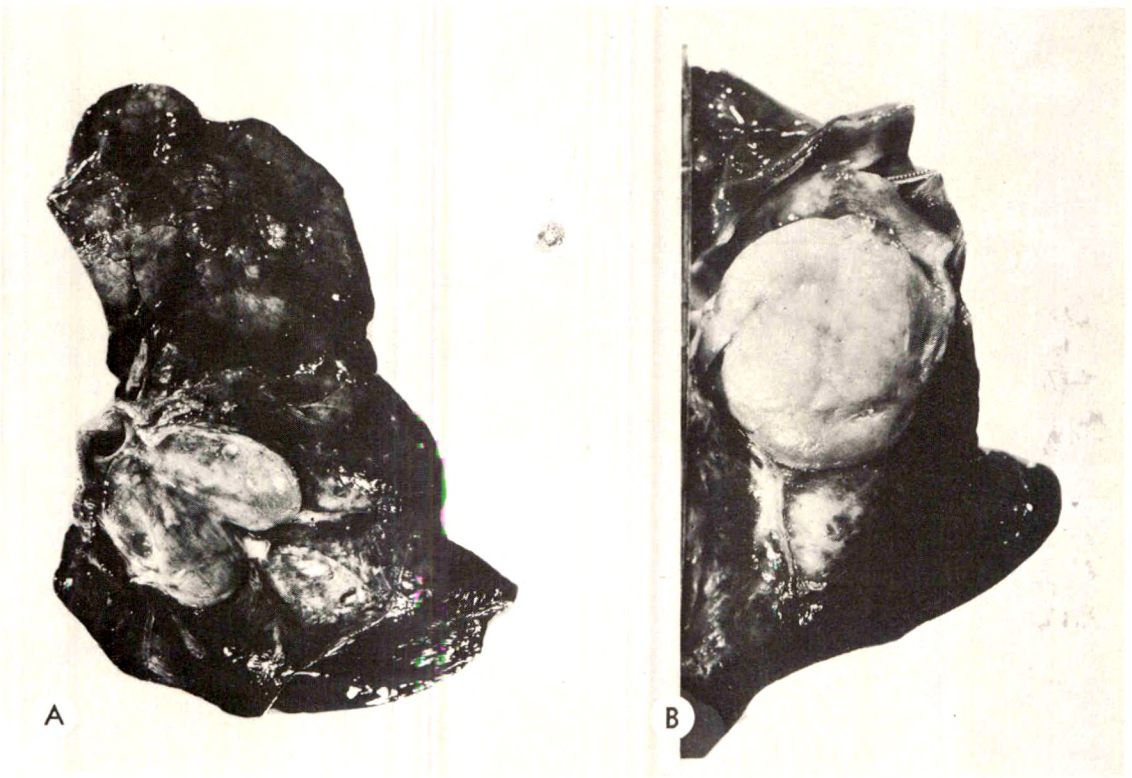
causing partial to complete bronchial obstruction. About 20 per cent of these tumors arise peripherally and are not associated with bronchial obstruction. The roentgenographic finding of this type of adenoma may appear as a solitary pulmonary nodule.

There are 3 types of bronchial adenoma: the carcinoid type; cylindroma (adenoid cystic carcinoma); and mucoepidermoid tumor.[4] Most of these lesions are found in and about the main bronchi. The carcinoid type frequently protrudes into the bronchial lumen which can produce partial or complete obstruction of the bronchus. The cylindroma and mucoepidermoid tumors tend to occur more proximally than the carcinoid type and are more likely to extend along the bronchial wall. The intrabronchial portion of the tumor frequently ulcerates producing hemoptyses.

Unlike the intestinal carcinoid, carcinoid syndrome is very rare in carcinoid of the lung. Biopsy is an important diagnostic measure, although it may result in signifi-

FIG. 7. (*A*) Right middle and lower lobes, with ovoid mass arising from the walls of their secondary bronchi, bulging into their lumens, and extending into the parenchyma. (*B*) Cut surface of the mass, which was homogeneously tan and showed some calcific stippling.


A
B


A
B

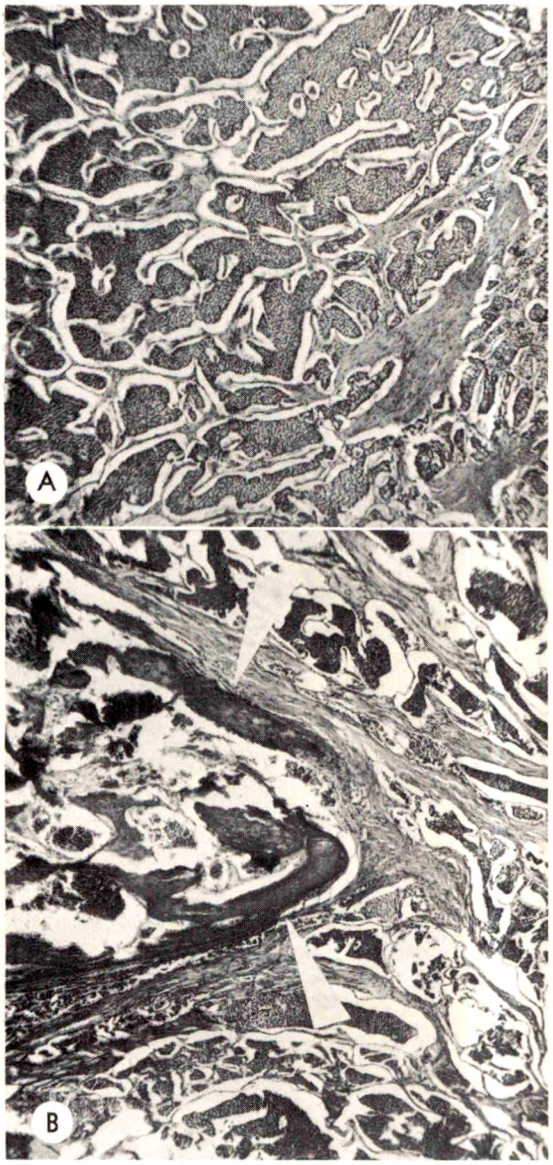


FIG. 8. (*A*) Focal ossification (arrows) within the tumor mass. (*B*) Characteristic pattern composed of well delineated nests of uniform cells.

cant hemorrhage. Treatment is generally surgical removal, since the tumor is not radiosensitive.

Bronchial adenomas are tumors of low-grade malignancy which may eventually metastasize. McBurney *et al.*[3] reported that about 10 per cent of all bronchial adenomas metastasize distantly. It is generally conceded that cylindromas metastasize more readily than carcinoid adenomas and that the mucoepidermoid types are the least malignant.

SUMMARY

A case of carcinoid bronchial adenoma treated by lobectomy is reported.

The diagnostic roentgenologic features are presented and the angiographic characteristics are discussed in relation to the differentiation of this entity from other pulmonary lesions.

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REFERENCES

1. DONAHUE, J. L., WEICHERT, R. F., and OCHSULS, J. L. Bronchial adenoma. *Ann. Surg.*, 1968, *167*, 873–885.
2. GIBBONS, J. H., SABESTON, D. C., and SPENCER, F. C. Surgery of the Chest. W. B. Saunders Company, Philadelphia, 1969.
3. MCBURNEY, R. P., KIRKLIN, J. W., and WOOLNER, L. Metastasizing bronchial adenoma. *Surg., Gynec. & Obst.*, 1953, *96*, 482–492.
4. PAYNE, W. S., ELLIS, F. H., JR., WOOLNER, L. B., and MOERSCH, H. J. Surgical treatment of cylindroma (adenoid cystic carcinoma) and muco-epidermoid tumors of bronchus. *J. Thoracic & Cardiovasc. Surg.*, 1959, *38*, 709–726.

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Fifty-seventh Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

EDITORIALS

SEVENTY-FIFTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-fifth Annual Meeting of the American Roentgen Ray Society was held at the San Francisco Hilton Hotel, San Francisco, California, from Tuesday, September 24 through Friday, September 27, 1974.

This is the second time that the Society met in "Everybody's Favorite City," a legendary place beside a beautiful bay, with exquisite towers, such as the 853 foot Pyramid, gracing its sparkling skyline and pastel sun-bathed houses commanding its magically restful hills. The first—very successful—Annual Meeting in San Francisco was held in 1966, with Dr. Clyde A. Stevenson of Spokane, Washington, as the President.

At 8:30 A.M. Tuesday, President William B. Seaman of New York called the meeting to order and in a brief ceremony he installed Colin B. Holman as the new President of the Society.

Incoming President Holman, as his first function, remitted to outgoing President Seaman the Past Presidents' Book, inscribed with his name and containing the photographs in color of the Pfahler and Erskine gavels, and a list of all Past Presidents.

President Holman then gave a timely and most appropriate Inaugural Address on "Nothing endures but change," the bit of wisdom attributed to ancient Heraclitus and quoted many times. "This truism implies," said President Holman "that at least one of the qualifications for continuing success should be the ability to change." Then he went on to narrate the historic changes throughout the *75 years* of the American Roentgen Ray Society, which were made in an "orderly and evolutionary manner, to best serve the needs of patient care and scientific advancement through organized radiology." A second group of major changes concerns the concept of "images in the technical sense," and also includes the need to consider changes in the Annual Meeting, Society Membership, Requirement of Radiologists, and Changes on the National Level which were detailed and are published *in extenso* in the leading article of this issue of the JOURNAL.

President Holman concluded his splendid Inaugural Address: "The opportunity to consider, debate, and evaluate these proposals, the changes that have already been implemented in the format of the Annual Meeting, and, most significantly, the continued superiority of our official JOURNAL through a dedicated editorial staff serve to verify the vitality of this *venerable organization* on its *Diamond Anniversary*."

This year's format of the Annual Meeting differed from that of former Annual Meetings in several respects:

(1) For the first time in the history of the Society a "registration fee" was reluctantly charged to hold down the continuously increasing expense in the arrangement of the meeting.

(2) For the same reason, the Annual Banquet, which year after year was the highlight of the social activities, has been replaced by a General Reception to all registrants; this was held on Thursday evening.

(3) The number of copyrighted papers accepted by the Program Committee has

been increased from an average of 44 to 66. This necessitated the arrangement of 2 parallel sectional meetings on Wednesday and Friday.

(4) For the second time the Caldwell Lecture was given on Wednesday at 11:30 A.M. instead of Tuesday evening.

(5) For the second time a Section for Therapeutic Radiology papers was established.

(6) The invitation of Award papers from the various contributing Specialty Societies was maintained. This year the Society for Pediatric Radiology (Tuesday), the American Society of Neuroradiology (Wednesday), and the Society of Gastrointestinal Radiology (Thursday), contributed valuable papers, of interest to all radiologists.

On Wednesday, there were 2 Parallel Sections: one a Diagnostic Program, and a second a Radiation Therapy Program.

At 11:30 A.M. the 2 Sections merged for the Caldwell Lecture.

This year's Caldwell Lecturer was Collin S. MacCarty, M.D., Professor and Chairman, Department of Neurologic Surgery, Mayo Clinic, Rochester, Minnesota, and the title of his Lecture was "A Significant Symbiotic Relationship."

Dr. MacCarty was introduced by C. Allen Good, M.D., former Professor and Chairman, Department of Diagnostic Radiology, Mayo Clinic, and presently Secretary of the American Board of Radiology.

There was an overflow attendance at this brilliant Lecture of utmost timely interest, which was published in the November issue of the JOURNAL.

At the conclusion of the Lecture, President Holman awarded the Caldwell Gold Medal to Dr. MacCarty, the Society's highest award for meritorious scientific achievement.

The second parallel Section Meeting was held Friday morning. This was necessary to accommodate diagnostic papers presented during the first half of the meeting. The sessions were in adjacent facilities so that one could pass from the one to the other between presentations.

On Wednesday afternoon, 1:30–2:45 P.M., The American College of Radiology, with Paul A. Riemenschneider, M.D., President of ACR as Moderator, conducted a most interesting session on the following topics: The Feds and You, by John W. Travis, M.D.; Our Equipment Problems by Albert S. Dunn, M.D.; People Programs or Regulations?, by Reynold F. Brown, M.D.; and Recertification ABR-ACR Liaison, by Robert E. Wise, M.D.

The Scientific Exhibits were numerically by far the largest in the Society's history (116 Exhibits); qualitatively they were most outstanding, faithfully reflecting the tremendous progress that has been made recently in the science of Radiology. Dr. James Franklin Martin, enthusiastic Chairman, and his untiring Scientific Exhibits Committee excelled all previous standards in assembling these splendid exhibits, the great success of which will be difficult to surpass in the future. A detailed description of the Scientific Exhibits is given in the following pages of this issue of the JOURNAL.

The Technical Exhibits, commensurate with the difficulties of our time, were less numerous. They displayed, however, many important contributions to the dynamic evolution of radiology. They were assembled by Dr. George A. Kling, under the guidance of Dr. James C. Cook, Consultant, and Mr. Clifford L. Sherratt, Honorary President of the Society. This issue of the JOURNAL also gives an account of these exhibits with a detailed list of the exhibiting firms.

The Committee on Local Arrangements under the enthusiastic Chairmanship of Dr. Tom M. Fullenlove prepared splendid local activities.

On Monday, preceding the meeting, the Annual Golf Tournament was held at the famous Olympic Club. On a beautiful course and with sunny skies, shotgun competition was held for the Willis F. Manges Trophy awarded for the low net score and

the Exhibitors' Trophy for the low gross score. This was followed by cocktails and an excellent late luncheon. There was also a tennis tournament at the Golden Gate Club for tennis members.

The Ladies' Program, under the equally enthusiastic Chairmanship of Mrs. Tom M. Fullenlove and Co-Chairmanship of Mrs. John C. Bennett and their able Committee was most attractive. It was as follows:

1. *Monday*, September 23, 9:00–4:00 P.M. Wine Country Tour and Luncheon. Drive across the Golden Gate Bridge to the Napa Valley and the splendid privately-owned vineyards that dot the countryside. There was a wine tasting at 2 of the famous wineries: Inglenook, Christian Brothers, or Charles Krug. A specially-catered luncheon was served in the Vineyard Room of the Mondavi Winery. After luncheon, there was a ride up the Napa Valley to browse and shop at Vintage 1870, an old winery which has been remodeled to house a complex of colorful shops, art gallery and chutney kitchen. A stop at the Hurd Candle Factory completed the day in the wine country.
2. *Tuesday*, September 24, 9:30 A.M.–1:30 P.M. Cosmopolitan City Tour. The breath-taking panorama from some of San Francisco's Seven Hills was seen. Beautiful residential areas and Consulate homes, new buildings (some controversial), old homes (some splendidly Victorian), back alleys and little known roads, all part of the City, were viewed. There was time to see the contrast from the wooded Presidio and old Fort Point to the bustling Embarcadero and modern architecture in the Financial Center.
3. *Wednesday*, September 25, 9:30 A.M.–3:30 P.M. Sausalito-Tiburon Adventure. Delightful ride on the ferry boat across San Francisco Bay, past Alcatraz Island, Angel Island and a spectacular view of the Golden Gate and the beautiful skyline of the City. After luncheon, time for shopping in the boutiques and craft shops along the waterfront was available, with return by bus over the Golden Gate Bridge.
4. *Thursday*, September 26, 10:00 A.M.–12 noon; and 1:30 P.M.–2:30 P.M. A truly unique opportunity for the visitor to San Francisco. An authentic Chinese grocery store, banks, herb shops, newspaper printing, a fortune cookie factory, temples and shrines were part of this tour.

The Press Relations were conducted with skill and good judgement by Dr. Alexander Margulis and Dr. Glenn Sheline, with the expert assistance of Mr. Otha W. Linton, Director of Public Relations of the American College of Radiology.

To the Management and Staff of the San Francisco Hilton Hotel and the San Francisco Convention Bureau, the Society expresses its sincerest thanks for the many courtesies rendered under the difficult circumstances of our time.

President Holman deserves the distinct admiration and unbounded thanks of the Society for rendering the *Diamond Anniversary* an important milestone in the history of this *venerable organization*.

TRAIAN LEUCUTIA, M.D.

THE APEX PULMONIS

THE apex of the lung has come of radiologic age: its aberrations need no longer be confined to "thickened pleura" or "superior sulcus tumor," but should include a variety of normal and morbid states. To understand these states, a detailed knowledge of its anatomy is needed.

As a practicing anatomist and as custodian of purity in anatomic nomenclature, the radiologist should consider the apex of the lung the cephalic portion bounded by the first costal arch.[1] Relying on the findings in the dissecting room to the exclusion of findings given by the roentgen method will lead to grave error in understanding the apex.[2]

The apex receives no support from the intercostal musculature, but is incompletely invested by the scalene muscles anteriorly and laterally. Posteriorly, it rises to the height of, and is protected by, the head and neck of the first rib. Anatomists refer to Sibson's fascia, an inconstant aponeurotic layer with an inconstant muscle complement, deep to the parietal pleura of the apex, giving the apex further support. (The only pulmonary hernia I have personally encountered was in the apex.)

Anteriorly, the subclavian artery courses over the apex making a groove from its retropleural position. The subclavian vein follows its artery, but makes no groove. (Hence, the subclavian artery is visualized radiographically, but its vein is not. The groove in the left apex for the origin of the common carotid permits its radiologic visualization just medial to the origin of the left subclavian artery. A proper analysis of these interfaces just above the aortic arch may permit the suspected diagnosis of aberrant origin of the right subclavian artery.[3]

Medially, the apices approximate posterior to the esophagus, their parietal pleurae separated by the little connective tissue within the posterior mediastinum. The parietal pleura of the right apex covers the lateral surface of the right wall of the esophagus and the lateral surface of the right wall of the trachea where the trachea starts its contact with the lung. The parietal pleura of the left apex is separated from the left wall of the esophagus and from the left wall of the trachea by the subclavian and common carotid arteries, but at times it will contact the esophagus and trachea, with attendant roentgen visualization of these structures.

Superiorly, the apex with its parietal pleura projects into the connective tissue continuum of the neck and mediastinum, and is thus in potential contact with any process coming from neck or mediastinum.

The foregoing account gives anatomic basis for most of the normal and abnormal radiographic findings, which will best be depicted on roentgenograms made with three phase current, high voltage, and with screens and grid of superior quality. The radiologist is accustomed to study the apex on the standard posteroanterior roentgenogram as an impure structure, since it is viewed half-axially superimposed upon subjacent lung.

The apical cap has been exhaustively studied by Heitzman's extraordinarily productive school of roentgen-pathologic correlation.[4] Most caps are scars merging into the visceral pleura, such a characteristic lesion conditioned possibly by chronic apical ischemia, and are unilateral or bilateral. Apical caps of some older people with normal lungs may be due to a bumpy, rather than smooth, lung-extrapleural interface.

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[1] Brash, J. C. Cunningham's Textbook of Anatomy. Ninth edition. Oxford University Press, London, 1953.

[2] Cimmino, C. V. In: Golden's Diagnostic Radiology. Section V: Digestive Tract. Williams & Wilkins Company, Baltimore, 1969, Figure 15, p. 34.

[3] Branscom, J. J., and Austin, J. H. M. Aberrant right subclavian artery: findings seen on plain chest roentgenograms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1973, *119*, 539–542.

[4] Renner, R. R., Markarian, B., Pernice, N. J., and Heitzman, E. R. Apical cap. *Radiology*, 1974, *110*, 569–573.

The myth of the tuberculous nature of the inflammatory apical cap continues to die hard. Its postinflammatory nature in most cases is a diagnosis of exclusion, and may be accepted only after considering several normal and abnormal states:

1. The smooth, sharply outlined companion shadow of soft tissue inferior to the first rib (as well as subsequent ribs) should cause the radiologist little diagnostic difficulty.[4]

2. The right and left subclavian arteries are sharply delineated in their arcuate course over the apex towards the tubercle of the first rib,[5] and are commonly, but erroneously, interpreted as thickened pleura. Their identification is certain when both cephalic and caudal borders are present. The right subclavian artery is visualized less commonly than the left because its course is often more cephalic, merging into the right apical extrapleural soft tissues.

The subclavian artery is relatively unprotected anteriorly. In the proper clinical setting of severe injury to the neck with loss of blood, the apical cap should suggest rupture,[6] identified by emergency arteriography.

The subclavian vein has no pulmonary interface and is thus not visualized, as contrasted to the superior vena cava and the adjoining segment of the right innominate vein, but the subclavian vein acquires an interface and becomes visualized by extravasation from false passage of a catheter or from external trauma, or when it becomes ectatic in degenerative or obstructive states. Under these conditions, an apical cap is formed.

Similarly, degenerative, infectious or traumatic ectasias of any of the great root arteries of the thoracic inlet may cause an intrusion upon the apex with the formation of an apical cap.

Angiography will identify all these vascular conditions.

3. Retropleural hematoma associated with injury to the first rib is the commonest cause of the non-postinflammatory apical cap, from which it is differentiated by the day to day fluctuations in size. The usual injury is fracture, from either direct blow or violent muscular pull, such as sustained in basketball or wrestling. Callus from such an injury may later mimic an apical pulmonary tumor, the question resolved only by laminagraphy. Complete dislocation of the first rib at its costovertebral joint comprises a rare injury, with which I have just had my first experience.

4. A severe soft tissue injury to the neck without associated fracture may produce an apical cap. The hematoma depresses the apex, and may dissect extrapleurally beyond the confines of the apex. This cap also should vary from day to day, in contrast to the simple postinflammatory cap. My very few efforts in the morgue to reproduce this injury with simulated hematoma from percutaneously injected saline into the base of the neck have failed.

5. Migration of morbid fluid contents from the mediastinum into the connective tissue of the neck may depress the apex and dissect between rib and parietal pleura, just as cervical hematoma, and may form a similar apical cap. The commonest cause for such a process is rupture of the aorta, and the presence of a changing apical cap gives firm support to the radiologic diagnosis in the proper clinical setting.

6. A severe injury to the neck with avulsion of lower cervical nerve roots may produce an apical cap formed by an extrapleural pseudomeningocele, with free flow of pantopaque back and forth from the cap into the subarachnoid space.[7] Or, cerebrospinal fluid from a traumatic fistula between the dorsal spine and the mediastinum

[5] GONDOS, B. Roentgen image of subclavian artery in pulmonary apex. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 1058–1062.

[6] STURM, J. T., STRATE, R. G., MOWLEM, A., QUATTLEBAUM, F. W., and PERRY, J. F., JR. Blunt trauma to subclavian artery. *Surg., Gynec. & Obst.*, 1974, *138*, 915–918.

[7] EPSTEIN, B. S., and EPSTEIN, J. A. Extrapleural intrathoracic apical traumatic pseudomeningocele. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1974, *120*, 887–892.

may migrate cephalad to produce an apical cap.[8]

7. Migrations of cervical phlegmons caudad, and of mediastinal phlegmons cephalad, both producing an apical cap, are theoretical possibilities.

8. The right apex abutting on the right wall of the esophagus (with air in the esophageal lumen) forms the esophagopleural stripe.[9] Effacement of this interface means interposition of soft tissue, and produces a medial apical cap. Such interposed tissue is commonly pulmonary and pleural scar, but I have encountered it malignant. The left apex abutting on the left wall of the esophagus is too inconstant to be of much diagnostic worth.

9. Loss or thickening of the interface between right apex and right wall of the trachea requires explanation. The commonest cause for such change is interposed lymph nodes, benign or malignant, but make sure the thickening is not due to intrinsic disease of the trachea, such as degenerative, inflammatory or neoplastic states. I have encountered such intrinsic thickening even in heart failure. The inconstant presence of an interface between left apex and left wall of the trachea diminishes the importance of its loss.

10. Both apices approximate behind the esophagus somewhat to the left of the midline, such approximation forming the posterior mediastinal line,[10] which is composed of the two pleural coverings of each apex and of the connective tissue of the interposed mediastinum. Do not confuse this line with the left wall of the trachea. The commonest cause for permanent thickening of this line is postinflammatory thickening of each or of one apical pleural cap medially. I have seen this line so thick in a patient with congestive failure as to merit the designation of *stripe*, rather than *line*, only to revert to its linear status after relief of the heart failure: such thickening is due to edematous pleurae and pleural fluid, just as occurs in the more radiologically accessible pleurae in the bases.

11. Tumors may cause an apical cap. I prefer not to use the term superior sulcus tumor because the sulcus (pleural space) may not be involved, nor to use the term Pancoast tumor since this term has clinical and pathologic connotations which indeed may be absent. Bilateral caps do not exclude tumor; unilateral caps do not invariably indicate tumor. Tumors may be benign or malignant, primary or secondary, arise in the rib, lung, in either pleura, or extra-pleurally. Exclusion of tumor must be implicit in any diagnosis of postinflammatory apical cap.

In summary, much information is tucked away into that little volume of apex. Normal and abnormal mechanisms can produce the apical cap. Some of these mechanisms are suggested by the clinical picture and by the radiologic findings elsewhere in the lungs. Other mechanisms require angiography and other standard clinical and laboratory tests for elucidation. Postinflammatory scar tissue is the commonest cause, but implicit in this diagnosis is the exclusion of tumor.

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[8] Zilkha, R., Reiss, J., Schulman, K., and Schechter, M. M. Traumatic subarachnoid-mediastinal fistula. *J. Neurosurg.*, 1970, *32*, 473–475.

[9] Cimmino, C. V. Further notes on esophageal-pleural stripe. *Radiology*, 1961, *77*, 974–978.

[10] Cimmino, C. V., and Snead, L. O., Jr. Posterior mediastinal line on chest roentgenograms. *Radiology*, 1965, *84*, 516–518.

THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, held at the San Francisco Hilton Hotel, San Francisco, California, September 24–27, 1974, were numerically by far the largest in the Society's history; qualitatively, they were of beautiful visual display, and documentatively of the greatest scientific contributions.

In recognition of the over-all excellence, the Committee on Awards has presented one Gold Award, one Silver Award, one Bronze Award and 23 Certificates of Merit.

The following is a detailed account of the Exhibits.*

GOLD AWARD

On the Pathogenesis of Abnormal Modeling of Bone. Joseph P. Whalen, M.D., Lennart Krook, DVM, and Eladio A. Nunez, Ph.D., The New York Hospital, Cornell Medical Center, New York, New York.

A re-evaluation of normal developing human bone was presented and how alteration of this process is reflected in their roentgenographic appearance. Both experimental and clinical material was shown to elucidate the pathogenesis and possible humoral (parathormine, calcitonin) effect on these processes. Manipulative experiments carried out in the Exhibitor's laboratory have demonstrated the effect of Ca diets (high and low), calcitonin and parathormine on bone modeling. These have resulted in modeling errors as detected roentgenographically and histologically, and at the ultrastructural level.

* Editor's note. The assembly by Dr. James Franklin Martin, Chairman of the Committee on Scientific Exhibits, and his able committee of this magnificent array of exhibits is a fitting tribute to the *Diamond Anniversary* of the Society; their descriptive abstracts were prepared especially and are published *in extenso* to illustrate the tremendous progress made in the science of radiology.

SILVER AWARD

The Aortic Arch and Its Pleural Reflections; Their Relationship to Mediastinal Fat. Edward J. Lane, M.D., E. R. Heitzman, M.D., and David B. Hammack, M.D., State University of New York, Upstate Medical Center, Syracuse, New York.

The pleural reflections of the aorta, outlined by contiguous aerated lung, predicate the roentgenographic appearance of the left side of the mediastinum. Mediastinal fat is visualized frequently, causing these reflections to appear commonly as lines rather than lung-mediastinal interfaces.

This exhibit presented a correlated roentgen-anatomic study of these reflections and emphasized their appearance in the "aortic-pulmonic window" and over the left paraspinal fat. Reflections over the left subclavian artery and the left superior intercostal vein were shown as were those which produce the anterior junction line and the aorticopulmonary line. Alterations produced by lymphadenopathy, mediastinal tumors, vascular abnormalities and paraspinal disease were demonstrated.

BRONZE AWARD

The Emi-Scan, A New Dimension. R. Ethier, M.D., W. Feindel, M.D., D. Melanson, M.D., G. Belanger, M.D., D. Galloway, M.D., S. Taylor, M.D., and C. Chiu, M.D., Montreal Neurological Institute, Montreal, Quebec, Canada.

A variety of cerebral lesions were demonstrated using this new technique. Also the importance of a rapid infusion of large quantities of opaque medium was stressed.

In many patients, this new technique will alleviate the need for either pneumoencephalography or carotid angiography. The authors anticipate a complete change in the diagnosis and treatment of cerebral

diseases with this new diagnostic approach.

Certificates of Merit

Mediastinal Neuroblastomas and Ganglioneuromas. Jacob Bar-Ziv, M.D., and M. Bernadette Nogrady, M.D., Department of Radiology, The Montreal Children's Hospital, Montreal, Quebec, Canada.

Selected cases of primary and secondary mediastinal neuroblastomas were displayed to compare and contrast the roentgenologic findings. Widening of the intercostal space and rib erosions are the most characteristic findings in primary tumors. The intrathoracic soft tissue mass may not be readily recognizable. The rib changes disappear subsequent to therapy. Neurologic deficit may be minimal or absent even when complete block is evident by myelography. The secondary tumors are not associated with the above described rib changes.

In conclusion, the roentgenologic evidence of rib abnormalities adjacent to these mediastinal masses is practically pathognomonic for primary tumors.

Cervical Spine Tomography in Trauma. Eugene F. Binet, M.D., Joseph J. Moro, R.T., John P. Marangola, M.D., and Charles J. Hodge, Jr., M.D., Departments of Radiology and Neurosurgery, Upstate Medical Center, Syracuse, New York.

Cervical spine tomography for trauma was performed in 136 patients at the Upstate Medical Center between the years 1965–1973. In approximately 50 per cent of the patients examined, the tomograms yielded significant information not present or not appreciated on the plain cervical spine roentgenograms. The tomograms altered the patient's management in 37.5 per cent of the cases.

The normal tomographic anatomy of the cervical spine was demonstrated. Examples of odontoid fractures, spurious odontoid fractures, subluxations, facet fractures, congenital anomalies and miscellaneous lesions diagnosed on tomography were shown. The indications for cervical spine tomography in trauma were given.

Roentgen Diagnosis of Hilar Lymphadenopathy. C. H. Joseph Chang, M.D., University of Kansas Medical Center, Kansas City, Kansas.

The roentgen anatomy of hilar lymph nodes was presented and a rather typical distribution of hilar lymph nodes on the chest roentgenogram was identified. Roentgenographic techniques were also discussed.

A characteristic roentgen appearance of hilar lymphadenopathy was described and its differential diagnosis briefly discussed. The "Ripple Doughnut" sign is the characteristic roentgen finding of hilar lymphadenopathy. As indicated in this presentation, it appears that the diagnosis of hilar lymphadenopathy is best made with lateral body section roentgenograms.

Coronary Angiographic and Scintigraphic Findings in Experimental Cardiac Contusion. Ching L. Chiu, M.D., Raymundo Go, M.D., Donald Doty, M.D., James D. Roelofs, M.D., Kenneth Bell, M.D., and James H. Christie, M.D., Departments of Radiology and Surgery, University of Iowa Hospitals and Clinic, Iowa City, Iowa.

An isolated myocardial injury was produced using the impact of a captive-bolt handgun to the intact chest wall of dogs. Scanning with stannous 99m polyphosphate and tissue distribution studies showed good localization of the radiopharmaceutical in the injured myocardium (normal:injured ratio up to 1:14) up to 72 hours after injury. Postmortem angiography demonstrated sequential coronary vascular changes at 24 hours, 72 hours, and 1 week post trauma.

Computerized Axial Tomography of the Cerebral Ventricles and Subarachnoid Spaces. Steven H. Cornell, M.D., James H. Christie, M.D., Ching L. Chiu, M.D., and Lynn W. Lyon, M.D., University of Iowa Hospitals, Iowa City, Iowa.

The cerebral ventricles and subarachnoid spaces are particularly well shown by EMI scanning. From the scans of more than 500 persons the following conditions were il-

lustrated: 1. normal structures; 2. hydrocephalus of varying etiology; 3. cerebral atrophy; 4. porencephaly; 5. ventricular displacement, distortion or compression secondary to intracranial mass lesions; 6. congenital anomalies; 7. subdural hematoma; 8. shunt failure; and 9. postoperative changes after shunting procedures.

Thin Section Roentgenography of the Temporal Bone. John T. Eyre, M.D., Cincinnati General Hospital, University of Cincinnati, Cincinnati, Ohio.

A review of the entire subject of temporal bone tomography was presented. There were sections on normal anatomy, with a three dimensional model for orientation, congenital anomalies, infection, cholesteatoma, neoplasm, trauma, and otodystrophies. There were representative cases of each major disease entity.

Rickets; Differential Diagnosis. Norma E. Fallis Harris, M.D., Helene M. Tanous, M.D., and Edward B. Singleton, M.D., Baylor College of Medicine, Houston, Texas.

Various entities that give rachitic changes in the bones were presented, in addition to the vitamin D deficient rickets. Differentiating features were stressed in each instance—roentgenologic, clinical and biochemical.

Also included were several conditions that have caused some confusion in differential diagnosis, such as biliary rickets, glomerular rickets, Blount's disease, hypophosphatasia, coxa vara, and metaphyseal dysostosis. Emphasis was on the conditions where recognition and treatment might be curative or palliative and where vitamin D would not affect the course of the disease and might even be toxic.

Cardiac Tumors of Infancy and Childhood: Clinical and Roentgenographic Manifestations. Edmund A. Franken, Jr., M.D., John A. Smith, M.D., and Roger A. Hurwitz, M.D., Indiana University Medical Center, Indianapolis, Indiana.

Cardiac tumors affecting children are of varied types and locations and differ in clinical and roentgenographic features. Because surgical therapy may be curative, recognition and delineation of these tumors is critical.

In this exhibit the various tumors affecting the heart were classified and discussed on the basis of their hemodynamic effects. Selective obstruction of great veins, right heart, pulmonary arteries, pulmonary veins, and left heart were illustrated, and some of the unique plain film and angiographic characteristics of cardiac tumors were identified. The roentgen findings were correlated with the clinical picture in the illustrated cases.

Roentgen Evaluation of the Post-Pneumonectomy Chest: Normal and Abnormal Findings. Robert E. Hanson, M.D., Reinhard Kloiber, M.D., Richard R. Lespérance, M.D., and Darrill D. Munro, M.D., Department of Diagnostic Radiology, Royal Victoria Hospital, the Departments of Radiology and Surgery, the Montreal Chest Hospital Centre and McGill University, Montreal, Quebec, Canada.

Chest roentgenography plays an important role in the immediate and late postoperative assessment of pneumonectomy patients.

The sequence of events that occurs in obliterating the pleural space following pneumonectomy was evaluated on serial roentgenograms. By reviewing a large series of clinical cases, a definite pattern of the normal postoperative changes was recognized and formed the basis of this presentation.

Thorough familiarity with this pattern is essential since deviation from it may provide the earliest clue to serious complications such as bronchopleural fistula and empyema. The exhibit presented a number of examples illustrating both the normal and abnormal roentgen features of this study.

Retropharyngeal Tendinitis. Cosmo L. Haun, M.D., Emory University, Atlanta, Georgia.

Acute inflammation with calcareous deposits in the longus colli tendons is a rare entity with a very characteristic roentgeno-

graphic pattern and clinical presentation. Roentgenographic findings in the acute phase with follow-up to resorption of the calcium were demonstrated. The similarity to calcific tendinitis in the shoulder was emphasized. Examples of calcific tendinitis in other less common locations were also included in this exhibit.

Compensatory Mechanisms in Communicating Hydrocephalus. A. Everette James, Jr., M.D., William J. Flor, Ph.D., Timothy Merz, Ph.D., Donald L. Price, M.D., Barry Burns, Ph.D., and Melvin Epstein, M.D., The Johns Hopkins Hospital, Baltimore, Maryland.

Communicating hydrocephalus appears to progress rapidly in the early phases until compensatory mechanisms resist further ventricular enlargement. Compensation probably occurs by either diminished cerebrospinal fluid (CSF) production or absorption by alternative pathways.

After creation of communicating hydrocephalus in dogs by subarachnoid injection of silastic, cisternograms, ventriculograms and CSF pressure measurements were made. Serial microscopic and ultrastructural studies were obtained. Autoradiographs were used to image transependymal macromolecular movement. CSF production was monitored by ventriculo-cisternal perfusion studies and measurement of the metabolic activity of the choroid plexus by the ampulla microdiver technique.

These data support the concept that the major compensation is not due to changes in CSF production, but to alterations in absorption. Cisternograms, photographs, autoradiographs, graphs and drawings illustrated these techniques and findings.

Infusion Tomography of the Female Pelvis. Leon Love, M.D., Myron Melamed, M.D., Richard A. Cooper, M.D., Rogelio Moncada, M.D., and Harold Schwartz, M.D., Loyola University of Chicago Medical Center, Stritch School of Medicine, Maywood, Illinois.

Total body opacification has recently been described in the evaluation of the gallbladder, adrenal cysts, fibroids, ovarian cysts and other entities. When coupled with tomography, total body opacification can be used to evaluate the female pelvis. This is a simple modification of the intravenous pyelography that is frequently ordered prior to gynecologic surgery.

The method of this procedure as well as several examples were presented. Also, the history and physiology of total body opacification were briefly discussed.

The Roentgenologic Findings in Henoch-Schoenlein Purpura. Roderick Ian Macpherson, M.D., Children's Hospital of Winnipeg, Winnipeg, Manitoba, Canada.

Henoch-Schoenlein purpura is a well-known clinical syndrome in which nonthrombocytopenic purpura is associated with arthralgia, abdominal pain and abnormalities of the urinary sediment. In most cases, it is a benign, self-limiting disease, rarely concerning the radiologist. Occasionally, gastrointestinal, urinary and pulmonary complications may occur and roentgenologic investigations will be requested.

This exhibit presented a study of 90 consecutive children with Henoch-Schoenlein purpura. The roentgenologic findings in the gastrointestinal tract, urinary tract and chest were demonstrated.

The most common and most spectacular lesions are those found in the small bowel. Here, areas of mucosal and/or submucosal hemorrhage and edema produce characteristic filling defects, which may cause transient bowel obstructions or lead to intussusceptions. These lesions could develop before the rash or joint symptoms and their roentgenologic demonstration could prove instrumental in avoiding unnecessary surgery.

Tracts in Granulomatous Colitis and Diverticulitis. Richard H. Marshak, M.D., Rubem Pochaczevsky, M.D., Jerold Kurzban, M.D., and Daniel Maklansky, M.D., Mt. Sinai School of Medicine, New York, New York.

The Exhibitors previously reported 10 cases of granulomatous colitis in association with diverticula and described what

was considered to be a pathognomonic roentgen finding, consisting of a fistulous tract parallel to a thickened bowel wall, usually exceeding 10 cm. in length. Since that time they have accumulated approximately 75 cases with this type of tract. Eight of these cases were proven to be diverticulitis and, therefore, the Exhibitors must amend their previous concept.

The differential diagnosis is aided in granulomatous disease by thickened folds, ulceration of the mucosa, and other roentgen findings of Crohn's disease of the colon. Characteristic cases demonstrating the 2 entities were presented and it is the Exhibitors' conclusion that a long intramural tract is still a highly reliable sign in granulomatous colitis.

The Nonopacified Gallbladder: Incidence, Causes, Significance, and Fate. Zuheir Mujahed, M.D., Joseph P. Whalen, M.D., and John A. Evans, M.D., The New York Hospital, Cornell Medical Center, New York, New York.

The authors reviewed 5,000 oral cholecystographies performed in the last 3 years to determine the incidence, causes and significance of nonopacification of the gallbladder. The value of a third or a fourth repeat study, as well as the contribution of intravenous cholangiography to the diagnoses of gallbladder diseases were also examined. The study led to the following conclusions:

As much as 25 per cent of patients require a second dose; therefore, the examination is not complete unless the gallbladder is adequately opacified or a second consecutive dose has been given.

Nonopacification of the gallbladder on 2 consecutive oral cholecystographies indicates obstructive gallbladder disease or chronic cholecystitis. Intravenous cholangiography could differentiate between these 2 conditions by virtue of the fact that it does not depend on the concentrating ability of the gallbladder for opacification. It will fill the gallbladder in chronic cholecystitis, but not in obstructive disease.

Oral cholecystography is more reliable than intravenous cholangiography in the investigation of disease of the gallbladder, since a chronically inflamed gallbladder which has lost its concentrating ability will fill and appear roentgenographically normal on the intravenous study.

Intravenous cholangiography is indicated in the investigation of diseases of the gallbladder only when the oral study is impractical, as in acute emergencies.

Lesions Involving the Gasserian Ganglion and Trigeminal Nerve. Enrique Palacios, M.D., Behrooz Azar-Kia, M.D., and Albert V. Messina, M.D., Loyola University of Chicago, Maywood, Illinois and The New York Hospital, Cornell University Medical College, New York, New York.

Roentgenography may disclose the causes of "trigeminal neuralgia." The normal anatomic features of the gasserian ganglion and trigeminal nerve were presented, along with multiple cases of lesions involving the gasserian ganglion and trigeminal nerve producing symptoms mimicking trigeminal neuralgia.

The importance of using modern roentgenographic techniques, such as plain film roentgenography, tomography, angiography, pneumography, and the new breakthrough in Computerized Axial Tomography with the EMI scanner was stressed.

The Cranial Arachnoid Granulation. D. Gordon Potts, M.D., Daniel G. Gomez, Ph.D., Gerald F. Abbott, M.D., and Joseph F. Sackett, M.D., Cornell University Medical College, New York, New York.

The arachnoid granulations are complex protrusions of arachnoid cells through small dural defects into the superior sagittal sinus, lacunae laterales or adjacent veins. The stroma of the granulation is formed by arachnoid cells and the granulation is covered by endothelium continuous with that lining the vein. The granulation is traversed by multiple endothelial-lined tubules that open at its surface. The sheep has arachnoid granulations that are similar to those in man.

This exhibit displayed some of the roent-

genologic, physiologic, structural and ultrastructural studies of the granulations in sheep and in man.

Roentgenographic-Pathologic Correlation of Wrist Involvement in Rheumatoid Arthritis. Donald Resnick, M.D., Veterans Administration Hospital, San Diego, California.

Analysis of wrist alterations in rheumatoid arthritis was undertaken to demonstrate particular roentgenographic-pathologic correlations.

The material included wrist roentgenograms of 100 rheumatoid patients, wrist arthrograms in 40 rheumatoid patients, pathologic sections of entire wrist joint in 6 rheumatoid cadavers, and anatomic sections of 20 wrists.

Specific abnormalities in the prestyloid recess of the radiocarpal cavity, volar radial recesses, inferior radio-ulnar joint, and extensor carpi ulnaris tendon sheath produced characteristic roentgen alterations. Pisiform-triquetral cavity involvement is also distinctive. Peculiar patterns of bone fragmentation were noted and apparently explained by dynamic alterations in the rheumatoid wrist.

Painful Total Hip Prosthesis: Roentgenologic Evaluation. Wallace M. Roy, M.D., and Michael Tam, M.D., St. Joseph's Hospital, Toronto, Ontario, Canada.

Ten per cent of patients with total hip prosthesis might ultimately develop complications. This exhibit demonstrated the following features of painful hip prosthesis: loosening of prosthesis in both acetabular and femoral components; loosening of acrylic cement from the bony socket and unstable position of cement plugs; malalignment of prosthesis such as femoral tip protruding outside cementation or perforating bony cortex; and malangulation of plastic socket.

Displayed also were the many roentgenographic manifestations associated with inflammation developing in total hip replacement. These were: thickened synovial membrane; enhanced lymphatic drainage; hypervascularity; abscesses in soft tissue and in bone; fistula formation; and chronic osteomyelitis.

Post-traumatic complications included femoral shaft fracture distal to prosthesis, subluxation and dislocation of prosthesis. Other causes of painful hip also shown were periarticular hypertrophic ossification and loose bodies in joint space.

The need of hip arthrography with positive contrast material to demonstrate many of these abnormalities was also emphasized.

The Roentgenology of Systemic Lupus Erythematosus: a New Evaluation. Lorraine Goldine Shapeero, M.D., and Wallace T. Miller, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

Systemic lupus erythematosus has multisystemic manifestations and can masquerade as numerous clinical entities. In an analysis of 141 patients with systemic lupus seen at the Hospital of the University of Pennsylvania, an organized approach toward the roentgenologic diagnosis of this disease has been developed.

This evaluation and its correlation with the clinical manifestations were presented and discussed.

The Navicular Fat Stripe: a Useful Roentgen Feature for Evaluating Wrist Trauma. Daniel W. Terry, Jr., M.D., and James E. Ramin, M.D., The Medical College of Wisconsin, Milwaukee, Wisconsin.

Diagnosis of navicular fracture remains a challenge for the radiologist. The navicular fat stripe (NFS), a lucent line adjacent to the lateral border of the navicular bone, is a useful roentgen feature in diagnosing wrist trauma.

On roentgenograms of 400 normal wrists examined in our emergency department, 96 per cent showed an identifiable, nondisplaced NFS. In contrast, the NFS was absent or significantly displaced in 28 of 32 fractures involving the bones of the radial side of the wrist. A positive NFS sign was present in 13 of 15 navicular fractures.

Localization of Orbital Lesions by Venography. Jacqueline Vignaud, M.D., Claude

Clay, M.D., and Larissa Bilaniuk, M.D., Fondation Ophtalmologique Rothschild, Paris, France, Hôtel Dieu, Paris, France, and University of Pennsylvania, Philadelphia, Pennsylvania.

High accuracy (95 per cent) in localization of orbital lesions was illustrated by cases selected from 480 venographies performed at Fondation Ophtalmologique Rothschild in Paris. Characteristic displacements were shown on roentgenograms and explanatory diagrams.

B-Scan Ultrasound Evaluation of the Pancreas. William J. Walls, M.D., Giomar Gonzalez, M.D., Norman L. Martin, M.D., and Arch W. Templeton, M.D., University of Kansas Medical Center, Kansas City, Kansas.

Ninety-four patients examined by B-scan ultrasound for suspected pancreatic disease had a proven final diagnosis. The diagnostic accuracy of ultrasound, upper gastrointestinal series, isotope pancreatography, and arteriography was determined and compared in these patients. Pancreatic sonography provided the most accurate (84 per cent) and least expensive method for evaluating the pancreas with no known patient morbidity. The upper gastrointestinal series and isotope study were complementary to sonography. Arteriography was then useful in selected cases.

The diagnostic ultrasound features of pancreatic neoplasm, pancreatitis and pancreatic pseudocyst were described and illustrated. Ultrasound should be the initial examination for the evaluation of patients with suspected pancreatic disease.

Other Excellent Exhibits Were:

The Specificity of Renal Vein Thrombosis. Peter H. Arger, M.D., Charles Mulhern, M.D., Wallace T. Miller, M.D., and Arnold Chait, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

The clinical aspect of adult renal vein thrombosis has previously been described as nonspecific. Also, the intravenous pyelographic changes have been characterized as a nonspecific spectrum.

There are 4 fundamental causes of renal vein thrombosis: (1) thrombosis of inferior vena cava and the renal vein secondary to extension of thrombi from pelvic and leg veins; (2) tumor compression or invasion of the inferior vena cava and renal veins; (3) thrombosis of renal veins secondary to primary renal disease; and (4) primary renal vein thrombosis. Except in the infant, primary renal vein thrombosis is rare.

The clinical and roentgenographic findings in renal vein thrombosis are dependent upon the completeness of occlusion of the renal vein or veins and the rapidity of development of collateral venous drainage.

If complete occlusion of the renal vein occurs acutely with little chance for collateral venous drainage to develop, the affected kidney enlarges with consequent severe back pain, hematuria, proteinuria and oliguria. Renal failure and death generally occur if the condition is bilateral. If the occlusion is gradual or partial, collateral venous drainage will develop, allowing some preservation of renal function. In this most common situation, nephrotic syndrome generally develops.

Subtleties of the Knee. Richard Arkless, M.D., Portland Adventist Hospital, St. Vincent Hospital, Portland, Oregon.

Using arthrography, observations of unusual normal variations and interesting pathoses were made. These included septations and divisions of the suprapatellar pouch and loose and attached masses therein; the normal filling of tiny (occasionally large) draining veins around the knee; normal structures confused with meniscus tears; and how to avoid false positive and false negative diagnoses of meniscus injuries.

Subependymal Veins: Normal and Abnormal. Behrooz Azar-Kia, M.D., Enrique Palacios, M.D., Mohammad Sarwar, M.D., and Mannie M. Schechter, M.D., Loyola University of Chicago, Chicago, Illinois, and Albert Einstein College of Medicine, Bronx, New York.

The arterial phase of angiography may not always show definite diagnostic findings in cases of intraventricular or paraventricular lesions. Pneumography for the most part will demonstrate the localization of these lesions. However, changes in the subependymal veins may provide important diagnostic angiographic information in the presence of intraventricular or adjacent lesions of the lateral ventricles. The angiographic venous phase may demonstrate the following diagnostic findings in the subependymal veins: early filling, enlargement and multiplicity, spreading and distortion. Examples of these findings were shown in cases of intraventricular and paraventricular lesions.

Vascular Malformations of the Cecum. Jeanne Willner Baer, M.D., and Stephen Ryan, M.D., St. Luke's Hospital Center, New York, New York.

Fifty postmortem and surgically extirpated cecums were examined angiographically and microscopically in a search for vascular malformations and in order to define the normal vascular pattern. The findings were compared with the appearance of 2 surgically proved cases of vascular malformations of the cecum, 1 nonspecific cecal ulcer, and bleeding diverticula of the cecum.

Lordotic Projections for Improved Visualization of the Left Coronary Artery and Its Branches. Harold A. Baltaxe, M.D., and Thomas A. Sos, M.D., The New York Hospital-Cornell University Medical Center, New York, New York.

A new roentgenographic technique for the visualization of the left coronary artery and its branches was described. It consists of a lordotic right anterior and left anterior oblique projection. The left coronary artery is roentgenographed with the horizontal beam against the lateral changer, the tubes being angulated towards the head of the patient.

This projection allowed the Exhibitors to demonstrate lesions of the proximal left anterior descending coronary artery and of the diagonal branches which otherwise would have been hidden. The roentgenographic technique was described and multiple examples of lesions were shown to demonstrate the value of the technique.

The Pulmonary Varix. Bruce R. Baumgartner, M.D., Stewart R. Roberts, Jr., M.D., J. Luther Clements, Jr., M.D., and H. Stephens Weens, M.D., Emory University Affiliated Hospitals, Atlanta, Georgia.

The pulmonary varix is a localized enlargement of a pulmonary vein. It is an uncommon entity, usually of no clinical concern, which may be a complication of pulmonary venous hypertension or may represent an isolated finding. It may present as a pulmonary nodule which must be differentiated from other pulmonary mass lesions.

The exhibit illustrated 3 patients with rheumatic mitral valvular disease and pulmonary varix. Chest roentgenograms, fluoroscopy, tomography and venous phase angiography demonstrated the typical roentgenographic findings.

Immunologic Basis of Hypersensitivity Diseases of the Lung. Yahya M. Berkmen, M.D., New York Medical College, Metropolitan Hospital Center, New York, New York.

The Exhibit emphasized known facts about humoral and cell-mediated immunity. Importance of the thymus and lymphatic-reticuloendothelial system in immunology was stressed. Four types of allergic responses (Type I—IgE mediated; Type II—cytotoxic tissue-specific; Type III—immune complexes; Type IV—delayed hypersensitivity) were described and examples of each were given.

Ten examples were illustrated with case reports.

Chest Roentgenography as a Window to the Diagnosis of Takayasu's Arteritis. Yahya M. Berkmen, M.D., and Adam Lande, M.D., New York Medical College, Metropolitan Hospital Center, New York, New York.

This Exhibit presented plain chest roentgenographic findings in Takayasu's arteri-

tis, based on the study of 25 cases. Clinical angiographic correlation and examples of changes seen in the aorta, central and peripheral pulmonary arteries were demonstrated. The importance of awareness of the radiologist, who commonly is confronted with specific clinical manifestations and chest roentgenograms, in making or suggesting the diagnosis was stressed.

Shoulder Arthrography Technique: Diagnostic Value and Pitfalls. Edward I. Bluth, M.D., Bong S. Lee, M.D., Larissa T. Bilaniuk, M.D., Wallace T. Miller, M.D., and Won Sik Cynn, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

The valuable role that shoulder arthrography can play in evaluating shoulder pain was demonstrated. Normal anatomy was described and illustrated. The technique of the examination was explained and diagnostic pitfalls were discussed. Pathologic examples, including biceps tendon ruptures, and rotation cuff tears, were presented. The roentgenograms were accompanied by explanatory diagrams.

Atypical Teratomas of the Central Nervous System. R. N. Bryan, M.D., M. B. Camins, M.D., L. E. Swischuck, M.D., and R. L. Waldron, II, M.D., Neurological Institute of New York, New York, New York.

Atypical teratomas of the central nervous system often have a distinctive clinical and roentgenographic presentation. These tumors most frequently occur in the pineal region (accounting for more than 50 per cent of "pinealomas"), but are also found in the suprasellar region. Occasionally, patients present with separate lesions in both locations, in which case there is a unique roentgenographic picture. These tumors are also prone to metastasize elsewhere within the neural axis.

A review of these tumor cases in the Exhibitors' institutions included more than 60 tumors of the pineal region (not all histologically verified as atypical teratomas), 14 suprasellar tumors, and 7 multifocal midline lesions. The Exhibit demonstrated the typical plain film roentgenographic, angiographic, and ventriculographic findings of these tumors plus some unusual examples including metastasis.

Xeromammography: Comparison of Molybdenum and Tungsten Target Images. Thomas Carlile, M.D., Barbara Brookins, R.T., Willis J. Taylor, M.D., McClure H. Hall, M.D., and Kenneth D. Moores, M.D., The Mason Clinic, Virginia Mason Medical Center, Seattle, Washington.

Images were made on the same patient with 2 identical x-ray units, except that a tungsten tube was in one and a molybdenum tube in the other. Images were made on 50 patients with 3 techniques: 48 kv. molybdenum (Mo); 38 kv. Mo; and 38 kv. tungsten (W). The 48 kv. Mo images were rejected as lacking in detail and an additional 50 images were made with 38 kv. Mo and 38 kv. W techniques. Selection by 4 radiologists of preferred technique as a blind procedure was as follows: 48 kv. Mo—15 per cent; 38 kv. Mo—35 per cent; 38 kv. tungsten—50 per cent. Doses were measured by TLD dosimeters and from air measurements with BSF and F corrections with good agreement. Average dose with tungsten equalled 6.0 rads (range 3.1–8.1); 38 kv. Mo 9.01 rads (range 5.3–13.5); 48 kv. Mo 4.4 rads (range 3.1–6.1).

Although the 48 kv. Mo image had the lowest dose, the detail was unacceptable. The 38 kv. Mo image and the 38 kv. W image were close in quality, but the 50 per cent lower dose and the moderately higher selection factor favored the tungsten image.

Xerotomography of Pulmonary Parenchymal Lesions. Richard L. Carnovale, M.D., John E. Martin, M.D., Bao-Shan Jing, M.D., and Thomas S. Harle, M.D., The University of Texas Medical School at Houston Affiliated Hospitals, Houston, Texas.

Xeroradiography is recognized as a valuable diagnostic aid in the study of the breast because of its edge enhancement, wide roentgenographic latitude, detail, and demonstration of calcifications. Recently the Exhibitors have applied the technique of xerotomography in diagnostic evaluation

of pulmonary parenchymal lesions in hope of improving diagnostic specificity of plain film tomography.

Xerotomograms were obtained at the level of most information determined from review of plain film tomograms. The corresponding xerotomogram and plain film tomogram were compared for degree of diagnostic information.

The xeroradiographic plate was exposed at 120 kvp. at 200 to 300 mas. A linear 40° arc was utilized, obtaining a thin section. Patient exposure was approximately 7.5 r for a single xerotomographic exposure.

After a review of their material, the Exhibitors' preliminary conclusions suggest that a single xerotomographic image, because of increased information, may supersede conventional film tomographic study in some cases.

Anatomic Variations of the Thoracic Duct and Mediastinal Lymph Nodes: Lymphangiographic Study. Eung Man Cha, M.D., and Pakorn Sirijintakarn, M.D., Mount Sinai Hospital Medical Center, Chicago, Illinois.

Anatomic details of the thoracic duct are not as yet clearly defined. The recent popularity of lymphangiography allowed the Exhibitors to investigate the thoracic duct on chest roentgenograms taken during the lymphangiographic procedure in 175 cases in the past 2 years. They found many variations of the thoracic duct, such as duplication, multiple channels, segmental plexus, or Y-shaped duct. The frequency of visualization of mediastinal lymph nodes was highly associated with the variations of the thoracic duct. The statistical study of the variations of the thoracic duct and visualization of the mediastinal lymph nodes was collaborated.

Roentgenologic postmortem studies were done to evaluate the thoracic duct, as well as microscopic studies of the thoracic duct and lymphatic valves.

This Exhibit described the anatomic variation of the thoracic duct in relation to the visualization of the mediastinal lymph nodes.

Roentgenologic Spectrum of Duodenal Diseases. HiKon Chon, M.D., Edward J. Petrella, M.D., and Larissa T. Bilaniuk, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

A roentgenologic spectrum of diseases involving the duodenum was presented. Differential aspects of the various types of ulcers, masses, distortion of loop and general dilatation were pointed out. The importance of multiple roentgenography and sequence of examinations in differentiating such entities as pseudodiverticulum from a giant ulcer, or inflammation and hemorrhage from scarring and neoplasm was illustrated.

Sequential Brain Scanning as an Adjunctive Scanning Procedure. James H. Christie, M.D., Raymundo Go, M.D., Yutaka Suzuki, M.D., and Norihisa Tonami, M.D., Section of Nuclear Medicine, University of Iowa, Iowa City, Iowa.

The purpose of this Exhibit was to show how sequential brain scanning helps in the detection of lesions not demonstrated by the routine brain scan.

Sequential brain scans were performed at various, arbitrarily chosen time intervals up to 4 hours after injection of $NaTc^{99m}O_4$ in patients who had previously undergone routine 1 hour brain scans which were considered normal or equivocal.

The main value of this procedure lies in its ability to demonstrate progressive and subtle count-rate changes between normal anatomic structures and pathologic processes observed in relation to time, thus allowing detection of a lesion. This cannot be appreciated on any single static scan viewed independently, nor on routine brain scans; thus detection of the lesion was not possible. This procedure is not only helpful in differentiating a lesion from an artifact or a normal variant; in detecting small lesions in the supratentorial areas; but is also more helpful in detecting lesions in the sellar and infratentorial areas.

Abdominal Angiographic Spectrum in Tuberous Sclerosis. William R. Compton, M.D., Myo M. Kyaw, M.D., and Patrick

D. Lester, M.D., University of Utah Medical Center, Salt Lake City, Utah.

Tuberous sclerosis does not always manifest as a classic triad of seizures, mental retardation and sebaceous adenomas. Occasionally, large cystic appearing kidneys may be found, representing both cysts and angiomyolipomas. The Exhibitors noted a number of examples, where clinical findings may or may not be classic, but with characteristic angiographic changes. These findings were presented and included arteriovenous malformations (renal and hepatic) and renal microcyst formation in addition to more familiar large renal cysts and angiomyolipomas.

Filling Defects of the Urinary Bladder Simulating Neoplasm. Orlando C. Cordero, M.D., Ricardo Baldonado, M.D., Rajendra Kumar, M.D., Restituto Maravilla, M.D., and Robert Monaco, New Jersey Medical School, Martland Hospital, Newark, New Jersey.

Benign lesions of the urinary bladder, and benign lesions arising in the pelvic cavity, ranging from the more common to the rare entities may cause filling defects in the urinary bladder that can mimic carcinoma. Many of these lesions present with distinguishing roentgenographic features, so that differentiation of these benign lesions from carcinomas affecting the urinary bladder can be made. The roentgen features of these entities were described, and an attempt to classify them into extrinsic and intrinsic categories was made.

In a few of these entities, however, the roentgenographic appearance is not enough to exclude carcinoma. This is particularly true for lesions that affect the bladder wall. The diagnosis for some of these lesions depends on clinical grounds and the awareness that these entities do exist.

The Modulus of Elasticity of Human Cortical Bone: An in vivo Measurement. J. Duncan Craven, BM Bch., Moses A. Greenfield, Ph.D., David S. Wishko, M.D., and Richard Stern, Ph.D., University of California—Los Angeles, Department of Radiological Sciences, Los Angeles, California.

A technique was demonstrated for measuring modulus of elasticity in human cortical bone *in vivo*, a measurement not previously obtainable except by *in vitro* methods.

The technique involves a combination of the measurement of the velocity of ultrasound and measurement of bone mineral content by a photon absorption method. Data were presented comparing the results obtained from patients and normal volunteers and some implications were discussed.

Cystic Fibrosis: A Commonly Ignored Disease. David Jennings Curtis, M.D., and Massoud Majd, M.D., Department of Radiology, Walter Reed General Hospital, Washington, D. C., and Children's Hospital, National Medical Center, Washington, D. C.

A multisystem review of the roentgenographic patterns and manifestations of cystic fibrosis was presented and discussed. Included were the characteristic changes in the paranasal sinuses; the early, chronic and protean changes in the chest; recently described small bowel patterns; the not so simple meconium ileus complex of congenital small bowel disorders; changes in the skeletal system; and a previously undescribed finding of soft tissue ossification.

Ankle Arthrography. Won-Sik Cynn, M.D., Patricia Gureghian, M.D., Barry L. Levin, M.D., and James Nixon, M.D., Departments of Radiology and Orthopedics, University of Pennsylvania School of Medicine, Graduate Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

Ankle arthrography is a simple, safe and accurate method to evaluate the soft tissue injury of the ankle joint. The indications for and technique of this procedure, and normal variations as well as abnormal ankle arthrograms were presented. The authors demonstrated: (1) ruptured anterior talofibular ligament; (2) filling of peroneus tendon sheath from ruptured calcaneofibular ligaments; and (3) rup-

tured deltoid ligaments and ruptured syndesmosis. Proper treatment of the acute tear of ligaments around the ankle is important to prevent a chronically unstable joint.

Results of Nonsurgical Treatment of Atherosclerotic Iliac Narrowing. Charles T. Dotter, M.D., Josef Rösch, M.D., Janice Anderson, M.D., and Ruza Antonovic, M.D., University of Oregon Medical School, Portland, Oregon.

Percutaneous transfemoral balloon dilatation was carried out in 40 patients with clinically significant iliac arterial stenoses. The use of a balloon gave generous dilatation *via* a small entry hole; a specifically designed caged balloon catheter was used to provide the needed distending force. Most patients were treated because of symptoms due to known iliac stenoses; in several, however, dilatation was done in conjunction with retrograde diagnostic catheterization for other purposes (visceral or coronary angiography).

Follow-up periods ranged up to 5 years and showed good results in simple, short stenoses. Diffuse stenoses were more likely to recur; in some instances, redilatation was successfully carried out.

It was concluded that transluminal dilatation of stenotic iliac arteries is a safe, effective and a far more pleasant alternative to major surgery.

Early Detection of Breast Cancer. Robert L. Egan, M.D., American College of Radiology, Chevy Chase, Maryland.

This Exhibit described, through use of a flow-chart, the life giving benefit of early detection of breast cancer and death resulting from nondetection. This message was conveyed through photographs, fiberoptics and palpable rubber breasts.

Polytome Pneumography to Evaluate the C.N.S. in Infants and Children. Taher El Gammal, F.F.R., Medical College of Georgia, Augusta, Georgia.

With the advent of the EMI scanner, conventional pneumoencephalography will be infrequently performed.

The technique of polytome (a) pneumoencephalography, (b) ventriculography and (c) air myelography was briefly illustrated. Examples of detailed normal anatomy and of interesting pathologic cases were shown. These included dysraphism, Arnold-Chiari malformation, cyst of septum pellucidum, aqueduct obstruction, hamartoma of the thalamus, medulloblastoma, ependymoma, and pontine glioma.

The Roentgenographic Manifestations of Endometriosis. Charles Joseph Fagan, M.D., University of Texas Medical Branch, Galveston, Texas.

Endometriosis is a common disease developing in a large number of women during their active menstrual life. The lesions of this disorder may be roentgenographically apparent.

It was the purpose of this Exhibit to review the clinical and roentgenographic aspects of endometriosis as it involves the genitourinary, gastrointestinal and respiratory tracts. The author presented examples of endometriosis of the lung and catamenial pneumothorax. The importance of correlating the clinical aspects of the disease with the roentgenographic changes, permitting one to suggest the correct diagnosis, was stressed.

Osteomyelitis in Childhood. Alvin Harold Felman, M.D., and Milton Steve Stratos, M.D., University of Florida, Gainesville, Florida.

Pyogenic osteomyelitis remains a significant pediatric disease, although its manner of presentation has changed somewhat in recent years. The roentgenologist often assumes an important role in the diagnosis and treatment of these patients.

This Exhibit reviewed some of the classic roentgen patterns of osteomyelitis and illustrated the roentgenograms of patients whose clinical and roentgenographic findings differed from the expected. Comparison with other noninfectious abnormalities was stressed. Illustrations and discussions of the correlative histopathology completed this Exhibit.

Lesions of the Larynx: Roentgen Appearance with Clinico-Pathologic Correlation.

Pieretta S. Ferro, M.D., Jindra A. Kalus, M.D., Robert M. Komorn, M.D., Jyung-Whan Min, M.D., and Luceil B. North, M.D., Veterans Administration Hospital, Baylor College of Medicine, Houston, Texas.

Different lesions of the larynx, mostly malignant tumors, were studied clinically and evaluated roentgenographically with tomographic and contrast studies and correlated with the surgical and pathologic specimens.

Photographs of the specimens, laryngograms and tomograms were presented.

Neurogenic Disorders of the Foot in Diabetes Mellitus. Nathaniel Finby, M.D., Ernest Kraft, M.D., and Efthimios Spyropoulos, M.D., St. Luke's Hospital Center, V.A. Hospital, Outpatient Clinic, New York, New York.

Two types of diabetic neuropathy were distinguished: (1) *Charcot Joint* (destructive type) affects tarsal bones; and (2) *Bone Absorption* (mutilating type) is confined to the forefoot. Both types may occur in the same foot and even bilaterally.

Specific roentgen findings: (1) can be correlated with a known diabetes; (2) can serve as a clue to the detection of an unknown diabetes; and (3) can precede an overt diabetes and, thereby, aid in the management of the latent metabolic disorder.

Perforated Viscus Presenting with Gas in the Soft Tissues (Subcutaneous Emphysema). Thomas W. Fiss, Jr., M.D., Olcay S. Cigtay, M.D., Albert J. Miele, M.D., and Homer L. Twigg, M.D., Georgetown University Hospital, Washington, D. C.

Eight cases which represented the spectrum of gas in the soft tissue following a perforated hollow viscus were reviewed. These included 2 cases each of perforation secondary to appendicitis, colonic carcinoma and diverticulitis. A single case each of perforation of the ileum and a duodenal diverticulum was reported. These cases revealed gas dissecting into the retroperitoneum, abdominal and chest walls, mediastinum, neck and leg. Pathways, mechanisms, trends and differential diagnosis were reviewed.

A New Cholecystographic Agent: Iocetamic Acid. John P. Fotopoulos, M.D., and Jerold B. Weinberg, M.D., Evanston Hospital, Northwestern-McGraw Medical Center, Evanston, Illinois.

Cholebrine® was compared with Iopanoic acid (Telepaque®) as regards radiodiagnostic quality, the number of repeat examinations necessary for conclusive diagnosis, and number of side effects. Of 255 Cholebrine cholecystograms, only 3.5 per cent demonstrated improved visualization with repeat study. Side effects, particularly diarrhea, were very low with 2 per cent of of 202 patients interviewed reporting poor tolerance.

New Thoughts on Xanthogranulomatous Pyelonephritis (XP). Stephen Gammill, M.D., Bob Peace, M.D., Elliot Himmelfarb, M.D., and Jack Rabinowitz, M.D., University of Tennessee Medical School, Memphis, Tennessee.

The authors demonstrated why they believe that XP forms no neovascularity, but rather rearranges the normal renal vascularity and produces an appearance that may be confused with but distinguished from tumor vascularity. This was accomplished by presenting the clinical, angiographic and pathologic findings of 12 patients (ages 14–63) with XP. The angiographic findings were compared with findings in patients with renal carcinoma, renal abscesses and proliferative pyelonephritis which was not XP.

A Spectrum of Duodenal Abnormalities. Francis J. Hahn, M.D., and Rolf L. Schapiro, M.D., University of Iowa Hospitals, Iowa City, Iowa.

Relatively uncommon lesions occurring in the duodenum include both benign and malignant diseases; several of these produce characteristic morphologic features which permit specific roentgenologic diagnoses with a high degree of certainty. Duodenobiliary fistulae are usually attributable to primary biliary disease, rarely due to peptic ulcer disease. Aortoduodenal

fistulae usually represent a late sequel of reconstructive aortic surgery, but occasionally occur as a complication of atherosclerotic aortic aneurysm. Hematomas characteristically produce a submucosal lesion in the distal descending duodenum and may occasionally constitute an element of child abuse. Dilatation of the duodenum is encountered proximal to benign strictures, obstructing neoplasms, in scleroderma, and in the superior mesenteric artery syndrome. Intraluminal diverticula may cause intermittent pain and vomiting and render a pathognomonic roentgenographic appearance.

Little difficulty is encountered in recognizing the malignant features of primary duodenal carcinoma, lymphoma, or ampullary carcinoma, although distinction between these specific neoplasms is not always possible. Benign neoplasms present as an intraluminal filling defect, frequently movable over short distances.

Roentgenologic Evaluation of Acute Cervical Spine Injuries in the Emergency Room. Sang Yeung Han, M.D., David F. Powell, M.D., and David M. Witten, M.D., University of Alabama School of Medicine, Birmingham, Alabama.

Essentials of roentgenologic examination of patients with acute cervical spine injuries in the emergency room were presented with emphasis on the tomogram and oblique anteroposterior view of the cervical spine. Injuries were classified according to the mechanism (hyperflexion, hyperextension, and rotation) and anatomic levels.

The tomogram of the upper cervical spine in the anteroposterior projection is most useful for rotational injuries of the atlantoaxial joint and bursting fracture of the atlas (Jefferson fracture).

The oblique anteroposterior view is valuable in demonstrating fracture and dislocation of the articular facets of the lower cervical spine.

Acute Septic Arthritis. Norma F. Harris, M.D., J. Frederick Johnson, M.D., and Robert S. MacIntyre, M.D., Baylor College of Medicine, Houston, Texas.

Representative cases from over 80 cases reviewed at Ben Taub General Hospital were selected for presentation, in addition to a limited amount of statistical data with respect to age, sex, site of organisms, clinical presentation and course. The cases were chosen to illustrate the radiologist's role in the diagnosis of acute septic arthritis, and stressed the early signs and the appearance of specific infectious agents, as well as the complications. The fact that these cases presented within a 4 year period indicated that this is still a diagnostic and therapeutic problem.

Detection and Evaluation of Localization Error Using Therapy Verification Film Technique. Arthur G. Haus, M.D., James E. Marks, M.D., Melvin L. Griem, M.D., and Kurt Rossmann, Ph.D., Department of Radiology, University of Chicago, Chicago, Illinois.

The therapy verification film technique makes use of a film that can be left under the patient during each treatment to give an accurate record of the anatomy included within the treatment volume. In this Exhibit was demonstrated: (1) how various exit dose exposures fit the characteristic curve of the film; (2) types of errors commonly detected due to physician, technician, and patient; (3) the calibration technique for determining the variation of exit dose over the treatment field; (4) sequential verification films, taken during the entire course of treatment, illustrating daily alterations in localization and incidence of localization error throughout the treatment series; and (5) data from localization error studies involving treatment of the pelvis, head and neck, and treatment fields used for Hodgkin's disease and malignant lymphoma.

Pediatric Hip Diseases. J. Bruce Hauser, M.D., and Robert T. Bramson, M.D., Washington University School of Medicine, Mallinckrodt Institute of Radiology, St. Louis, Missouri.

A study of a variety of children's hip diseases was presented including: congenital hip dislocation; Legg-Perthes dis-

ease; slipped capital epiphysis; septic hip; congenital hip dysplasias such as metatrophic dwarfism, multiple epiphyseal dysplasia, and others; idiopathic coxa vara; fibrous dysplasia; and other bone softening diseases. Roentgenograms were analyzed to provide clues to sort out the differential features of the multiplicity of hip diseases. Examples of the subtle and early features of many of these diseases were shown.

Extra-Ventilatory Air Syndromes of the Premature Infant. Carl G. Hoffman, M.D., and Patrick D. Lester, M.D., University of Utah Medical Center, Salt Lake City, Utah.

With the increased early survival of premature infants, more extra-ventilatory air syndromes are being seen, especially in Newborn Intensive Care Units where assisted respiration is commonly utilized. Their early recognition, including interstitial emphysema and pneumopericardium, is often critical in the management of these seriously ill infants. Examples of the spectrum at presentation and in resolution were demonstrated.

Retention of Small Foreign Objects in the Duodenum: a Sign of Incompletely Obstructing Duodenal Anomalies. E. George Kassner, M.D., Judith Rose, M.D., Peter K. Kottmeier, M.D., Morton Schneider, M.D., and Glenn M. Gallow, M.D., Departments of Radiology and Surgery, (Division of Pediatric Surgery), State University of New York, Downstate Medical Center, Brooklyn, New York.

According to several large published series, small round, oval or cuboidal foreign objects nearly always pass promptly through the gastrointestinal tract. Stasis of such objects in the stomach or duodenum is extremely uncommon. The Exhibitors have observed prolonged retention of such objects in 3 children who had no clinical or plain roentgenographic evidence of partial duodenal obstruction. In each, barium meal demonstrated a congenital anomaly of the duodenum: duodenal stenosis in a 9 year old boy; redundant duodenal diaphragm ("windsock duodenum") in a 3 year old girl; and annular pancreas in a 4 year old girl.

Hypotonic Colonic Examination. James Magner Keegan, M.D., Leslie Louis Lemak, M.D., Charles Wilbur Yates, Jr., M.D., Patricia Ann Athey, M.D., and Victor Carlson, M.D., St. Joseph Hospital, Houston, Texas.

Barium enema examinations have provided much diagnostic information, usually with little discomfort to the patient. However, many patients, especially those with colonic pathology, have severe spasm preventing diagnostic filling of the colon. The use of intramuscular Pro-Banthine (30–60 mg.) has proved to be a safe and effective means of alleviating spasm. This often allows evaluation of both ends of a lesion and permits more adequate examination of mucosal detail. Cases illustrating the use of Pro-Banthine with various types of colonic pathology were presented with premedication and postmedication barium enema roentgenograms.

Gas Myelography with Hypocycloidal Tomography. Senichiro Komaki, M.D., Marlen F. Junck, M.D., Henry J. Bradley, M.B., and Yong W. Kim, M.D., The Medical College of Wisconsin, Milwaukee County General Hospital, Veterans Administration Hospital, Milwaukee, Wisconsin.

Gas myelography offers a number of advantages over pantopaque myelography. When combined with hypocycloidal tomography, diagnostic films of high quality are usually obtained, demonstrating a number of lesions exceptionally well. Fifteen cases of cervical, thoracic, and lumbar pathologic entities were presented. These included examples of neoplastic, traumatic, disk, infectious, congenital, and miscellaneous lesions.

A Study of the Patterns of Cancer Care in Radiation Therapy. Simon Kramer, M.D., and William E. Powers, M.D., Department of Radiation Therapy and Nuclear Medicine, Thomas Jefferson University Hospital, Philadelphia, Pennsylvania, and Mallinckrodt Institute of Radi-

ology, St. Louis, Missouri.

A 3 year study on the Patterns of Cancer Care in Radiation Therapy is being conducted under the auspices of the American College of Radiology through a grant from the Division of Cancer Control of the National Cancer Institute. Under the direction of Simon Kramer, M.D., Chairman of the Department of Radiation Therapy and Nuclear Medicine at Thomas Jefferson University Hospital, Philadelphia and with technical assistance provided by David F. Herring, Inc., La Jolla, a nationwide survey of radiation therapy facilities will be conducted. The study will direct itself to problems of the quality and accessibility of radiation therapy care, manpower needs, financial reimbursement policies, training programs, etc. The first step undertaken in the study has been the establishment of a master list of radiation therapy facilities throughout the United States. At the same time, committees of radiation therapists representing all geographic sectors of the country and practicing radiation therapy in all types of settings have developed questionnaires concerning treatment of specific disease sites with radiation therapy. These questionnaires will be applied to the review of a small number of appropriate medical records in 150 facilities per year, chosen on the basis of a statistically derived sampling design. In each case the sampling will be so limited as to make individual assessment an impossibility, but the over-all picture obtained should delineate the patterns of radiation therapy care presently prevailing in the United States. The audiovisual Exhibit describing this study consisted of colored slides and a narration by William Powers, M.D., a member of the executive committee for the Patterns of Care Study.

Intracoronary Rubidium 81 for Determining Myocardial Scar versus Ischemic Muscle. David A. Krause, M.D., Merle K. Loken, M.D., and Kurt Amplatz, M.D., University of Minnesota Hospitals, Minneapolis, Minnesota.

$Rb^{81}Cl$ injected into the left coronary artery of patients showed excellent differentiation between ischemic, but viable, myocardium and scar. Coronary arteries may be present in areas of scar on good angiograms. $Rb^{81}Cl$ does not appear significantly in scar because of the relatively few cells in fibrous tissue. This technique is useful prior to deciding on coronary bypass surgery, as it helps prevent bypassing a stenotic lesion into scarred tissue.

Inflammatory Lesions of the Duodenal Loop. Arthur F. Kriner, M.D., Reed P. Rice, M.D., and William M. Thompson, M.D., Duke University Medical Center, Durham, North Carolina.

The roentgenologic diagnosis and differentiation of inflammatory lesions of the duodenal loop were stressed in this Exhibit.

The spectrum of duodenal changes associated with pancreatitis varies from a subtle mass effect to gross distortion with complete duodenal obstruction. These changes were compared to annular duodenal spasm and/or stricture resulting from postbulbar ulcer. Regional enteritis of the duodenum can mimic other inflammatory lesions, and in at least half of the Exhibitors 23 patients there was smooth narrowing of the first and second portions of the duodenum which is virtually pathognomonic of regional enteritis. Rarer inflammatory lesions were included for comparison.

Systematized Approach to the Roentgenographic Diagnosis of Asymptomatic Space-Occupying Lesions of the Kidney. Erich K. Lang, M.D., Eugene C. St. Martin, M.D., Hoyet L. Chance, M.D., Raymond W. Turner, M.D., Burdette E. Trichel, M.D., and Don L. McCormick, M.D., LSU School of Medicine in Shreveport, Confederate Memorial Medical Center, Shreveport, Louisiana.

A sequential roentgenographic approach to the diagnosis of asymptomatic renal mass lesions was developed. The intravenous urogram, nephrotomogram, ultrasound, arteriogram, and the cyst puncture and aspiration test complex were grouped in a hierarchical fashion to permit progres-

sive affirmation of the diagnosis. The confidence level and accuracy of these various diagnostic tests and their contributions to the final diagnosis were statistically analyzed on a series of 498 patients. Appropriate sequential deployment of the above battery of roentgenographic examinations guarantees diagnosis of benign renal cysts, inflammatory renal mass lesions, asymptomatic neoplastic lesions, and congenital variance provides an adequate confidence level at minimum cost in monetary expense and risk to the patient.

Legg-Perthes Disease: a Prognostic Roentgenographic Classification. Patrick D. Lester, M.D., Jay Lukins, M.D., and Sherman S. Coleman, M.D., University of Utah Medical Center, Salt Lake City, Utah.

Legg-Perthes disease has classically been staged according to the state of healing on the roentgenogram, *e.g.*, "stage of fragmentation," etc. This is useful in approximating the evolution of the disease but cannot be used to prognosticate results or determine treatment. The Catterall classification (4 groups), based upon the extent of involvement of the femoral head on the initial roentgenogram (regardless of pathologic stage), can accurately indicate the eventual outcome. Deformity (or lack of deformity) in the healed stage can be predicted. The grouping can be of value in determining the method of treatment.

Roentgenographic Recognition of Characteristic Bronchial Patterns. Robert Levitt, M.D., John V. Forrest, M.D., and Stuart S. Sagel, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

The plain film roentgenographic findings in bronchiectasis, when characteristic, are well known and easily recognized. Other forms of local bronchial disease may also have typical plain film roentgenographic, tomographic and bronchographic appearances. Clinical and roentgenographic correlation of findings in mucus impaction due to allergic aspergillosis in asthmatics will allow appropriate treatment. The use of xenon ventilation scans to document air trapping behind bronchial atresia was also emphasized. The radiologist often can distinguish all these unusual bronchial shadows from neoplasms and so help avoid unnecessary surgery.

Computerized Transverse Axial Tomography (EMI): Some Observations on Deep and Midline Mass Lesions. Thomas Lott, M.D., and Taher El Gammal, F.F.R., University Hospital and Medical College of Georgia, Augusta, Georgia.

To demonstrate the usefulness of the EMI scanner in evaluation of patients with midline and posterior fossae masses, the EMI scans were compared to conventional neuroradiologic studies.

Specific tissue diagnosis with the EMI scanner was presented, as in intracerebral and intraventricular hematoma and lipoma of the corpus callosum.

The value of intravenous contrast medium injection as an adjunct to the routine EMI scan was demonstrated.

Hypertelorism. Roderick Ian Macpherson, M.D., Robert Wan, M.D., and Martin Howard Reed, M.D., Health Sciences Children's Centre, Winnipeg, Manitoba, Canada.

Hypertelorism means an abnormal increase in the distance between the eyes. Its presence can be determined roentgenologically by measurement of interorbital distance and comparison with normal standards.

There are a variety of mechanisms by which hypertelorism may develop; for instance, it can be associated with midfacial defects (*i.e.*, cleft palate), premature closure of sutures and diseases that increase the size of the interorbital structures.

It is a common factor in a variety of rare, and not so rare, clinical syndromes, and its recognition serves as a clue in the identification of many of these.

In this Exhibit, the method of measuring hypertelorism was demonstrated and a classification of diseases associated with hypertelorism was presented. The salient features of several of these syndromes were demonstrated with particular emphasis on their roentgenologic diagnosis. These in-

cluded craniodiaphyseal dysplasia, oto-palato-digital syndrome, hypertelorism-hypospadias syndrome, Apert's syndrome, and medial cleft face syndrome.

Cystic Adenomatoid Malformation of the Lung: Morphologic Analysis. John E. Madewell, M.D., John T. Stocker, M.D., and Jack M. Korsower, M.D., Armed Forces Institute of Pathology, Washington, D.C.

Cystic adenomatoid malformation of the lung with its consequent, and, at times severe, respiratory embarrassment, is an important roentgenographic diagnosis in the pediatric age group. The gratifying response to surgical treatment makes it imperative that the roentgenographic findings in this disease are fully understood.

The Exhibitors' experience with cystic adenomatoid malformation at the AFIP was critically reviewed and subjected to morphologic analysis for diagnostic purposes. While the correlating pathologic findings were briefly discussed, the emphasis was placed on the roentgenologic findings which most clearly define the gross parameters of this disease.

Tumors of the Hand and Wrist. Norman L. Martin, M.D., W. E. Pinke, M.D., Kyo Rak Lee, M.D., Fred F. Roberts, M.D., and James Kifer, M.D., University of Kansas School of Medicine, Departments of Diagnostic Radiology and Pathology, Kansas City, Kansas.

Tumors arising from bones of the hand and wrist are uncommon lesions. Several series of over 1,000 tumors originating in bone showed the hand to account for less than 5 per cent of the total. Soft tissue tumors such as ganglions, squamous and basal cell carcinomas, xanthomas of the tendon sheath and glomus tumors are more common in the hand and wrist but have fewer distinguishing roentgenographic characteristics than those arising from the bone. Metastatic tumors to the hand and wrist, as stated by many authors, are rare.

Because of the rarity of tumors affecting the bones of the hand and wrist, many physicians will not have seen the spectrum of tumors that can involve the hand and wrist. When tumors are primary in the hand, they offer a good chance for cure because of their location, thus failure in their recognition may delay or affect appropriate treatment and cure.

The Exhibit demonstrated tumors of the hand and wrist from the more common benign to the rare malignant and metastatic: initially, as roentgenographic unknowns with a characteristic biopsy slide specimen, with pertinent points depicted, followed by the answer and summary of the case.

The Exhibit concluded with a group of lesions many times mistaken for a tumor of the hand and wrist with the differential points noted.

Peribiliary Fat: a New Roentgenographic Finding. Harvey Ilsley Meyers, M.D., and Michael Stephen Shaub, M.D., Los Angeles County, University of Southern California Medical Center, Los Angeles, California.

Radiolucent linear densities have been observed in the right upper quadrant in the area of the liver in some individuals. Analysis of intravenous cholangiograms and oral cholecystograms and anatomic dissections indicates that these lucencies represent peribiliary fat, an extension of the extraperitoneal fat in the area of the liver. The importance of this finding is that the lucencies be recognized as peribiliary fat and not confused with gas within the biliary or portal system. These lucencies may also be of help in identifying, locating, and measuring the diameter of the common bile duct when it is only faintly visualized on an intravenous cholangiogram or when its anatomic course is atypical.

Hypotonic Colon Examination with Glucagon. Roscoe E. Miller, M.D., Stanley M. Chernish, M.D., and Jovitas Skucas, M.D., Indiana University School of Medicine, Indianapolis, Indiana.

The effect of glucagon, atropine sulfate, and glucagon plus atropine sulfate on the colon was compared to placebo in 48 double blind crossover barium enema examinations. Medications were given 10 minutes

prior to each barium examination. The number and intensity of side effects were much less with either placebo or glucagon than with atropine sulfate alone or atropine sulfate plus glucagon. For a barium enema examination, the subject is much more comfortable, the colon and small bowel more relaxed, intracolonic pressure is less, and the examination completed quicker after giving glucagon than after the administration of placebo or atropine sulfate.

Selective Temporal Arteriography in Giant Cell Arteritis Polymyalgia Rheumatica. Rogelio Moncada, M.D., Dennis Baker, M.D., Herbert Rubinstein, M.D., Dinesh Shah, M.D., and Leon Love, M.D., Loyola University of Chicago, Stritch School of Medicine, Maywood, Illinois.

The difficulty in the diagnosis of arteritis in polymyalgia rheumatica is well established. Histologic confirmation has always been attained through direct temporal artery biopsy. However, the small yield of positive biopsy findings *vs.* characteristic clinical criteria prompted a combined study of selective temporal arteriography and biopsy in the hope of establishing the presence of vascular involvement distal to the site of routine biopsy.

Twenty cases that fulfilled the clinical criteria for polymyalgia rheumatica were included in this study. The results were presented.

Cervical Aortic Arch. Rogelio Moncada, M.D., Michael Shannon, M.D., Robert Miller, M.D., Alois Hastreiter, M.D., Ira DuBrow, M.D., Sheldon Burman, M.D., Harvey White, M.D., Jamie Friedman, M.D., and Wade H. Shuford, M.D., Loyola University of Chicago, Stritch School of Medicine, Maywood, Illinois.

The anatomic development of the aortic arch and great vessels usually lends itself well to interpretation of the origin of its congenital anomalies. One of the rarest of these anomalies, the cervical aortic arch, however, has never been adequately explained embryologically. Only 15 cases have been reported in the past in the literature.

This exhibit presented 10 cases of the anomaly, 5 left-sided and 5 on the right, and 6 of which have never before been reported. The embryologic hypothesis of development and angiographic findings were stressed, as well as pathologic and clinical characteristics.

Patient and Beam Positioning System. George D. Oliver, Jr., Ph.D., Carlos A. Perez, M.D., and William E. Powers, M.D., St. John's Mercy Hospital, Division of Radiation Therapy, St. Louis, Missouri.

The technique of a new fiber glass casting system has replaced plaster of paris for patient repositioning and immobilization and electron shielding. Once the area requiring therapy has been determined, the basic cast which immobilizes the patient in the therapeutic position is molded and cured with an ultraviolet light. Pictorial examples illustrated a few of the possible uses of this system in radiation therapy.

The Water Double Contrast Barium Enema Examination: a New Method of Roentgen Examination of the Colon. Rubem Pochaczevsky, M.D., City Hospital Center at Elmhurst, and Mount Sinai School of Medicine, New York, New York.

A new technique for double contrast examination of the colon utilizing water instead of gas was described.

The study is carried out by means of a disposable prepackaged double bag enema kit utilizing a one-stage, closed system. Reliable postevacuation roentgenograms of the colon are achieved simply by passive syphoning and without the use of tannic acid.

Diaphragmatic Anomalies in the Neonate. Catherine A. Poole, M.D., and Howard A. Wexler, M.D., University of Miami, Jackson Memorial Hospital, Miami, Florida.

This Exhibit displayed the spectrum of anomalies that may involve the neonatal diaphragm. These range from the not uncommon foramen of Bochdalek hernia to a unique case of "congenital" bilateral paralysis. Included were examples of congenital and acquired eventrations with gross and microscopic correlation. In many instances

recognition of certain patterns on plain film roentgenograms of the chest and abdomen obviates the need for more elaborate examinations.

Etiologic factors and the high association of some lesions with chromosome aberrations were also discussed.

Computerized Transverse Tomography of the Brain. Barry D. Pressman, M.D., Donald R. Smith, M.D., Frank S. Jannotta, M.D., Hugo V. Rizzoli, M.D., and David O. Davis, M.D., George Washington University Medical Center, Washington, D. C.

Computerized transverse tomography is a revolutionary new method of studying intracranial disease. It is rapid, noninvasive and has no attendant morbidity. The basic principles of operation as well as the data presentation method which is utilized, were explained in detail. Interpretation of the computerized tomograms requires a knowledge of normal anatomy as depicted on the scans. To this end, normal brain sections with comparison scans were presented and normal anatomic features were delineated.

Cases were presented in several categories including tumors, infarcts, orbital lesions, abnormalities of ventricular size, posterior fossa lesions and vascular abnormalities. These were used to demonstrate the interpretation of the scans and to explain the appearance of the different lesions on the scan. Furthermore, the value of computerized tomography in the evaluation of these categories of lesions was explained. Inherent difficulties of this system as they apply to interpretation of the scans were discussed and illustrated.

How Specific is the Air-Bronchogram? a Radiologic-Pathologic Study. James C. Reed, M.D., and John E. Madewell, M.D., Armed Forces Institute of Pathology, Washington, D. C.

The air-bronchogram is one of the most confusing of the classic signs in chest roentgenology. It is considered to signal an end-air-space ("alveolar") filling process.

The objective of this Exhibit was to show that it may also occur in a variety of interstitial disease processes. A number of cases using gross and macroscopic sections correlating the roentgenologic findings was used to exemplify these situations. The diseases considered included: alveolar proteinosis; hemosiderosis; bronchioloalveolar cell carcinoma; lymphoma; pseudolymphoma; and sarcoidosis.

The Decaying Human Sacro-Iliac Joint. Donald L. Resnick, M.D., Gen Niwayama, M.D., and Thomas G. Goergen, M.D., Veterans Administration Hospital, San Diego, California.

Over 50 human sacro-iliac joints were removed *in toto* at random during routine autopsies and analyzed to define the roentgenographic and pathologic alterations occurring in middle-aged and elderly patients. Each joint was sectioned, photographed and roentgenographed; large microscopic slides of the entire joint were obtained.

The spectrum of "ankylosing" degenerative sacro-iliac disease was described and contrasted with that of ankylosing spondylitis. Although roentgenographically the 2 may simulate each other, the presence of para-articular osteophytosis leading to focal ankylosing in degenerative disease differs from the widespread intra-articular bone fusion occurring in ankylosing spondylitis.

Immunology and the Lung. Stewart R. Roberts, Jr., M.D., Hans U. Tschersich, M.D., David Frolich, M.D., and H. Stephen Weens, M.D., Department of Radiology, Emory University Affiliated Hospitals, Atlanta, Georgia.

Pulmonary disease was categorized and compared on an immunologic basis. The origin and function of antibody and cellular immunity were outlined and the 4 immune responses were illustrated.

Routine Double Contrast Upper Gastrointestinal Series Utilizing the "Gas Pill" Technique: a Review of 1,500 Consecutive Examinations. Lee Frank Rogers, M.D., Otto Munoz, M.D., and Thomas Stanley Harle, M.D., Department of Radiology, The University of Texas Medical School at

Houston, Houston, Texas.

This Exhibit demonstrated the renewed interest in double contrast techniques for the examination of the upper gastrointestinal tract. Many proposed techniques are cumbersome or poorly tolerated. Originally the double contrast technique was intended for use under special circumstances.

The Exhibitors developed a simplified technique utilizing 6 gas forming tablets, that is well tolerated and may be utilized routinely. Fluoroscopy, spot films, and standard follow-up films were employed. The visualization of the stomach and duodenum was greatly improved, smaller lesions were readily visualized and finer details were demonstrated. This presentation was based on an experience with 1,500 consecutive examinations.

Acquired and Congenital Systemic Venous Abnormalities in Children. Nancy S. Rosenfield, M.D., and Kenneth E. Fellows, M.D., Children's Hospital, Medical Center, Boston, Massachusetts.

In the inclusive period 1967–1973, *91* phlebographic examinations were performed at Boston Children's Hospital Medical Center. A variety of abnormalities were encountered and form the basis of this Exhibit.

A non-tourniquet technique of phlebography was employed in most children to study peripheral venous problems; examples of thrombophlebitis (post-traumatic and idiopathic) as well as dysplasias of the venous and lymphatic systems were provided. Special problems such as dialysis shunt thromboses and congenital venous aneurysm were also illustrated.

Obstructive abnormalities of the inferior vena cava (IVC) included tumor compression and invasion, thrombosis (usually associated with renal vein thrombosis) and constriction secondary to hepatomegaly. In the course of inferior vena cavography, a double IVC, left-sided IVC, and congenitally interrupted IVC were also encountered and were illustrated.

Choledochal Cyst: Roentgenographic Techniques. Nancy S. Rosenfield, M.D., S. Treves, M.D., and N. Thorne Griscom, M.D., Children's Hospital Medical Center, Boston, Massachusetts.

Twenty-four cases of choledochal cyst were reviewed. Only 7 were definitively diagnosed preoperatively. I^{131} rose bengal scanning led to the highest percentage of cyst visualization (3 out of 4), but the study most helpful in suggesting the diagnosis in a general way was the upper gastrointestinal series.

Abnormalities detected by plain film roentgenograms, intravenous urography and the total body opacification effect, barium studies, intravenous cholangiography, arteriography, and technetium-sulfur colloid and rose bengal scans were shown. Roentgenographic techniques of postoperative study were also illustrated.

Cholevue—a New Contrast Medium for Intravenous Cholangiography and Cholecystography. E. Nicholas Sargent, M.D., Andre Schulman, M.D., Harvey I. Meyers, M.D., Richard B. Guttler, M.D., John T. Nicoloff, M.D., and Louis DiFazio, Ph.D., Los Angeles County, University of Southern California Medical Center, Los Angeles, California.

Preliminary experience and results with the use of Cholevue (meglumine iodoxamate) for intravenous cholangio-cholecystography were presented. Meglumine iodoxamate (40 per cent) in its present formulation is lower in bound iodine content than the formulation of meglumine iodopamide (52 per cent). Extensive clinical and laboratory monitoring have shown no significant adverse effects.

Opacification of the bile ducts occurs following the end of the injection time (10 minutes) and opacification of the gallbladder occurs 30 to 60 minutes following the end of the injection. Roentgenographic visualization of the biliary ducts and gallbladder was excellent and prolonged. Further control studies comparing meglumine iodopamide to meglumine iodoxamate are being conducted, as this preliminary study seemed to indicate that the new contrast medium provides visualization

far superior to cholografin.

A Comparative Study of Home Administered Bowel Cleansing Agents for Outpatients Scheduled for Roentgenography. Jirair N. Sarian, M.D., Los Angeles, California.

Outpatient diagnostic roentgenology requires a simplified patient administered bowel preparation at home. The success previously achieved with a standardized senna concentrate powder as a single-dose evacuant for roentgenographic preparation led to the present study of a liquid extract of senna fruit.

In this study 98 patients (56 male and 42 female) ranging in age from 19 to 86 years were prepared for the various roentgenographic procedures by the administration of a prescribed dose of liquid senna extract the afternoon prior to the roentgen procedure. Data were presented on the quality of the visualization obtained and the incidence of abdominal griping and nausea.

Splenic Circulation: Normal and Pathologic. James H. Scatliff, M.D., Otis N. Fisher, M.D., and W. Bonner Guilford, M.D., University of North Carolina, School of Medicine, Chapel Hill, North Carolina.

A canine model was developed for analysis of the normal splenic angiogram and alterations produced by trauma, infarction, and simulated intrinsic and extrinsic masses.

In vivo and specimen injection studies showed two phases of splenic circulation, lymphoidal and sinusoidal. Magnification arteriography, microradiography and corrosion casts demonstrated this circulation, particularly the marginal sinus vascularity of the malpighian corpuscles. Disturbances in this circulation in the animal model as well as correlative clinical examples in the entities listed were shown.

Retroperitoneal Masses in Children (Excluding Those of Renal Origin). Robert F. Scheible, M.D., Robert T. Bramson, M.D., and William H. McAlister, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

Roentgenographic evaluation of the retroperitoneal space includes plain film roentgenography, tomography, venography and urography, retroperitoneal pneumography, and arteriography. Manifestations of retroperitoneal masses in children were presented with an emphasis on the less common disease entities.

Lesions arising from the pancreas include pseudocysts and the rare carcinoma of the pancreas. Adrenal lesions include the common neuroblastomas as well as the unusual functioning adrenal tumors. Masses of mesenchymal origin are varied, ranging from abscess and hemorrhage to unusual tumors such as lipomas, lymphangiomas, hemangiomas, rhabdomyosarcomas and lymphomas. Teratomas and anterior meningoceles are lesions of embryonic origin. Nonretroperitoneal lesions, such as hepatoblastoma may also present in the retroperitoneal space.

Early Clinical Experience with the ACTA (Automatic Computerized Transverse Axial) Tomographic Scanner. Dieter Schellinger, M.D., Giovanni DiChiro, M.D., Stewart P. Axelbaum, M.D., Homer L. Twigg, M.D., and Robert S. Ledley, Ph.D., Georgetown University Medical Center, Washington, D. C.

An alternative method of computerized axial tomography has been achieved with a new device. The ACTA scanner permits the study of any body part.

Normal and pathologic ACTA scans of the head and brain, neck and spinal cord, chest, abdomen and limbs were presented.

Stress Injuries and Developmental Changes of Lower Extremities. Harold J. Schneider, M.D., Athena Y. King, M.D., and Edward H. Miller, M.D., University of Cincinnati, School of Medicine, Cincinnati, Ohio.

Roentgenograms of lower extremities of 64 professional ballet dancers were reviewed. A variety of abnormalities were seen, some similar to those found in other athletes, but, in addition, specific abnormalities not observed in other groups were noted.

These specific findings included: cortical hypertrophy of the long limb and first 3 metatarsal bones; multiple stress fractures

of the femoral neck and tibia; and talar notch and spur. This group of findings was sufficient to identify the ballet dancer.

Improved Operative Cholangiography. Fred C. Shipps, M.D., Good Samaritan Hospital and Medical Center, Portland, Oregon.

This Exhibit was presented to stimulate radiologists to review the quality of operative cholangiography as it exists in their hospital.

It demonstrated how quality improvement in one institution preceded reduction in "missed stones," and also preceded convincing many of the surgeons of the value of routine operative cholangiography. The basic requirements of quality operating room cholangiography with a portable x-ray machine were covered. A new technique and new apparatus were presented.

Peripheral Vascular Perfusion Scanning. Michael E. Siegel, M.D., Frank A. Giargiana, Jr., M.D., Robert I. White, Jr., M.D., Buck A. Rhodes, Ph.D., Barry H. Friedman, M.D., and Henry N. Wagner, Jr., M.D., The Johns Hopkins Medical Institution, Department of Radiology and Radiological Science, Baltimore, Maryland.

Arteriography is essential in providing detailed morphologic data on large vessel disease of the extremities. With scintillation scanning of the distribution of an intra-arterial injection of radioactive albumin microspheres, one can also evaluate the physiologic effect on the relative distribution of perfusion of the arteriographically demonstrable, as well as small vessel disease.

In analysis of over 100 scans, 5 distinct patterns were noted and correlated with the arteriographic findings. They were: the normal pattern; focal large vessel disease; diffuse small vessel disease; multifocal disease; hyperemia in ischemic ulcer disease; and hyperperfusion in osseous disease of the extremity.

A summary of the conclusions was as follows: (1) perfusion scanning may be the only objective abnormality in patients with small vessel disease; (2) the presence and degree of hyperemic response found by evaluating the perfusion scan in patients with an ischemic ulcer are excellent prognostic indicators of healing ability; (3) relative perfusion distribution can be used to optimize amputation level selection; and (4) changes in relative perfusion may offer objective data for optimizing the choice of a reconstructive surgical approach.

Femoral Head Scintigraphy and Avascular Necrosis. Robert C. Stadalnik, M.D., Gerald L. DeNardo, M.D., Richard L. Riggins, M.D., Robert D'Ambrosia, M.D., and Sally J. DeNardo, M.D., Sacramento Medical Center of University of California, Davis, Sacramento, California.

Early diagnosis of avascular necrosis of the femoral head is important in reducing the morbidity of this disease. Radioisotopic techniques with a "bone-localizing" radiopharmaceutical represent a potential method for determining the vascularity of the femoral head. The use of F^{18} and a commercial scintillation camera with positron detector in coincidence mode for tomographic effect permitted the detection of radioactivity in the femoral head of dogs and human patients. Thirteen patients had acute fracture of the femoral neck, and 4 patients had painful arthritis due to an earlier fracture. Scintigraphy showed absent radioactivity in the femoral head of 10 patients, normal radioactivity in 5, and increased radioactivity in 2 patients. These results correlated with the results of histology and tetracycline fluorescent labeling in all instances and confirm that F^{18} distribution is an indicator of blood flow to bone. Normal or increased radioactivity indicated the presence of blood flow, decreased or absent radioactivity indicated an absence of blood flow. Random studies with F^{18} using the scintillation camera with parallel or pinhole collimator, proved to be less satisfactory than the scintillation camera with positron detector. Current studies with the scintillation camera with low energy converging collimator and Tc^{99m} diphosphonate suggest that the latter method is the simplest and as ac-

curate as the former methods.

Spectrum of Roentgenographic Findings in Antibiotic-Related Pseudomembranous Colitis. Robert J. Stanley, M.D., G. Leland Melson, M.D., and Francis J. Tedesco, M.D., Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

The Exhibit illustrated the diagnostic roentgenographic features of antibiotic related pseudomembranous colitis and the gross and colonoscopic appearance. Roentgenographic techniques designed to enhance the visualization of the characteristic mucosal lesions were included. Data from a prospective study on 200 patients administered a course of cleocin were included. Ten per cent developed biopsy proven pseudomembranous colitis. When the antibiotic was stopped early in the course of diarrhea, the clinical course improved over a 7 day period. If the course of antibiotic was completed or the antibiotic was continued after diarrhea had started, the patient's course was protracted and complicated by secondary hypokalemia, volume depletion, and hypoalbuminia. No deaths were encountered.

Pseudomembranous colitis is a self limiting disease when detected early and when the antibiotic is stopped immediately.

Roentgenology of Congenital Vascular Malformations of the Extremity. Tom W. Staple, M.D., and Johnny Bliznak, M.D., Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

The Exhibitors reviewed their experiences with 18 cases of congenital vascular malformations of the extremity. They were classified to offer a systematic approach to roentgenologic diagnosis.

The roentgenologic classifications included: (1) localized arteriovenous malformation; (2) diffuse arteriovenous malformation; (3) small vessel malformation; and (4) venous malformation. The first 3 groups required arteriography and the last venography. Historical and physical findings needed to choose correct filming and injection techniques were listed and illustrated with examples. Surgical and clinical follow-ups were provided.

Computer Screening of Brain Scans. Richard S. Steffens, Jr., M.D., James L. Lehr, M.D., Lewis J. Garrotto, M.D., Thomas A. Verdon, M.D., and Mary M. Kazmierzsak, University of Missouri-Columbia, Columbia, Missouri.

The Exhibitors studied 185 patients representing 712 separate scintigrams in evaluating the computer as an aid to the physician in brain scan interpretation. These were digitized by a SEL 840 A dissector camera and processed with an IBM 360-50 computer. Intensity histograms were constructed, the brain area was evaluated by combined chain code and unique region techniques. Lesions were evaluated by area, centroids and location. The final product was a view-by-view printout of the computer's interpretation. In detection of positive cases, physician accuracy was 71 per cent and computer accuracy was 66 per cent. Combined accuracy was 90 per cent. Utilization of the computer for a "second look" maximized brain scan detection efficiency.

A New Cardiac Pacemaker and Valve Identification Chart. Robert M. Steiner, M.D., Dryden P. Morse, M.D., Gerald Lemole, M.D., and Joel H. Shapiro, M.D., Institution Deborah Heart and Lung Center, Browns Mills, New Jersey.

Pacemaker Identification and Cardiac Valve Identification Charts were presented. The Exhibitors believe that this material is of great value to the clinician and the radiologist in proper identification of the type of cardiac valves and pacemakers on the plain film chest roentgenogram. Because of the mobility of our population, many patients are seen after the valve or pacemaker has been implanted. These charts would facilitate the early identification of the type and character of the pacemaker or valve in such patients.

Renal Tumor Diagnosis by Contrast Medium Spread Patterns Following Antegrade Injection. G. Melvin Stevens, M.D.,

and Roger J. Jackman, M.D., Palo Alto Medical Clinic, Palo Alto, California.

Contrast medium spread patterns following antegrade injection of renal tumors provide a reliable method of differentiation from renal cysts and normal parenchyma. This method is particularly useful for the identification of avascular or relatively avascular tumors, where angiography and nephrotomography are of limited usefulness. Evidence refuting the potential hazard of tumor dissemination was discussed. Typical contrast material spread patterns in a variety of benign and malignant neoplasms, hemorrhagic renal cysts, normal parenchyma and perinephric tissue were demonstrated. Experience with this technique consisted of *in vivo* or specimen injections of 30 renal tumors.

Mass Screening for Early Breast Cancer. Philip Strax, M.D., Guttman Institute, New York, New York.

Results of mass screening involving over 40,000 examinations from 1971 to 1973 for earlier detection of breast cancer were presented. Examinations involved a tandem approach of interview, clinical examination, mammography and thermography. The contribution of each modality to the yield was evaluated.

The advantages of periodic examination and the potential of mass screening programs for breast cancer were stressed.

Instrumental Perforation of the Esophagus. Michael A. Sullivan, M.D., B. Eugene Berry, M.D., and William A. Ferrante, M.D., Ochsner Clinic, New Orleans, Louisiana.

Perforation of the esophagus during medical instrumentation is becoming more and more frequent with the increasing variety of endoscopic equipment. Certain roentgenographic findings which predispose or make perforation more likely were demonstrated and the radiologist was encouraged to report them so that the endoscopist could be forewarned.

The various roentgenographic manifestations of instrumental perforation of the esophagus were demonstrated and the importance of the different types of examinations and procedures in diagnosing instrumental perforation of the esophagus were stressed.

If the diagnosis of instrumental esophageal perforation is made quickly and the site of perforation demonstrated, surgical correction can include a definitive repair of the underlying primary esophageal abnormality.

The Neonatal Chest (Uncommon Findings in Common Disease). Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D., Department of Radiology, University of Texas Medical Branch, Galveston, Texas.

Common conditions such as hyaline membrane disease, TRDN—wet lung syndrome, lobar emphysema, and pneumothorax, etc., can present with unusual and puzzling roentgenographic findings. Often these are unexpected and may be so puzzling that diagnosis is delayed or never made.

Among the problems covered in this Exhibit were: (1) which bubbles in hyaline membrane disease represent interstitial emphysema and which represent dilated alveolar ducts and terminal bronchioles?; (2) the varied patterns of congenital lobar emphysema, hyperlucent, opaque, and cystic; (3) the varying patterns of the TRDN—wet lung syndrome, classic streaky lungs, nodular pneumonia-like lungs; (4) congenital lobar emphysema presenting as pneumothorax; (5) the hyperlucent hemithorax and cloaking signs of pneumothorax; and (6) patent ductus arteriosus in premature infants, with and without respiratory disease.

The Complications and Causes of Cardiac Pacemaker Failure. Charles John Tegtmeyer, M.D., University of Virginia, Medical Center, Charlottesville, Virginia.

Surgical implantation of a pacemaker to stimulate the heart is a well-established procedure. After a pacemaker has been installed, the physician's attention is directed not only to the patient's illness, but also to the frailties of the pacemaker.

Roentgenograms are a valuable resource

which can be used to predict pacemaker failure and to detect its causes. Roentgenograms depicting the complications and causes of pacemaker failure were presented.

Anal-Rectal Malformations and the Radiologist. William M. Thompson, M.D., Michael Oliphant, M.D., and Herman Grossman, M.D., Duke University Medical Center, Durham, North Carolina.

Patients with congenital ano-rectal malformations have been perplexing problems to pediatricians, surgeons, and radiologists. The information needed in these patients is to know the level at which the gastrointestinal tract ends, and the status of the urinary tract and spine.

A simplified combined clinical and roentgenographic approach was illustrated.

If the gastrointestinal tract opens on the perineum, in the raphe of the scrotum or penis, or the vestibule of the introitus, the opening can be visualized by physical examination. These lesions are surgically treated by a "cut back" or a perineal procedure. All other lesions require colostomies and, at a later date, repair by an abdominal perineal procedure.

The value and limitations of inverted abdominal roentgenograms, retrograde urethrograms, vaginograms, and fistulograms were illustrated. The types of genitourinary and spine abnormalities were discussed.

Male Gonadal Shielding in Diagnostic Radiology. John C. Villforth, M.S., William S. Cole, M.D., Joseph S. Arcarese, M.S., and Samuel Sperling, M.S., Bureau of Radiological Health, Food and Drug Administration, Rockville, Maryland.

The use of gonadal shielding is an important radiation protection technique, intended to reduce unnecessary radiation exposure of the gonads of patients from certain diagnostic roentgenologic procedures.

The Exhibit covered the significance of roentgen-ray exposure to the gonads; which diagnostic examinations should include gonadal shielding; what types of gonadal shields are available; and how effective are they in reducing patient exposure?

Population Exposure to X-Rays, 1964 and 1970 Studies. J. C. Villforth, W. S. Cole, P. Roney, and S. Sperling, Bureau of Radiological Health, Rockville, Maryland.

Infraglottic and Tracheal Lesions in Infants and Children. Alfred L. Weber, M.D., and Sandra Sue Kramer, M.D., Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital, Boston, Massachusetts.

This Exhibit presented infraglottic and tracheal lesions seen at the Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital in the last several years. The different types of examinations done for evaluation of the trachea and larynx were presented.

Lesions shown were congenital tracheal stenosis; traumatic stenosis; granuloma; and benign tumors such as subglottic hemangioma and fibroma. Lesions that compress the trachea extrinsically such as vascular anomalies and tumors were presented.

The Empty Orbit. John Powell Williams, M.D., P. Noel Connaughton, M.D., Henry F. W. Pribram, M.D., and John B. Hearn, M.D., University of Maryland Hospital, Baltimore, Maryland.

The empty orbit or blank eye refers to that roentgenographic appearance caused by absence of all or part of the posterior orbital wall. Despite absence of the usual landmarks and a decrease in bone, the involved eye may appear increased in density due to proptosis. This appearance is classically seen in the bony dysplasia of neurofibromatosis, but may result from malignant or benign intraorbital or intracranial tumors and some systemic diseases.

The Exhibitors listed many causes of the empty orbit, the etiology of which depends upon biopsy, previous history (including surgical history), and trauma.

Macrophalangeal Fibrolipomatosis (M.P.F.L.). Issa Yaghmai, M.D., and Frank McKowne, M.D., Medical College of Virginia, Richmond, Virginia.

This condition has previously been described as macrodactyly, megalodactyly, macrodystrophia lipomatosa, dactylomeg-

aly and local gigantism. M.P.F.L. is a form of macrodactyly noticed soon after birth and usually affects multiple adjacent digits. Roentgenograms show bony enlargement mainly in phalangeal regions with thickening soft tissue predominantly of fat density mixed with bands of denser connective tissue. The etiology is obscure but may be related to fibro-fatty tumors impinging on nerves supplying the enlarged digits which are often found at surgery. It is a benign condition of unknown etiology that can be diagnosed easily by plane roentgenograms. The term M.P.F.L. describes the findings roentgenographically and pathologically.

Normal Variations and Lesions of the Sphenoid Sinus. Heun Y. Yune, M.D., Robert Holden, M.D., and John A. Smith, M.D., Indiana University Medical Center, Indianapolis, Indiana.

The sphenoid sinus is involved in various anomalies and diseases in both primary and secondary fashion. A wide spectrum of positive roentgenographic signs is associated with many of these conditions, some of which are fairly characteristic of the lesion.

A collection of these positive roentgenographic signs in common and rare lesions was presented.

General Case of the Day. G. Melvin Stevens, M.D., and Roger J. Jackman, M.D., Palo Alto Clinic, Stanford University, Stanford, California.

A challenging adult roentgenologic diagnostic problem was presented daily, with awards for correct diagnoses posted on the following days.

Pediatric Case of the Day. John Gwinn, M.D., and Fred A. Lee, M.D., Children's Hospital, Los Angeles, California.

A different pediatric case was presented each day. The history and pertinent roentgenograms permitted a single diagnosis. The answer was given the following day with a discussion and color demonstration of the pathology. A reward was given to all correct diagnosticians.

James F. Martin, M.D.
Chairman, Committee on Scientific Exhibits

THE TECHNICAL EXHIBITS

The Technical Exhibits of the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, were held at the San Francisco Hilton Hotel, San Francisco, California, September 24–27, 1974.

The individual displays were prepared with utmost care and they represented a dazzling array of the most recent modifications and improvements made in radiologic equipment and important ancillary devices, thereby enhancing immeasurably the attractiveness of the Annual Meeting as a whole.

The Society wishes to express sincere thanks to the following firms which participated in this year's Technical Exhibits:

AGA Corporation, Secaucus, New Jersey; Atomic Energy of Canada Limited, Ottawa, Ontario, Canada; Barnes-Hind Pharmaceuticals Inc., Sunnyvale, California; Bar-Ray Products, Inc., Brooklyn, New York; Becton-Dickinson, Rutherford, New Jersey; CGR Medical Corporation, Baltimore, Maryland; Cook, Incorporated, Bloomington, Indiana; Cordis Corporation, Miami, Florida; Coreco Research Corporation, New York, New York; Lester A. Dine Inc., New Hyde Park, New York; Du Pont Company, Wilmington, Delaware; Eastman Kodak Company, Rochester, New York; Eimac, Division of Varian, Salt Lake City, Utah; Elecath, Rahway, New Jersey; Elema-Schonander, Inc., Elk Grove Village, Illinois; Encyclopaedia Britannica, Inc., Chicago, Illinois; Eureka X-Ray Tube Company, Chicago, Illinois; E-Z-EM Company, Inc., Westbury, New York; Flow Pharmaceuticals, Inc., Palo Alto, California; Fluitron Company, Elyria, Ohio; GAF Corporation, New York, New York; General Electric Company, Medical Systems Division, Milwaukee, Wisconsin; Grumman Health Systems, Bethpage, New York; Hewlett-Packard, McMinnville, Oregon; Ilford Inc., Paramus, New Jersey; Katum Corporation, Longmont, Colorado; Kilgore International, Coldwater, Michigan; Liebel-Flarsheim Company, Cincinnati, Ohio; J. B. Lippincott Company, Philadelphia, Pennsylvania; LogEtronics Inc., Springfield, Virginia; Low X-Ray, Valhalla, New York; Machlett Laboratories, Inc., Stamford, Connecticut; Mallinckrodt, St. Louis, Missouri; Medi-Physics, Inc., Emeryville, California; Medi-Tech Division, Cooper Scientific Corporation, Watertown, Massachusetts; Medrad, Incorporated, Pittsburgh, Pennsylvania; Metrix, Inc., Denver, Colorado; C. V. Mosby Company, St. Louis, Missouri; N & H Instruments, Inc., Carrollton, Texas; Neutron Products, Inc., Dickerson, Maryland; Pako Corporation, Minneapolis, Minnesota; Philips Medical Systems, Inc., Shelton, Connecticut; Picker Corporation, Cleveland, Ohio; Procter & Gamble Dist. Company, Cincinnati, Ohio; The Purdue Frederick Company, Norwalk, Connecticut; S & S X-Ray Products Inc., Brooklyn, New York; Sakura Medical Corporation, Elmwood Park, New Jersey; W. B. Saunders Company, Philadelphia, Pennsylvania; Frank Scholz X-Ray Corporation, Needham Heights, Massachusetts; Siemens Corporation, Iselin, New Jersey; Spectrum X-Ray Corporation, Westville, New Jersey; E. R. Squibb & Sons, Inc., Princeton, New Jersey; Technical Innovator, Clifton, New Jersey; Charles C Thomas, Publisher, Springfield, Illinois; The Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Xcelray, Sacramento, California; Xerox Corporation, Pasadena, California; Year Book Medical Publishers Inc., Chicago, Illinois.

NEWS ITEMS

AMERICAN ROENTGEN RAY SOCIETY
Elects New Officers

At the Seventy-fifth Annual Meeting of the American Roentgen Ray Society, held at the San Francisco Hilton Hotel, San Francisco, California, September 24–27, 1974, the following officers were elected for 1974–1975: *President*, Colin B. Holman, M.D., Rochester, Minn.; *President-Elect*, Ted F. Leigh, M.D., Atlanta, Ga.; *1st Vice-President*, Seymour F. Ochsner, M.D., New Orleans, La.; *2nd Vice-President*, Richard H. Marshak, M.D., New York, N. Y.; *Secretary*, James Franklin Martin, M.D., N. C. Baptist Hospital, Winston Salem, N. C. 27103; *Treasurer*, J. D. Calhoun, M.D., 500 S. University Ave., Little Rock, Ark. 72205.

Chairman, Executive Council, Melvin M. Figley, M.D., University Hospital, Seattle, Wash. 98195.

New Member, Executive Council, J. P. Tampas, M.D., Burlington, Vt.

Seventy-sixth Annual Meeting, Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

SOCIETY FOR PEDIATRIC RADIOLOGY

The Society for Pediatric Radiology at its Annual Meeting has elected the following officers for 1974–1975:

President: John L. Gwinn, M.D., Los Angeles, Calif.; *President-Elect*: Lawrence A. Davis, M.D., Louisville, Ky., *Secretary-Treasurer*: John P. Dorst, M.D., The Johns Hopkins Hosp., Baltimore, Md. 21205.

The next Annual Meeting will be held at the Marriott Hotel, Atlanta, Ga., September 29–30, 1975.

PRESIDENT'S AWARD OF THE AMERICAN ROENTGEN RAY SOCIETY

The American Roentgen Ray Society will sponsor an annual award for the most outstanding paper submitted on a clinical application of radiology. The resident may be the sole author or may work in conjunction with others; in the latter case, it is expected that he will have performed the majority of the work and be the senior author. The paper will be presented at the Annual Meeting of the American Roentgen Ray Society and will be submitted for publication to the AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED. The paper should not exceed 5,000 words and illustrations should be limited to 10.

The winner will be presented with a certificate entitled "The President's Award" and receive an honorarium of $1,000.

Three copies of the paper and illustrations should be submitted no later than May 1, 1975, to M. Paul Capp, M.D., Chairman, Research and Educational Committee, American Roentgen Ray Society, University of Arizona School of Medicine, Tucson, Arizona 85721.

The winner will be announced by July 15, 1975. Manuscripts of unsuccessful candidates will be returned.

NEWBORN RADIOLOGY SEMINAR/ASPEN

The University of Colorado School of Medicine is pleased to announce the Third Annual Newborn Radiology Seminar to be held at the Given Institute of Pathobiology in Aspen, Colorado, February 10–15, 1975.

The Seminar covers all aspects of Neonatal Radiology, including the latest advances in the field. The Guest faculty includes: Drs. George R. Barnes, Jr., John L. Gwinn, and Andrew K. Poznanski, plus other participants.

The Seminar is approved for Category 1 credit for the A.M.A. Physicians Recognition Award.

Reservations and further information may be obtained by writing to: The Office of Postgraduate Medical Education, University of Colorado School of Medicine,

4200 East 9th Avenue, Denver, Colorado 80220.

ASPEN SEMINAR ON DIAGNOSTIC RADIOLOGY OF THE URINARY TRACT

The Fourth Seminar on Uroradiology, sponsored by the Department of Radiology, Louisiana State University School of Medicine in Shreveport, will be held at Paepcke Auditorium, Aspen Institute for Humanistic Studies, Aspen, Colorado, February 23–27, 1975.

The Guest Faculty includes Drs. John A. Evans, Leon Love, Tom Meaney, Fatollah K. Mostofi, Alphonse Palubinskas, Richard C. Pfister, Harry Schoenberg, Russell Scott, Keith Waterhouse, Willet F. Whitmore, Jr., and the Faculty of the Departments of Radiology and Urology, LSU School of Medicine, Shreveport, Louisiana.

This course qualifies as an approved program of continuing medical education. This accreditation enables the physician to qualify for 20 credit hours toward the Physicians Recognition Award of the American Medical Association. It is also acceptable for elective credit by the American Academy of Family Physicians.

For further information, please contact: The Program Director, Erich K. Lang, M.D., Professor and Head, Department of Radiology, LSU School of Medicine in Shreveport, La.

POSTGRADUATE COURSE IN NUCLEAR MEDICINE

The Eleventh Annual Postgraduate Course sponsored by the Department of Radiology, Virginia Commonwealth University-Medical College of Virginia will be held at the Williamsburg Conference Center in Williamsburg, Virginia, February 24–28, 1975.

This course will provide up-dated information on a wide variety of topics in clinical nuclear medicine. It is designed for the practicing radiologists and the interested allied physicians in other specialties. The program is accredited by the American Medical Association.

The Guest Faculty includes Drs. James J. Conway, Frank H. DeLand, Leonard M. Freeman, C. Craig Harris, Paul B. Hoffer, Henry N. Wagner, Jr., and the members of the faculty of the Medical College of Virginia.

Attendance is limited to 250 registrants, and the applications for the course must be received no later than February 1, 1975.

For further information, please contact: Gary G. Ghahremani, M.D., MCV Station, P. O. Box 728, Richmond, Virginia 23298.

RADIOLOGY/ASPEN

The Fifth Annual Aspen Radiology Conference will be held at Aspen Institute for Humanistic Studies, Aspen, Colorado, March 3–7, 1975.

The topics for discussion will include advances in cardiovascular, gastrointestinal, bone and neuroradiology involving a tri-radiological approach. Each morning session will survey the advances in a single radiology subdivision as a refresher course with independent parallel diagnostic, nuclear medicine and therapy sessions. Instructive cases, illustrating these topics and previewed by the audience, will be presented for open discussion in the afternoons.

Sessions will be held in the early morning, late afternoon and evening, allowing adequate time for leisure activities such as skiing and other winter sports.

The Faculty includes Drs. Stanley Baum, Frederick Bonte, Luther Brady, Duncan Burdick, Sam Carruthers, Marvin Daves, Gerald De Nardo, Henry Goldberg, Alexander Gottschalk, Samuel Hellman, William Hendee, Fred Hodges, Steve Holt, Eric Mayer, Will Nelp, Robert O'Mara, Theodore Phillips, Don Resnick, Philip Rubin, Bob Sanders, and John Stears.

The Conference Chairman is Dr. Emanuel Salzman, and the Conference Director is Dr. Maurice O'Connor, Denver General Hospital, University of Colorado.

Registrants are requested to arrange for their own housing. Especially selected

accommodations are being held for this conference. Reservations and information on all types of accommodations may be obtained by writing to: Reservations Manager, Post Office Box 1368, Aspen, Colorado 81611.

RADIOLOGY OF THE GASTROINTESTINAL SYSTEM

This Fifth in a series of Symposia of ACR, approved for credit, Category 1 (15 hours) will be held in Las Vegas, Nevada, March 7–9, 1975.

The Faculty includes Drs. Bernard S. Wolf, Thomas C. Beneventano, Arthur R. Clemett, Alexander R. Margulis, Richard H. Marshak, Morton A. Meyers, William B. Seaman, and Walter M. Whitehouse.

All registrations and inquiries regarding these seminars should be sent to: The American College of Radiology, 6900 Wisconsin Avenue, Chevy Chase, Maryland 20015.

OPHTHALMOLOGIC RADIOLOGY: RADIOLOGY OF THE EYE, THE ORBIT AND VISION

Columbia University College of Physicians and Surgeons will sponsor a course on radiology of the eye, the orbit and vision on Friday and Saturday, March 21 and 22, 1975.

The course will deal with the radiographic diagnosis of conditions affecting the eye and vision, including the orbit, the paranasal sinuses and the sella turcica. The role of routine radiography and special procedures including tomography, ultrasonography, angiography, venography, and dacrocystography will be presented.

The Faculty includes Drs. Coleman, Grove, Kieffer, Hui, Potter, Tenner, Tretter, Trokel, Valvassori, and Zizmor.

This course is accredited by the AMA Category 1 award for 14 hours.

For further information, please write to: Dr. Guy D. Potter, Professor of Radiology, Columbia-Presbyterian Medical Center, Director of the Course, 622 West 168th Street, New York, New York 10032.

POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY AND NUCLEAR MEDICINE

The third annual symposium on "Practical Advances in Diagnostic Radiology and Nuclear Medicine" will be held at the Turner Auditorium of the Johns Hopkins Medical Institutions on April 23–26, 1975.

The program has been approved for 29 hours of Category 1 Credit towards the certificate in Continuing Education of the American Medical Association.

The Guest Faculty includes John R. Amberg, M.D., Hillier L. Baker, Jr., M.D., Morton A. Bosniak, M.D., Marie A. Capitanio, M.D., Robert H. Freiberger, M.D., Richard H. Greenspan, M.D., Paul C. Kahn, M.D., and Manuel Viamonte, Jr., M.D.

The Local Faculty includes Martin W. Donner, M.D., Henry N. Wagner, Jr., M.D., Stanley S. Siegelman, M.D., E. James Potchen, M.D., Fred J. Hodges, III, M.D., Robert I. White, M.D., Paul S. Wheeler, M.D., A. Everette James, Jr., M.D., Bob W. Gayler, M.D., Richard H. Heller, M.D., Kook Sang Oh, M.D., Jack W. Bowerman, M.D., Donald P. Harrington, M.D., Frederick P. Stitik, M.D., and Roger C. Sanders, M.D.

The course will consist of formal lectures, a pediatric quiz session, symposia on radiologic equipment, early diagnosis of lung cancer, ultrasound-angiographic evaluation of pancreatic disease, and cardiac nuclear medicine.

Registrants will be given an opportunity to meet in small groups with the faculty during workshops in Pediatric Radiology, Orthopedic Radiology, Computerized Reporting, Cardiovascular Radiology, Chest Ultrasound, Nuclear Medicine, ENT, and Computerized Axial Tomography.

For further information, please contact: Stanley S. Siegelman, M.D., Division of Diagnostic Radiology, Johns Hopkins Hos-

pital, 601 North Broadway, Baltimore, Maryland 21205.

NEW ORLEANS RADIOLOGY SOCIETY

The New Orleans Radiology Society presents its Third Annual Spring Roentgen Conference at the Marriott Hotel, New Orleans, La., May 1–3, 1975.

The Guest Faculty includes: Chest, E. Robert Heitzman, M.D., State University of New York, Upstate Medical Center, Syracuse, New York; Pediatrics, Edward B. Singleton, M.D., St. Luke's Episcopal Hospital, Texas Children's Hospital, Houston, Texas; G. I., Arthur R. Clemett, M.D., St. Vincent's Hospital, Medical Center of New York, New York; Ultrasound, George R. Leopold, M.D., University of California, San Diego University Hospital, San Diego, California; A.C.R. Activities, Robert E. Wise, M.D., Lahey Clinic Foundation, Boston, Massachusetts; and G.U., Anthony F. Lalli, M.D., Cleveland Clinic, Cleveland, Ohio.

For further information, please contact: Coleman Schneider, M.D., General Chairman, P. O. Box 51706, New Orleans, Louisiana 70151.

BOOK REVIEW

BASIC SCIENCE PRINCIPLES OF NUCLEAR MEDICINE. Edited by Charles M. Boyd, M.D., Associate Professor of Radiology (Nuclear Medicine) and Medicine; Head, Division of Nuclear Medicine, University of Arkansas Medical Center; Chief, Nuclear Medicine Section, Veterans Hospital, Little Rock, Ark.; and Glenn V. Dalrymple, M.D., Professor and Chairman, Department of Radiology; Professor of Biometry, Physiology, and Biophysics, University of Arkansas Medical Center; Chief, Radiology Service, Veterans Hospital, Little Rock, Ark.; Professor of Medical Physics, University of Arkansas at Little Rock. Cloth. Pp. 272, with 253 illustrations. Price $21.00. C. V. Mosby and Company, 3301 Washington Boulevard, St. Louis, Mo., 63103, 1974.

As Nuclear Medicine continues to grow, there is an increasing need for a book which adequately covers the basic science of the specialty. To a considerable extent, this need has been satisfied by "*Basic Science Principles of Nuclear Medicine*" which represents the efforts of 10 staff members of the Division of Nuclear Medicine of the University of Arkansas Medical Center. The first chapter deals with principles of mathematics in a lucid manner. However, some topics included in this chapter appear to go beyond the coverage of mathematical principles as necessary for an understanding of nuclear medical physics. On the other hand, the omission of statistical considerations might be a significant oversight in a book dealing with the fundamentals of Nuclear Medicine. Chapter 2 covers elements of physics which are pertinent in a treatise of "basic principles." For some readers, the inclusion of sections on motion, relativity and quantomechanics might be considered superfluous.

The chapters on chemistry and tracer kinetics appear well conceived and appropriate. Likewise, the chapters on radiation biology and health physics are pertinent, although a closer tie to medical nuclear physics may have been worthwhile. Also, the dosimetry section may have benefited from specific examples in radionuclide dosimetry.

The chapters on electronics and nuclear instrumentation, including computer applications, are very well done.

This book is recommended reading for those desiring useful reference material relating to the basic science of nuclear medicine.

MERLE K. LOKEN, M.D., PH.D.

BOOKS RECEIVED

ATLAS OF CEREBRAL ANGIOGRAPHY WITH ANATOMIC CORRELATION. By Margaret M. Waddington, M.D., Clinical Assistant Professor of Medicine (Neurology), Dartmouth Medical School, Hanover, N. H.; with a foreword by A. Bradley Soule, M.D., Department of Radiology, University of Vermont College of Medicine. Cloth. Pp. 272, with 498 illustrations. Little, Brown and Company, 34 Beacon Street, Boston, Mass., 1974.

OCCUPATIONAL HEALTH HAZARDS. By G. Walker Daubenspeck. Cloth. Pp. 111. Price, $4.00. Exposition Press, Inc., 900 South Oyster Bay Road, Hicksville, N. Y. 11801, 1974.

RADIOLOGIC TECHNOLOGY: A FUTURE FOR YOU. By T. J. Jackson, M.H.A. Paper. Pp. 123, with some figures. Price, $3.95. The Tiresias Press, Inc., 116 Pinehurst Avenue, New York, N. Y. 10033, 1974.

RADIOLOGICAL EXPLORATION OF THE VENTRICLES AND SUBARACHNOID SPACE. By G. Ruggiero, J. Bories, A. Calabrò, G. Cristi, G. Scialfa, F. Smaltino, and A. Thibaut. Cloth. Pp. 152, with 279 illustrations. Price, $60.70. Springer-Verlag, New York, Inc., 175 Fifth Avenue, New York, N. Y. 10010, 1974.

WILLIAM DAVID COOLIDGE: A CENTENARIAN AND HIS WORK. By Herman A. Liebhafsky, General Electric Company, 1934–1967; Texas A & M University, 1967—. Cloth. Pp. 96, with some illustrations. Price, $6.95. John Wiley & Sons, Inc., Publishers, 605 Third Avenue, New York, N. Y. 10016, 1974.

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SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. James Franklin Martin, 300 S. Hawthorne Rd., Winston Salem, N. C. 27103. Annual Meeting: Marriott Hotel, Atlanta, Ga., Sept. 30–Oct. 3, 1975.

AMERICAN RADIUM SOCIETY
Secretary, Richard H. Jesse, Dept. of Surgery, M. D. Anderson Hosp. and Tumor Institute, Houston, Tex. 77025. Annual Meeting: Americana Hotel, San Juan, Puerto Rico, May 4–9, 1975.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., Dec. 1–6, 1974.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting Portland-Hilton Hotel, Portland, Oregon, March 31–April 4, 1975.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: Atlantic City, N. J., June 15–19, 1975.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following city during this year: Los Angeles, Calif., Dec. 9–13, 1974, International Hotel.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1975 was September 30, 1974.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

Application forms and further information are available from the American Board of Nuclear Medicine, 305 E. 45th St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Kenneth D. Williams, M.S., Dept. Radiol., Washington Hospital Center, 110 Irving St., N.W., Washington, D.C. 20010. Annual Meeting.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Robert W. Edland, 1836 South Ave., La Crosse, Wisc., 54601. Annual Meeting.

AMERICAN NUCLEAR SOCIETY
Program Chairman, Werner K. Kern, IRD, Space Nuclear Systems, F 309, U. S. Atomic Energy Commission, Washington, D.C. 20545.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N.W., Washington, D. C. 20037. Annual Meeting.

FOURTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
Rio de Janeiro, Brazil in 1977.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. W. A. Fuchs, M.D., Department of Diagnostic Radiology, University Hospital, Inselspital, CH-3010 Bern, Switzerland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Univ. of South Florida, Tampa, Fla.
Secretary of Congress, Dr. Alberto Mejia, Apartado Aéreo 53737, Bogotá 2, Colombia. Meeting: Feb. 16–21, 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary of Congress: Dr. Alberto Mejia, Apartado Aereo 53737, Bogotá 2, Colombia, S.A. Meeting: Bogotá, Colombia, Feb. 16–21, 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Secretary General, P.O. Box 14, 79 Lauriston Place, Edinburgh BHI IDB, Scotland, Meeting: June 22–27, 1975.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Secretary, Dr. Buenaventura U. V. Angtuaco, Congress Secretariat Headquarters 2161 Taft Ave., Manila, Philippines.
Meeting: Manila, Philippines, Nov. 10–14, 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lawrence E. Fetterman, 1720 Springhill Ave., Suite 201, Mobile, Ala. 36604. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY, CHAPTER ACR
Secretary-Treasurer, Dr. Maurice J. Coyle, 3200 Providence Ave., Anchorage, Alaska 99504.

AMERICAN OSTEOPATHIC COLLEGE OF RADIOLOGY
Annual Scientific Meeting: Americana Hotel, Bar Harbor, Fla. Program Administrator: Lawrence J. Jordan, D.O., Suite 21, Ponce American Bldg., 4601 Ponce de Leon Blvd., Coral Gables, Fla. 33146.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, John Wallace, Ph.D., Dept. Radiol., Thomas Jefferson Univ. Hosp., Philadelphia, Pa. 19107. Annual Meeting.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Robert J. Johnson, 1603 N. Tucson Blvd., Tucson 85716. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Mark M. Mishkin. The Graduate Hosp., Univ. of Pennsylvania, Philadelphia, Pa. 23rd Annual Meeting: San Diego, Calif., May 4–6, 1975.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Marshall J. Cantanzaro, Dept. of Radiol., Northside Hosp., Atlanta, Ga. 30342. Meets on fourth Thursday evenings during the academic year at a

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Maj. Marco J. DiBiase, M.D., Radiology Service, 130th Station Hosp., APO N. Y. 09102. Next meeting Dec. 5–8 in Nürnberg, Germany.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph H. Rosen, West Park Hospital, 3905 Ford Rd., Philadelphia, Pa. 19131.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosendale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joshua A. Becker, Downstate Medical Clin., 450 Clarkson Ave., Brooklyn, N. Y. 11203. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. George J. Alker, Jr., E. J. Meyer Mem. Hosp., Buffalo, N. Y. 14215. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Walter Joe Jacumin, P.O. Box 265, Rutherford College, N. C. 28671. Meets the last Thursday of every month, Holiday Inn, Morganton, N. C. at 7:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Alfred Brettner, Auburn Mem. Hosp., Auburn, N. Y. 13021. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Daniel E. Lewis, Grant Hosp., Columbus, Ohio 43215. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL TEXAS RADIOLOGY SOCIETY
Secretary-Treasurer, Dr. John M. Stoebner, Scott and White Clinic, Temple, Tex. 76501. Meets the fourth Monday of each month at Ponderosa Hotel, Temple, Tex. at 7:00 P.M.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harold J. Lasky, 55 E. Washington St., Suite 1735, Chicago, Ill. 60602. Meets third Thursday of each month, October to April, except December, at the Bismark Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Charles M. Greenwald, 7007 Powers Blvd., X-Ray Dept., Parma, Ohio 44129. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and fifth Monday of April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Gerald N. LaPierre, 759 Chestnut St., Springfield, Mass. 01107. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Richard C. Gasser, 1550 W. Rosedale, Rm. 100, Ft. Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert L. Stein, 43 Dormidera Ave., Piedmont, Calif. 94611. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Paul J. Popovich, 1350 S. Hickory St., Melbourne, Fla. 32901. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James P. Ahearn, 501 E. Buffalo Ave., Tampa, Fla. 33603. Meets on 4th Thursday of the months January, March, May, October, and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. E. P. Rasmussen, Piedmont Prof. Bldg., 35 Collier Rd., N.W., Atlanta, Ga. 30309.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER KANSAS CITY RADIOLOGICAL SOCIETY
President-Secretary, Dr. Keith W. Gallehugh, Bethany Med. Center, Kansas City, Kans. Meets 5 times a year.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert F. Conti, Holy Cross Hosp., Fort Lauderdale, Fla. 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, Dr. Donald Callahan, Northland Med. Bldg., Jennings, Mo. 63136.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Sidney B. W. Wong, Straub Clinic and Hosp., Honolulu, Hawaii 96813. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, Russell F. Cowing, P.O. Box 156, E. Weymouth, Mass. 02189. Annual Meeting.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John H. Truksa, 1605 Park Ave., Nampa, Id. 83651. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. Robert D. Dooley, Hinsdale Med. Center, Hinsdale, Ill. 60521. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. John A. Knote, 716 Carrolton Blvd., West Lafayette, Ind. 47906.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston, Jr., 1948 First Ave., N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Bernard Sams, St. Joseph Infirmary, 735 Eastern Parkway, Louisvlle, Ky. 40217. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Bruce Saxe, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Isaac Sanders, 1429 Valley View Rd. 13, Glendale, Calif. 91202. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference: Century Plaza Hotel, Los Angeles, Calif., Jan. 31–Feb. 2, 1975.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Roger H. Tutton, 1514 Jefferson Hwy., New Orleans, La. 70121.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Arnold G. Manske, P.O. Box 3008, Port Arthur, Tex. 71670.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. David S. O'Brien, Anne Arundel Hosp., Annapolis, Md. 21401.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Joseph T. Ferrucci, Jr., Massachusetts Gen. Hosp., Boston, Mass. 02114.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Thomas P. Coburn, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38163. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Bert Must, I.B.M. Bldg., Dayton, Ohio. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Francis P. Shea, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Michigan Society of Therapeutic Radiologists
Secretary-Treasurer, Dr. Delmar Mahrt, William Beaumont Hosp., Detroit, Mich. 48072. Meets bi-monthly during the academic year.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. William D. Stiehm, 37 Flower Hill Rd., Poughkeepsie, N. Y. 12603. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward L. Gieger, 969 Lakeland Dr., Jackson, Miss. 39216. Meets third Thursday, excluding summer months, at the Primos Northgate Rest., Jackson at 6:30 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. C. H. Agnew, Room 102, Doctors Bldg., Billings, Mont. 59101.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. Robert Bodmer, Suite 622 Doctors Bldg., Omaha, Neb. 68131. Meets the third Wednesday of the month, Oct. through March, at the Clarkson Hosp., Omaha, Neb. and the fourth Friday of April at the Lincoln Country Club, Lincoln, Neb.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. Charles F. Veverka, Carson Tahoe Hosp., Carson City, Nev. 89701.

N.E. Society for Radiation Oncology
Secretary, Dr. C. C. Wang, Mass. Gen. Hosp., Boston, Mass. 02114.

New England Roentgen Ray Society
Secretary, Dr. Melvin E. Clouse, 185 Pilgrim Rd., Boston, Mass. 02115. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass. at 3:00 P.M. Annual Meeting: May 16, 1975.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President. Spring Conference.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. Albert F. Keegan, 6 Secor Dr., Port Wash., N. Y. 11050.

North American Society of Cardiac Radiology
Secretary-Treasurer, Dr. Erik Carlson, Univ. Calif., San Francisco, Calif. 94143.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. Ernest B. Spangler, Wesley Long Hosp., Greensboro, N. C. 27402. Meets twice a year. The Spring Meeting is held at the time of the meeting of the North Carolina Medical Society. The Fall Meeting is held in November at the Mid Pines Club, Southern Pines, N. C.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Pablo Ramos, Box 1618, Bismarck, N. D. 58501. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of

Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Fla. 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Mark D. Reiss, 5301 F Street, Sacramento, Calif. 95819. Meets on the fourth Monday of Sept., Nov., Jan., March and May at the Mansion Inn, Sacramento, Calif.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Doug Ki Cho, 2730 Navarre Ave., Oregon (Toledo), Ohio 43616. Meets 3rd Wednesday from September to May.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Tearle L. Meyer, 175 S. Merkle Rd., Columbus, Ohio 43215.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Marvin A. Rawitch, 600 E. Washington, Santa Ana, Calif. 92701. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Craven, P.O. Box 12542, Portland, Ore. 97212. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Betty J. Wood, Dept. of Radiol., Vancouver General Hosp., Vancouver 9, B. C., Canada. Annual Meeting.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Robert B. Funch, Germantown Hosp., Philadelphia, Pa. 19144.

Philadelphia Roentgen Ray Society
Secretary, Dr. Marvin E. Ha kin, 230 N. Broad St., Philadelphia, Pa. 19102. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Klaus M. Bron, Presbyterian-Univ. Hosp., Pittsburgh 15213. Meets second Wednesday of month, September through May, at the Pittsburgh Athletic Association.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016 Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Mehdi S. Eslami, 1389 W. Main St., Waterbury, Conn. 06708.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. Daniel J. Alves, Rhode Island Hosp., Providence, R. I. 02902.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Charles E. Carter, 121 Sotoyome St., Santa Rosa, Calif. 95405. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Robert J. Bruneau, 1441 East Ave., Rochester, N. Y. 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1975.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. James R. Stewart, Oak Hills Medical Ctr., San Antonio, Tex. 78209. Meets third Wednesday of each month at Fort Sam Houston Officers Club at 6:30 P.M.

San Diego Radiological Society
President, Dr. Stanley G. Seat, 476 Prospect St., LaJolla, Calif. 92037. Meets the first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary-Treasurer, Dr. Charles A. Gooding, Univ. of Calif. Med. Ctr., San Francisco, Calif. 94122.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Albert M. Zelna, 21 Masters St., Potomac, Md. 20854. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section of Radiology, National Medical Association
Secretary, Dr. Ruby Brooks, Dept. Radiol., Veterans Adm. Hosp., P.O. Box 511, Tuskogee, Ala. 36083. Annual Meeting.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary-Treasurer, Dr. John P. Dorst, 601 N. Broadway, Baltimore, Md. 21205. Annual meeting: Marriott Hotel, Atlanta, Ga., Sept. 28–29, 1975.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. John R. Amberg, Vanderbilt University Hospital, Nashville, Tenn. 37232.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual Meeting.

South Bay Radiological Society
Secretary, Dr. John H. Callaghan, Redwood Med. Clinic, 2900 Whipple Ave., Redwood City, Calif. 94062. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society Chapter of ACR
Secretary-Treasurer, Dr. Richard T. Love, Santa Barbara Med. Clin., 215 Pesetas Lane, Santa Barbara, Calif. 93110. Meets fourth Tuesday of alternate months, Sept., Nov., Jan., March and May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Duane J. Gillum, 250 W. Pueblo St., Santa Barbara, Calif. 93105. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Annual Meeting: Grand Hotel, Point Clear, Ala., January 31–February 2, 1975.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Charles W. Kimsey, 111 Whitehall Bldg., 960 E. Third St., Chattanooga, Tenn. 37403. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Francis E. O'Neill, 1128 Nix Prof. Bldg., San Antonio, Tex. 78205.

The Fleischner Society
Secretary, Dr. E. Robert Heitzman, State University of New York Upstate Medical Center, Syracuse, N. Y. 13210. Annual Meeting: St. Francis Hotel, San Francisco, Calif., May 28–31, 1975.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Thomas K. Orton, St. Mark's Hosp., Salt Lake City, Utah 84117. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. Charles P. Winkler, 3500 Kensington Ave., Suite 2-A, Richmond, Va. 23221.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Robert M. Allen, 9312 Convento Terr., Fairfax, Va. 22030.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Donald J. Hesch, 3216 N. E. 45th Place, Seattle, Wash. 98105.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society, other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. James O. Richardson, Jr., 280 Mamaronek Ave., White Plains, N. Y. 10605. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. June Unger, Wood VA Hosp., Wood, Wis. 53193. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Alvin Greenberg, Radiother. Ctr., Univ. Hospitals, Madison, Wisc. 53706. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Rafael Umaña-Umaña, Apartado 8-5340, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Miranbell Solis, Apartado 3352, San José, Costa Rica, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de Salvador
Secretary, Dr. Carlos Mejija, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0–05, Zona 1, Guatemala

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panama, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
121 Boul. Taschereau, Greenfield Park, P. Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretaries, D. H. Traspnell, M. Cohen, 32 Welbeck St., London, W1M/7PG, England.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Ross O. Hill, Suite 806, 1440 St.

Catherine St. West, Montreal 107, Que., Canada. Annual Meeting: Toronto, Ont., May 11–16, 1975.

EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

FACULTY OF RADIOLOGISTS
Honorary Secretary, Robert Morrison, Lincoln's Inn Fields, London WC2A 3PN, England.

MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M8AE, 1 England.

SOCIÉTÉ CANADIENNE-FRANÇAISE DE RADIOLOGIE
Secretary-General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P. Q., Canada. Meets every third Tuesday from October to April.

THE HOSPITAL PHYSICISTS' ASSOCIATION
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

THE ROYAL AUSTRALASIAN COLLEGE OF RADIOLOGISTS
Honorary Secretary, Dr. T. P. Loneragan, 45 Macquarie St., Sydney, N.S.W. 2000, Australia.

SOUTH AMERICA

COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Lauro de Abreu Coutinho, Caixa Postal 5984, São Paulo, Brazil.

FEDERACIÓN ARGENTINA DE SOCIEDADES DE RADIOLOGÍA
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.
Congress will be organized by Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario in 1975.

SOCIEDAD ARGENTINA DE RADIOLOGÍA
Secretary-General, Dr. Tomás J. Gutiérrez F, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. Manuel Neira, Casilla 13426, Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

SOCIEDAD COLOMBIANA DE RADIOLOGÍA
Secretary-General, Dr. Gustavo Sánchez Sánchez, Bogotá, Colombia. Meets last Thursday of each month.

SOCIEDAD ECUATORIANA DE RADIOLOGÍA
Secretary, Dr. Luis Blum, Casilla 3712, Guayaquil, Ecuador.

SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

SOCIEDAD PERUANA DE RADIOLOGÍA
Secretary-General, Dra. Ladis Delpino, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

SOCIEDAD DE RADIOLOGÍA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

SOCIEDAD DE RADIOLOGÍA DEL NORDESTE ARGENTINO
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

SOCIEDAD DE RADIOLOGÍA DE LA PLATA
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

SOCIEDADE DE RADIOLOGÍA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

SOCIEDAD DE ROENTGENOLOGÍA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

SOCIEDAD DE RADIOLOGÍA, RADIOTERAPÉUTICA Y MEDICINA NUCLEAR DE ROSARIO
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

SOCIEDAD SALTEÑA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Treasurer, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina

SOCIEDAD VENEZOLANA DE RADIOLOGÍA Y MEDICINA NUCLEAR
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

CONTINENTAL EUROPE

ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

SOCIÉTÉ ROYALE BELGE DE RADIOLOGIE
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
Secretary, Dr. J. Corbaton, Clinica Infantil "La Paz," Av. Generalissimo, 117 Madrid 34, Spain. Annual Meeting: Madrid, Apr. 24–26, 1975.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 730, Paris Cadet 15, France.

SOCIÉTÉ FRANÇAISE DE RADIOLOGIE MÉDICALE, MÉDECINE NUCLÉAIRE ET ÉLECTROLOGIE, and its branches: SOCIÉTÉ DU SUD-OUEST, DU LITTORAL MÉDITERRANÉEN, DU CENTRE ET LYONNAIS, DU NORD, DE L'OUEST, DE L'EST, DU MASSIF CENTRAL, D'OUTRE-MER ET D'ÉTRANGER.
Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru, Paris 75008, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Faculty of Radiologists of the Royal College of Surgeons in Ireland
Honor. Secretary, Max. J. Ryan, St. Stephen's Green, Dublin 2. Annual Meeting, Oct. 25 and 26, 1974.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langenfeldt, Tretommervej 20a, DK. 8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. F. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägn 5 C., Finland. Annual Meeting.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereinigung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Honor. Gen. Secretary, Dr. S. P. Aggarwal, 10-B Kasturba Ghandi Marg, New Delhi 110001. 28th Indian Congress of Radiology will be held in Hyderabad in 1975.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Jakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Majid Rooholamini, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month. 5th Iranian Congress of Radiology, Apr. 12-17, 1975. Deadline for acceptance of papers, Jan. 20, 1975.

Israel Radiological Society
Secretary, Dr. M. A. Lerner, Hesharon Hosp., Petah-Tigva, Israel.

Philippine College of Radiology
Secretary-Treasurer, Dr. Eduardo V. Nievera, Box 1284 Commercial Center, Makati, Rizal D-708, Philippines. Meets every second Thursday of the month.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, C. A. Beetlestone, M.B., B.S., D.M.R.D., Dept. Radiol., University College Hosp., Ibadan, Nigeria. Thirteenth Annual Conference, in Ibadan, Feb. 8-10, 1975. Conference Secretary: Dr. A. O. Sobo, The J. F. Kennedy Medical, P. O. Box 1973, Monrovia, Liberia.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

McIvor, James. The radiological features of ameloblastoma. *Clin. Radiol.*, April, 1974, *25*, 237–242. (From: X-ray Department, Institute of Dental Surgery, Gray's Inn Road, London, W.C.2. Present address: X-ray Department, Hillingdon Hospital, Uxbridge, Middlesex, England.)

Ameloblastoma is a rare lesion accounting for about 1 per cent of all cysts and tumors of the jaws. It is more common in Africans than in white races; it is of varying degrees of aggressiveness; and the histologic appearance of a single tumor may vary considerably from one part of the lesion to another. It is locally recurrent and may extend into the base of the skull. Pulmonary metastases have been rarely reported, and its behavior has been compared to that of a basal cell carcinoma.

Of 16 patients who were seen when the tumor presented originally, 11 exhibited a radiolucent area in the jaw with at least three of the following additional features: expansion of the overlying cortical surface; a sharply defined dense scalloped margin; a multilocular appearance; and resorption of the root of an adjacent tooth. In the other 5 patients the solitary cystic lesion presented no radiologic feature to differentiate it from a simple cyst.

The differential diagnosis in the 11 characteristic roentgen studies included odontogenic keratocyst, fibroma, fibrosarcoma, hemangioma, aneurysmal bone cyst, giant cell tumor, reparative granuloma, fibrous dysplasia, and ameloblastoma or its malignant variant.

There were 6 cases in which the ameloblastoma was recurrent, and the roentgen appearance of these is even more characteristic. In all cases the lesion was scalloped with a sharply defined, dense border, and in all the lesion was mulilocular with perforation of the overlying cortex. Most of these occurred in edentulous regions in the ramus of the mandible.

Since the surgical treatment of an ameloblastoma is more extensive than that for a simple cyst, the value of the roentgen study is in indicating which cystic tumors should be subjected to biopsy before a definitive attack.—*E. Frederick Lang, M.D.*

Neck and Chest

Poser, H., Weidauer, H., and Jacobsen, J. Röntgenschichtuntersuchung bei Kehlkopfverletzungen. (Tomographic roentgen examination of laryngeal injuries.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, June, 1974, *120*, 707–718. (Address: Dr. med. H. Poser, Radiologische Univ.-Klinik, 23 Kiel, Arnold-Heller-Strasse 9, Germany.)

Laryngeal injuries are frequently overlooked or neglected, especially when attention is concentrated on simultaneous severe trauma to other parts of the body. The neck injuries may at first appear mild or innocuous. Nevertheless, tomograms, in addition to conventional roentgenograms, can demonstrate serious damage. When the full extent of the lesion is realized at an early date, prophylactic therapy can be instituted and irreparable damage avoided.

Tomograms in 2 planes are obtained, while the patient produces an "U" sound for optimal unfolding of the supraglottic space. In blunt trauma to the neck without a laryngeal tear, tomography can be readily performed even when a tracheotomy tube is *in situ*.

In the past 2 years, 15 patients with acute or chronic neck injuries were selected for tomographic studies. Specific causes included: stabbing during altercation (1); suicidal attempt (1); football injuries (2); falling against hard objects (2); steering wheel injury (1); hitting of flying objects or fist blow (3); foreign body aspiration (1); gunshot wounds (2); injuries from intubation (2).

The extent of a hematoma can be readily ascertained as contours remain sharper than in an edema. Emphysema from mucosal tears suggests an extensive injury and possibly a superimposed infection. This condition may result in a serious stenosis.

The diagnosis of fractures depends on the mineral content of affected cartilages. Dislocations usually occur at the cricoid cartilage in the region of the aryepiglottic fold. Damage from intubation may develop after an interval of 7 to 14 days in the form of granulation tissue.—*Ernest Kraft, M.D.*

Genereux, George P., and Emson, H. E. Talc granulomatosis and angiothrombotic pulmonary hypertension in drug addicts. *J. Canad. A. Radiologists*, June, 1974, *25*, 87–93. (Address: George P. Genereux, M.D., Department of Diagnostic Radiology, University Hospital, Saskatoon, Saskatchewan, Canada.)

Recent estimates suggest that there are 600,000 drug addicts in the United States, and between 10,000 and 20,000 in Canada. An unknown number of drug dependents use additive substances intravenously, and in so doing, expose themselves to several important cardiovascular and pulmonary complications.

It has been shown both experimentally and clinically that the 2 most deleterious fillers are talc and starch. The former, upon reaching the microvascular circulation of the lung, is capable of inciting granulomatous reactions in arterioles and capillaries, and ultimately thrombose these vascular channels. Fibrosis may result around small vessels as well as in the alveolar septa. Starch, on the other hand, is said to cause only bland thrombosis of arterioles. Since these lesions are so strategically placed in and around arterioles and capillaries, a slowly progressive oblit-

eration of the microvascular circulation occurs, leading to an obstructive type of precapillary pulmonary hypertension, the so-called "angiothrombotic pulmonary hypertension."

Twelve of 18 patients under treatment with methadone had radiographic evidence of diffuse parenchymal abnormality which was interstitial in distribution. The pattern consisted of micronodular, reticulonodular and reticular densities. The lesions showed a striking distribution at the hila with lesser involvement apparent in the extremes of both the upper and lower lobes. The cardiac silhouette was abnormal in 2 patients, consistent with right ventricular dilatation or hypertrophy, and this was combined with enlargement of the main pulmonary and both hilar arteries indicating chronic cor pulmonale.—*David Morse, M.D.*

HOYER, J., MALMEJAC, C., DELAYE, A., and HOUËL, J. Beitrag zur Echinokokkose des Herzens an Hand von sieben eigenen Beobachtungen. (Contribution to cardiac echinococcosis on the basis of seven observations.) *Thoraxchirurgie*, June, 1974, *22*, 197–206. (Address: Dr. J. Hoyer, Centre de Chirurgie Thoracique et Cardiaque, Hôpital Salvator, 249 Bd. de Ste. Marguerite, 130009 Marseille, France.)

Cardiac echinococcosis, which is found in about 1 per cent of all cases of echinococcosis, can be divided into 3 stages. The first or "latent" stage can last decades and produces no symptoms. In the second stage or "stage of manifestation" the patient complains of precordial pain and/or pressure, dyspnea and nausea. The third stage or "stage of complications" manifests itself by myocardial insufficiency and/or rupture of the hydatidiform cyst into the cardiac chambers or pericardium.

Radiographic studies are of value in all 3 stages. Any change in contour of the cardiac silhouette is suspicious for echinococcosis; calcifications along the cardiac periphery support further the diagnosis, if tuberculous pericarditis can be excluded. Size and location of the cysts can be best determined by angiocardiography and coronary arteriography.

In 7 cases, which were operated upon in a period of 15 years, 14 cysts were encountered; 8 were localized in the left and 4 in the right heart, the remaining 2 were found in the ventricular septum. Four patients had solitary cysts, while 3 patients were found to have multiple cysts. The correct diagnosis was made preoperatively in 6 of the 7 patients. In 5 patients the surgical intervention was successful; 2 patients died, 1 during surgery due to rupture of a large cyst in the right atrium followed by pulmonary embolization, and the other due to cerebral embolization secondary to postoperative rupture of an additional cyst in the left ventricle which was overlooked during surgery.—*Peter F. Winter, M.D.*

HERMANUTZ, K. D., and BÜCHELER, E. Traumatische, nichtperforierende Verletzungen der thorakalen Aorta und der intrathorakalen Aortenbogenäste. (Traumatic, nonperforating injury to the thoracic aorta and its intrathoracic aorta arch branches.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1974, *120*, 156–163. (Address: Dr. K. D. Hermanutz, Radiologische Klinik der Universität Bonn, 53 Bonn-Venusberg, Germany.)

Traumatic rupture of the thoracic aorta occurs distal to the subclavian artery in the region of the isthmus in 50 per cent and at the base of the aorta in 20 per cent of all cases. While the former is usually the result of abrupt horizontal deceleration, the latter is frequently caused by vertical deceleration. A blunt thoracic trauma with abrupt rotation of the neck to the contralateral side may lead to avulsion of the intrathoracic branches of the aorta. Ruptures at the base of the aorta are frequently associated with cardiac injuries and in most cases result in immediate death. In contrast, 10–20 per cent of the cases with rupture at the isthmus survive more than 1 hour allowing diagnostic studies and appropriate surgical treatment.

The radiographic findings are described by means of 4 cases. Superior mediastinal widening to the left is seen in patients with localized hematomas; if the first chest roentgenogram is equivocal, repeat roentgenograms at 15 minute intervals should be obtained within the first 2 hours. Perivascular extravasation might result in a double contour of the aortic arch, which is often best seen in the oblique projections. Displacement of the esophagus posteriorly and the trachea to the right indicates a progressive mediastinal hematoma. Frequently bleeding into the left pleural space occurs; if rib fractures are present this might be misinterpreted as bleeding from intercostal or thoracic vessels; however, in cases with a progressive or persistent bleeding from a chest tube, the possibility of a rupture of the aorta has to be given first consideration.

The diagnosis of an aortic rupture should be confirmed by angiography. Direct visualization of the aorta in 2 planes *via* the right axillary approach is the method of choice. Indirect aortography results in poor contrast, but has the advantage of giving information about the pulmonary and cardiac status. The angiographic findings in subadventitial and periaortic hematoma include localized dilatation of the aorta, irregular contours of the aortic wall, and saccular or fusiform pseudoaneurysms; the site of rupture might be seen as a radiolucent band. A total transverse rupture can result in complete stop of the contrast column.—*Peter F. Winter, M.D.*

MARTIN, NEIL D., ZARET, BARRY L., MCGOWAN, RONALD L., WELLS, HARRY P., JR.,

and Flamm, M. D. Rubidium-81: a new myocardial scanning agent; noninvasive regional myocardial perfusion scans at rest and exercise and comparison with potassium-43. *Radiology*, June, 1974, *111*, 651–656. (Address: Neil D. Martin, Col. USAF, MC, Section of Nuclear Medicine (SGHR-R), David Grant USAF Medical Center, Travis Air Force Base, Calif. 94535.)

The authors used inert gas wash-out technique to assess regional myocardial perfusion. Measurements were made employing a scintillation camera following the injection of xenon 133 into the coronary artery.

Twenty-four patients with known or suspected coronary artery disease were studied at the time of coronary arteriography. Wash-out curves were constructed, using 2 second time frames from the peak to 50 seconds afterward. In 6 patients, the characteristics of the wash-out curve were defined following collection of data for 30 minutes.

The 30 minute curves were subjected to compartmental analysis. Myocardial blood flow was estimated from the initial slope of the xenon clearance curve representing the first 50 seconds of wash-out data. Functional images were also made.

Three compartments were delineated by graphic analysis. The homogeneous distribution of the 2 faster compartments suggests that they represent myocardial perfusion; the slow compartment is consistent with blood flow to fat.

Regional myocardial flow was relatively homogeneous in patients with normal coronary arteriograms. Heterogeneities of flow were found in patients with single vessel disease and multiple vessel disease. The area of myocardium with altered perfusion was delineated better by functional image than by quadrantic flow values.

By combining the gamma camera image of the initial xenon distribution, the functional image, and quadrantic flow rates, patients with hemodynamically significant coronary artery disease could be separated from those with normal arteriograms.—*W. R. Carpentier, M.D.*

Holman, B. Leonard, Adams, Douglass F., Jewitt, David, Eldh, Per, Idoine, John, Cohn, Peter F., Gorlin, Richard, and Adelstein, S. James. Measuring regional myocardial blood flow with ^{133}Xe and the Anger camera. *Radiology*, July, 1974, *112*, 99–107. (Address: B. Leonard Holman, M.D., Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Mass. 02115.)

The reliability of adequately evaluating regional myocardial perfusion by imaging techniques with potassium 43 (K^{43}) has been established. Its production costs and photon energies make it an unlikely agent for routine use. Potassium 42 also has poor physical characteristics, and the isotopes of cesium are extracted poorly by myocardium. Rubidium has a chemistry and physiology more closely akin to potassium, and rubidium 81 (Rb^{81}) appears to have physical characteristics satisfactory for imaging. It has a half-life of 4.6 hours, and exhibits photopeaks at 190, 436, and 511 kev. The latter 2 occur as a result of electron capture and positron emission, and can be utilized by a rectilinear scanner as one broad peak. One of the daughter products, krypton 81m, produces the 190 kev. photopeak in its transition to krypton 81.

Rubidium 81 is cyclotron produced, and can be obtained as the sterile, pyrogen-free radionuclide. However, rubidium 82m routinely appears as a carrier. Because this carrier has a longer half-life (6.2 hours) and higher energy gamma photons, it is mandatory that the rubidium 81 be utilized as soon after production as possible. Dosimetry calculations based on an absolute maximum of 33 per cent of rubidium 82m contamination are as follows: total body and bone—less than 222 millirads/millicurie, and gonads—less than 281 millirads/millicurie.

This preliminary study comprises 15 patients, 1 normal and 14 with arteriosclerotic heart disease. Studies were performed on both the rectilinear scanner and the Anger-type scintillation camera, and all images were of diagnostic quality. However, the use of the camera requires the pinhole collimator with 3 mm. insert and the introduction of a 5 cm. lead shield between the patient and the camera to reduce lateral collimator penetration. Patients were studied at rest and post-exercise whenever possible. Resting studies were performed after a 12–24 hour fast, and the intravenous injection of 2 millicuries of Rb^{81}. Exercise was obtained *via* a motorized treadmill to the point of angina or fatigue. Four mc Rb^{81} was then injected intravenously and exercise continued 30–60 seconds, after which imaging was performed. Images were obtained at both the high and low photopeak settings in both the anterior and left anterior oblique positions. Ten of the 14 patients with arteriosclerotic heart disease, who were imaged with Rb^{81}, also had comparable images with K^{43}. Nine of the 14 also had confirmatory coronary angiography and left ventriculography.

Myocardial images of the normal subject with Rb^{81}, after exercise, exhibited a homogeneous distribution of the radioactivity throughout the cardiac mass. Four of the 14 with arteriosclerotic heart disease had recent myocardial infarcts and had only rest images with Rb^{81}, showing perfusion defects in the area of infarction. The remaining 10 patients had both rest and post-exercise images. Seven of the 10 had prior myocardial infarction, all of which were demonstrated on imaging; 4 of these patients' perfusion defects became larger after exercise, 3 did not. The other 3 of these 10 had only history of angina. Two of these 3 had normal rest images, but developed perfusion abnormalities after exercising. The third of

these 3 had an equivocal rest scan which failed to change after exercise.

On the basis of this preliminary study, it appears that Rb^{81} has similar imaging value to K^{43}. Its advantages lie in more satisfactory spectral energies, more economical production, and a more satisfactory half-life. A theoretic possibility exists for measurement of coronary blood flow by comparison of myocardial activity of Rb^{81} decay to that of its daughter, krypton 81. Obviously, further investigation of this radionuclide, for all these purposes, is indicated.—*F. C. Petty, M.D.*

Hauswaldt, Ch., and Wolf, G. Zeichen der Renalen Osteopathie auf der Thorax-Übersichtsaufnahme. (Signs of renal osteopathy seen on scout roentgenograms of the thorax.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1974, *120*, 186–191. (Address: Priv.-Doz. Dr. Christian Hauswaldt, Medizinische Universitäts-Klinik, 34 Göttingen, Humboldtallee 1, Germany.)

In 36 per cent of 103 patients with histologically proved renal osteodystrophy, the chest roentgenogram revealed definite osseous changes in the clavicle and vertebral bodies of the dorsal spine. In an additional 36 per cent of these patients, discrete bone changes were found. In a subgroup of 60 patients with advanced renal osteodystrophy, definite bone changes were found in 48 per cent with an additional 30 per cent having discrete changes.

The roentgenographic abnormalities in the clavicle included: thinning and/or ill-defined demarcation of the cortex; blurred and/or coarse spongiosa; subperiosteal absorption; and cysts. The changes were usually more pronounced in the lateral aspect of the clavicle.

The findings in the spine included unsharp contours of the upper and lower vertebral plates, accentuated vertical spongiosa and decreased bone density.

The described findings are not specific of renal osteopathy as they can also be seen in secondary hyperparathyroidism of other origin, primary hyperparathyroidism, intestinal osteopathy and heparin osteopathy.—*Peter F. Winter, M.D.*

Abdomen

Adams, James T. Adynamic ileus of the colon; an indication for cecostomy. *Surgery*, Oct., 1974, *109*, 503–507. (From: Department of Surgery, University of Rochester Medical Center, Rochester, N. Y.)

Significant adynamic ileus of the colon has been described as pseudo-obstruction or as Ogilvie's syndrome, and it is characterized by selective distention of the colon with gas in the absence of an organic obstruction. It can progress to perforation. The fundamental cause is not known, but is apparently either interference with the sympathetic nerve supply to the colon or imbalance between the sympathetic and parasympathetic innervation. It usually accompanies or follows some major abnormality in a system other than the gastrointestinal tract, such as extra-abdominal trauma, respiratory and urinary tract infection, cardiac disease, acute or chronic alcoholism, pancreatitis, or the postoperative state, particularly after orthopedic or pelvic surgery. It also occasionally occurs in patients with sepsis, shock, lead intoxication, renal failure or electrolyte imbalance.

The clinical picture is that usually associated with mechanical obstruction of the colon. Progressive abdominal distention occurs, with anorexia, nausea, and occasionally, vomiting. The patient may pass liquid feces and gas, while intestinal distention is progressing.

The most useful diagnostic measure is the plain roentgenogram of the abdomen, which shows dilatation of the colon out of proportion to any associated small intestinal distention. There is usually much less gas in the rectum, sigmoid, and portions of the descending colon than above in the colon.

The differential diagnosis includes mechanical obstruction of the descending colon, volvulus of the cecum, and vascular disease. Retrograde flow of barium into the distended sigmoid will exclude the possibility of a mechanical obstruction.

Conservative management with correction of the electrolyte and fluid imbalance and by aspiration with a nasogastric tube and a rectal tube may correct the situation. Prolonging this treatment makes pneumonia and thromboembolic complications more likely, particularly in the aged patient in whom such adynamic ileus commonly occurs. Perforation of the cecum is a real problem and has been associated with a mortality rate of almost 50 per cent.

Tube cecostomy is the procedure of choice after a short term of conservative therapy.

In a series of 20 patients seen over a 12 year period, 4 recovered after conservative treatment and 12 after cecostomy. The indications for operation were: (1) a cecal diameter of 12 cm. or more; (2) development of right lower abdominal tenderness or spasm; (3) failure to improve significantly as shown by roentgenographic evidence of decrease in colon size within 72 hours of the beginning of conservative management. While intussusception is the most frequent cause of intestinal tract obstruction in young children, its cause remains obscure in 90 per cent of these. In adults, on the other hand, intussusception is uncommon, but a specific cause can be found in almost 80 per cent. Large series have been reported indicating that primary malignant tumors account for 26 per cent to 36 per cent of the instances of intussusception in adults, and that metastatic tumors can also produce intussusception. Metastases from malignant melanoma are prominent in this latter group.

During a 13 year interval 15 patients were en-

countered at Roswell Park Memorial Institute with intussusception associated with malignant neoplasm. Of these, 11 had metastatic tumors as the leading points, and 6 of these metastatic lesions were malignant melanomas. In addition there were 2 with primary cecal tumors, and there were 2 in which the basis for the intussusception was not entirely explained, although a urogenital tumor was present.

Three of the patients were below 6 years of age, but the others were all 18 years or older. Almost all had abdominal cramps and most complained of nausea and vomiting. Plain roentgenograms showed dilated loops of small intestine suggesting either obstruction or paralytic ileus. Examination of the upper gastrointestinal tract demonstrated obstruction in 5 patients while in a sixth, the intussusception itself was identified. Barium examination of the colon disclosed the cecal defect in 1 and the intussusception in another.

The disease in these patients followed a chronic or subacute course characterized by an insidious onset, and this suggests self-reduction of the intussusception on 1 or more occasions. A palpable abdominal mass and blood in the feces were not frequently encountered. Differentiation between the primary disease and the intussusception as a complication was occasionally difficult. Tube decompression relieves the symptoms without reversing the pathologic process, and for this reason, early exploration appears to be important, particularly in patients with melanoma.—*E. Frederick Lang, M.D.*

Stanley, Robert J., Melson, Leland, and Tedesco, Francis J. The spectrum of radiographic findings in antibiotic related pseudomembranous colitis. *Radiology*, June, 1974, *111*, 519–524. (Address: Robert J. Stanley, M.D., Mallinckrodt Institute of Radiology, Washington University School of Medicine, 510 S. Kingshighway, St. Louis, Mo. 63110.)

Pseudomembranous colitis as a complication of antibiotic therapy has been receiving increasing attention in the recent medical literature. The antibiotics which appear to be associated with the development of this condition include: Tetracycline, chloramphenacol, penicillin, ampicillin, lincomycin (Lincocin), and clindamycin (Cleocin). Over a period of 6 months, the authors evaluated roentgenographically 9 patients who were subsequently proved to have pseudomembranous colitis. It was their feeling that there is a range of roentgenographic abnormalities including, in the most severe cases, an appearance which is strongly suggestive of this condition and possibly diagnostic.

Six of the patients had received Cleocin for varying intervals prior to the onset of diarrhea. One patient had received only ampicillin, another patient had received a combination of Keflin and Lincocin, and another patient a combination of Cleocin, Keflin, and ampicillin prior to onset of diarrhea. A low pressure barium enema study was done on these patients, when there was no obvious toxicity or signs of peritonitis or generalized sepsis.

The diagnostic roentgenographic findings in the presence of pseudomembranous colitis consist of a diffusely shaggy appearance of the colon due to innumerable plaque-like lesions which stud the entire mucosal surface producing rounded or oval filling defects measuring 2–7 mm. in diameter, when seen *en face*. These plaque-like lesions often were better demonstrated with the use of air studies. Ulceration of the mucosa was absent for the most part, being present only in the very late stages of the disease when the pseudomembrane was stripped from the mucosa. Edema of the wall of the bowel was indicated by irregular contour and unusual thickness of the haustral folds and this should suggest strongly the presence of pseudomembranous colitis. The differential diagnosis of these roentgenographic findings includes the pseudopolyps of ulcerative colitis and familial polyposis.

It is recognized that proctosigmoidoscopy is the preferable diagnostic procedure, but it is the feeling of the authors that the radiologist may at times be in a position to suggest the diagnosis prior to the endoscopic examination.—*Donald N. Dysart, M.D.*

Waldron, Robert L., II, and Kenny, Gerard. Radiologic diagnosis of carcinoma of the biliary tree. *J. Canad. A. Radiologists*, June, 1974, *25*, 116–126. (Address: Robert L. Waldron, II, M.D., French Hospital, 1911 Johnson Avenue, San Luis Obispo, Calif. 93401.)

Transhepatic cholangiography is a highly useful diagnostic method in patients with obstructive jaundice. With bile duct carcinoma involving the upper duct system, changes at cholangiography are sufficiently characteristic to be diagnostic with a high degree of probability.

Lesions in this region are difficult or impossible to find at surgical exploration without prior radiologic localization. Celiac arteriography also plays a role in the diagnosis of bile duct tumors.

In 3 out of 3 cases a tumor stain has been demonstrated in the capillary phase of the carcinoma of the bile duct found at surgery. As this finding was present in carcinoma of the ampulla of Vater, it is felt that this diagnostic modality may be of value in differentiating carcinoma of the ampulla from pancreatic tumors.

Since tumors in the ampullary region afford a much greater chance of surgical extirpation and long-term survival than carcinoma originating in the pancreas, the value of this radiologic differentiation is evident.

Hepatomas can grow primarily in the bile duct and they can even mimic calculus disease.—*David Morse, M.D.*

GOLDSTEIN, HARVEY M., NEIMAN, HARVEY L., and BOOKSTEIN, JOSEPH J. Angiography evaluation of pancreatic disease: a further appraisal. *Radiology*, Aug., 1974, *112*, 275–282. (Address: Harvey M. Goldstein, M.D., Department of Diagnostic Radiology, University of Texas Cancer System Center, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025.)

Bookstein *et al.* in 1969 reported the angiographic evaluation of 300 patients with suspected pancreatic carcinoma at the University of Michigan Medical Center. In that series, there were 2 major findings: (1) Significant angiographic abnormality was present in all cases of pancreatic carcinoma and unusual in patients without pancreatic parenchymal disease; and (2) the discriminate value of certain angiographic features enabled differentiation of carcinoma and pancreatitis in a majority of cases. Since that initial report, controversy regarding the value of pancreatic angiography has continued.

The authors reviewed 76 cases of histologically proven pancreatic disease in an effort to correlate the angiographic findings with the final histologic diagnoses.

Selective celiac and superior mesenteric injections were carried out in each patient. One or more subselective injections were done in approximately 75 per cent of the patients and were usually combined with magnification studies. Also, since 1971, the authors have preceded superior mesenteric arteriography by the administration of 50 mg. of tolazoline hydrochloride into the superior mesenteric artery. Intraarterial angiotensin amide was also utilized when necessary to improve visualization of the pancreatic arteries.

Angiographic abnormality was evident in virtually all symptomatic patients with pancreatic carcinoma. Irregular arterial encasement, particularly of small intrapancreatic vessels, was recognized more frequently than in previous studies, largely as a result of the improved technique. Pathologic vascularity was noted less frequently, also probably because refined techniques allowed distinction of neovascularity from encasement of pre-existing arteries. The much higher incidence of venous abnormalities in the present series is largely attributable to the use of tolazoline.

The authors feel that confident differentiation of pancreatitis and carcinoma was accomplished in 85 per cent of the cases. From this study, it is apparent that demonstration of arterial encasement alone or venous abnormality alone is suggestive of pancreatitis, while encasement plus venous abnormality is indicative of the likelihood of carcinoma. Neovascularity was another feature predictive of carcinoma, while arterial tortuosity and angulations without narrowing were a newly recognized feature indicative of pancreatitis. Arteriosclerotic changes of intrapancreatic arteries were manifest as short smooth narrowings and were associated with arteriosclerosis elsewhere, while neoplastic encasement was usually longer with irregularity or obstruction. Lack of adjacent venous involvement and arteriosclerosis was also helpful in differentiation from carcinoma.

The authors believe that one of the major advantages of angiography in pancreatic carcinoma is its ability to predict resectability. In their series, nonresectability could have been correctly predicted in 25 of 29 lesions.

It is concluded that angiography plays an important role in the management of pancreatic carcinoma. It will establish the diagnosis, so that further diagnostic investigation, often including laparotomy, may be avoided. In addition, differentiation from primary liver diseases and pancreatic inflammatory disease, where surgical intervention might be harmful, is possible in a high percentage of cases. Also, the presence of accessory hepatic arteries or other anatomic variants of surgical significance will be demonstrated.—*E. J. Schoolar, M.D.*

MIEDERER, S. E., LÖFFLER, A., SIEDEK, M., and STADELMANN, O. Die endoskopische retrograde Cholangio-Pankreatographie (E.R.C.P.) als postoperative Untersuchungsmethode. (Endoscopic retrograde cholangiopancreatography [E.R.C.P.] as a postoperative method of examination.) *Med. Welt.*, Aug., 1974, *25*, 1285–1288. (Address: Dr. S. E. Miederer, Medizinische Poliklinik der Universität Bonn, 5300 Bonn, Wilhelmstrasse 35, Germany.)

Endoscopic retrograde cholangiopancreatography has become a successful routine procedure in the past 5 years. Difficulties of catheterizing the papilla of Vater have only been encountered in a few subtotal gastrectomy cases. There are no contraindications to the procedure, and occasional complications have proved to be mild.

In 415 successive examinations with the fiberoscope, the method has been successful in 88 per cent, with the biliary system visualized in 63 per cent and the pancreatic duct in 83 per cent. Significant findings have been demonstrated, especially in cases of postoperative jaundice and abdominal pain.

The method is indicated to determine the nature of a jaundice when serum bilirubin values exceed 2 mg. per cent. In biliodigestive anastomoses, residual calculi, secondary pancreatic changes, stenoses of the anastomosis and cholangitis can be demonstrated.

In a group of 84 postcholecystectomy patients, organic changes were found in 79 per cent, while marked epigastric distress and jaundice existed in 92 per cent. In 4 patients with resection of the tail of the pancreas and anastomosis with a jejunal loop, the extent of passage of contrast material could be determined.

The method is also recommended as a routine procedure prior to surgery on the pancreatic and bile ducts.—*Ernest Kraft, M.D.*

General

McNeil, Barbara J., Holman, B. Leonard, Button, Lawrence N., and Rosenthal, David S. Use of indium chloride scintigraphy in patients with myelofibrosis. *J. Nuclear Med.*, Aug., 1974, *15*, 647–651. (Address: Barbara J. McNeil, M.D., Department of Radiology, Harvard Medical School, 25 Shattuck Street, Boston, Mass. 02115.)

The distribution of In^{111} chloride by scintigraphy was investigated in 21 patients: 18 with myelofibrosis, and 3 with other hematologic disorders; 25 bone marrow scans were obtained on the 21 patients. All patients had bone marrow biopsies and 16 had ferrokinetic studies. Patients were injected with carrier free In^{111} chloride (2 to 3 mc) and scans were done 24 hours later. Liver and spleen scans were made after the bone scans. Ferrokinetic studies were performed by the Huff technique.

The distribution of the In^{111} in patients with myelofibrosis reflected primarily the patterns found in patients with the disease, and other patients had patterns that would be expected in the absence of extramedullary hematopoietic elements. Some activity was seen in the central skeleton (vertebrae and sacrum) in all patients; long bone activity was common; activity was seen in the liver in all cases at 24 hours; varying activity was noted in the spleen, and renal activity was seen in 9 of 14 cases with myelofibrosis.

A comparison of the distribution of In^{111} chloride with the results of bone marrow biopsies, ferrokinetic data, liver and spleen histology and the clinical status showed that in 15 cases the sacral uptake of In^{111} chloride correlated with the marrow biopsy, ferrokinetic data and clinical status. In 5 cases the scintigrams overestimated the erythroid elements as compared to the biopsies, but correlated with the ferrokinetic data and clinical status. The difference could be due to the biopsy sampling. Liver and spleen activity did not correlate with the presence of erythropoietic elements assessed histologically.

In^{111} chloride scintigraphy appears to be a reliable method of assessing erythroid elements in bone marrow, but not in the liver or spleen.—*Charles W. Cooley, M.D.*

RADIATION THERAPY

Jentys, Wanda. La radiothérapie du cancer primitif du vagin à l'Institut Oncologique de Varsovie. (Radiotherapy of primary carcinoma of the vagina at the Oncologic Institute of Warsaw.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1974, *55*, 457–464. (From: Service de Gynécologie de l'Institut d'Oncologie de Varsovie, Warszawa, ul. Wawelska 15, Poland.)

The author reports the results of radiation therapy in a series of 132 patients with carcinoma of the vagina treated at the Oncologic Institute of Warsaw, between 1947 and 1967.

There were: 64 patients with localized disease, Stages I and II; and 68 patients with advanced disease, Stages III and IV.

In addition to clinical staging, the degree of infiltration and its anatomic localization were significant factors in determining the results of treatment.

Patients with infiltration of one vaginal wall had a 52 per cent cure, while infiltration of one-half or more of the circumference of the vagina had a 19 per cent cure. The best results were in tumors of the anterior and superior aspects of the lateral vaginal walls and the worst results were in tumors infiltrating the lower vaginal walls.

In the early stages of the disease, intracavitary radium application was followed by external irradiation at 220–250 kv., HVL 2.0–3.5 mm. Cu. This sequence was reversed in cases of advanced disease.

The intracavitary radium application varied between 2,000 and 7,000 mg. hr. The anterior wall of the rectum received between 2,300 to 5,500 r. The dose to the urinary bladder varied between 600 and 5,000 r.

External irradiation was administered through 2 anterior and posterior portals, delivering approximately 3,000 r to the depth. Larger doses were attempted in the advanced stages of the disease.

There was a 51.5 per cent cure in Stages I and II, and 26.5 per cent cure in Stages III and IV. These results justify a prognosis for carcinoma of the vagina that is more favorable than is usually considered.—*William H. Shehadi, M.D.*

Scott, James, Robb-Smith, A. H. T., and Burns, Ian. Bilateral breast metastases from carcinoma of the prostate. *Brit. J. Urol.*, April, 1974, *46*, 209–214. (From: United Oxford Hospitals, Oxford, England.)

In patients with known carcinoma of the prostate, development of a breast tumor poses a diagnostic problem with significant therapeutic implications. Adenocarcinoma arising as a primary tumor within the breast would be treated appropriately by surgical means. Adenocarcinoma of the prostate metastatic to the breast would best be managed by hormonal manipulation.

It is often difficult to differentiate these 2 tumors by conventional histologic techniques; however, histochemical analysis of the tumor tissue for its acid phosphatase content may provide the diagnosis.

The authors describe a case in which these considerations were present and also review the literature describing the development of the technique for histochemical analysis.—*R. H. Troupin, M.D.*

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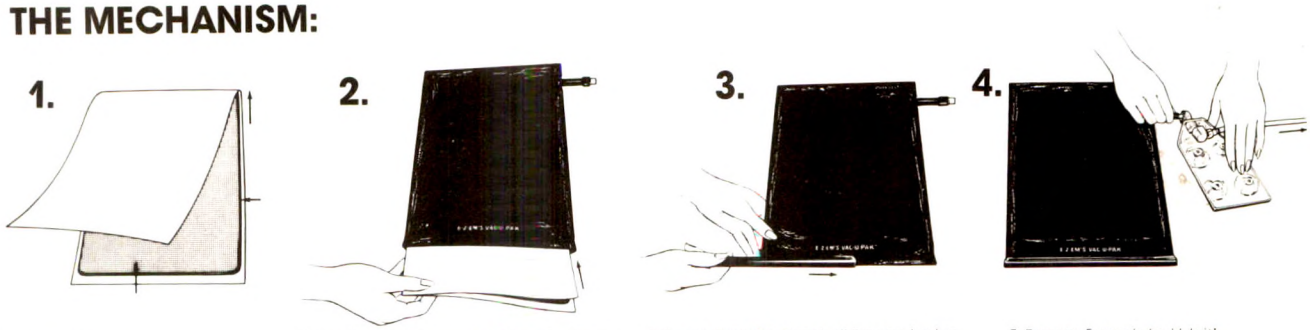
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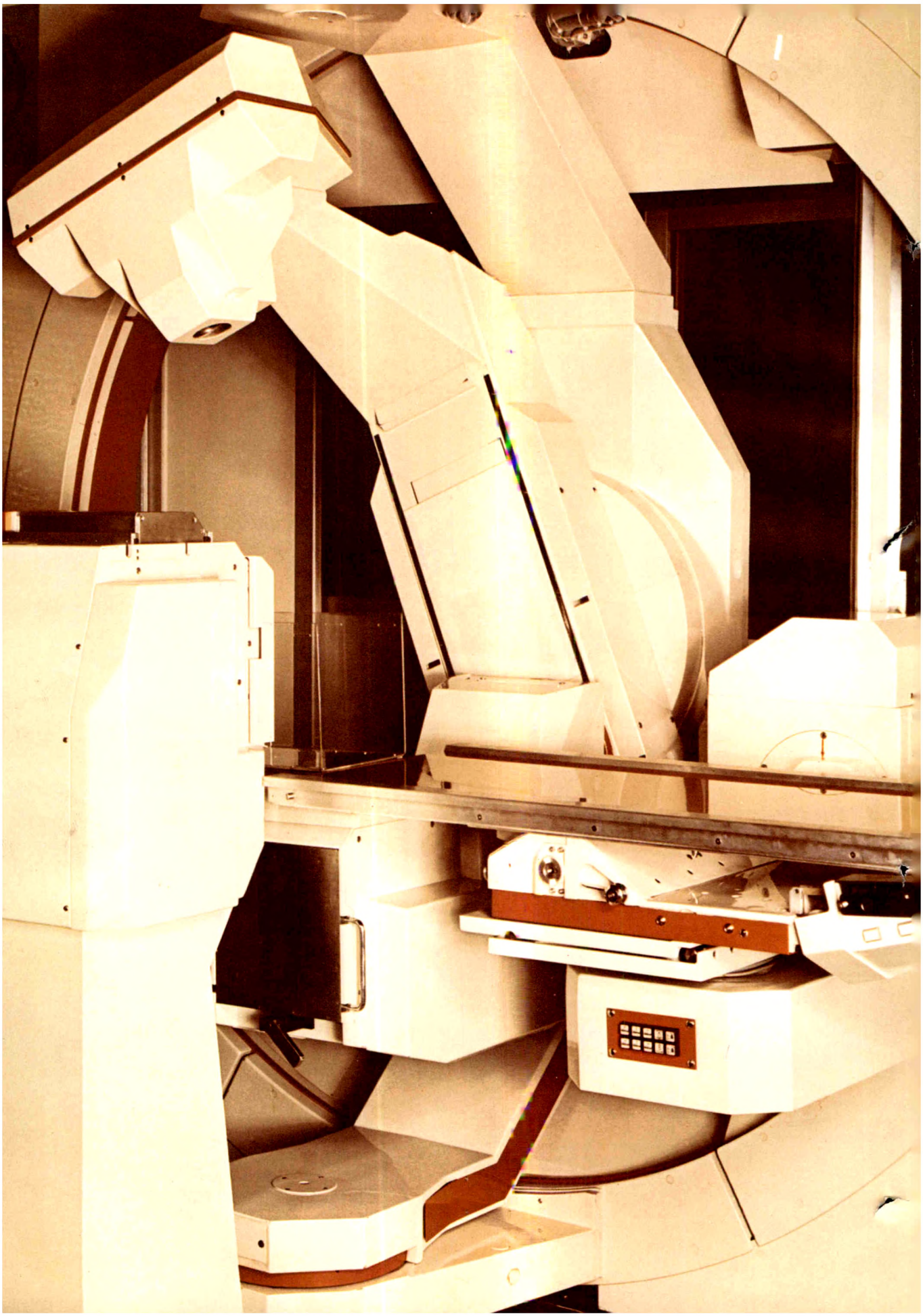
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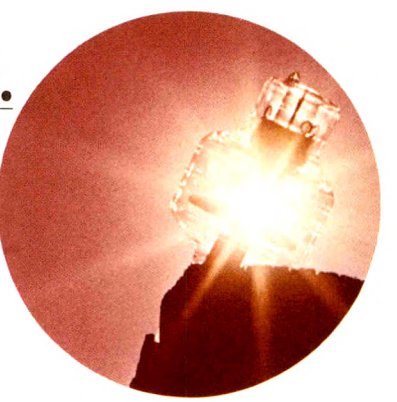
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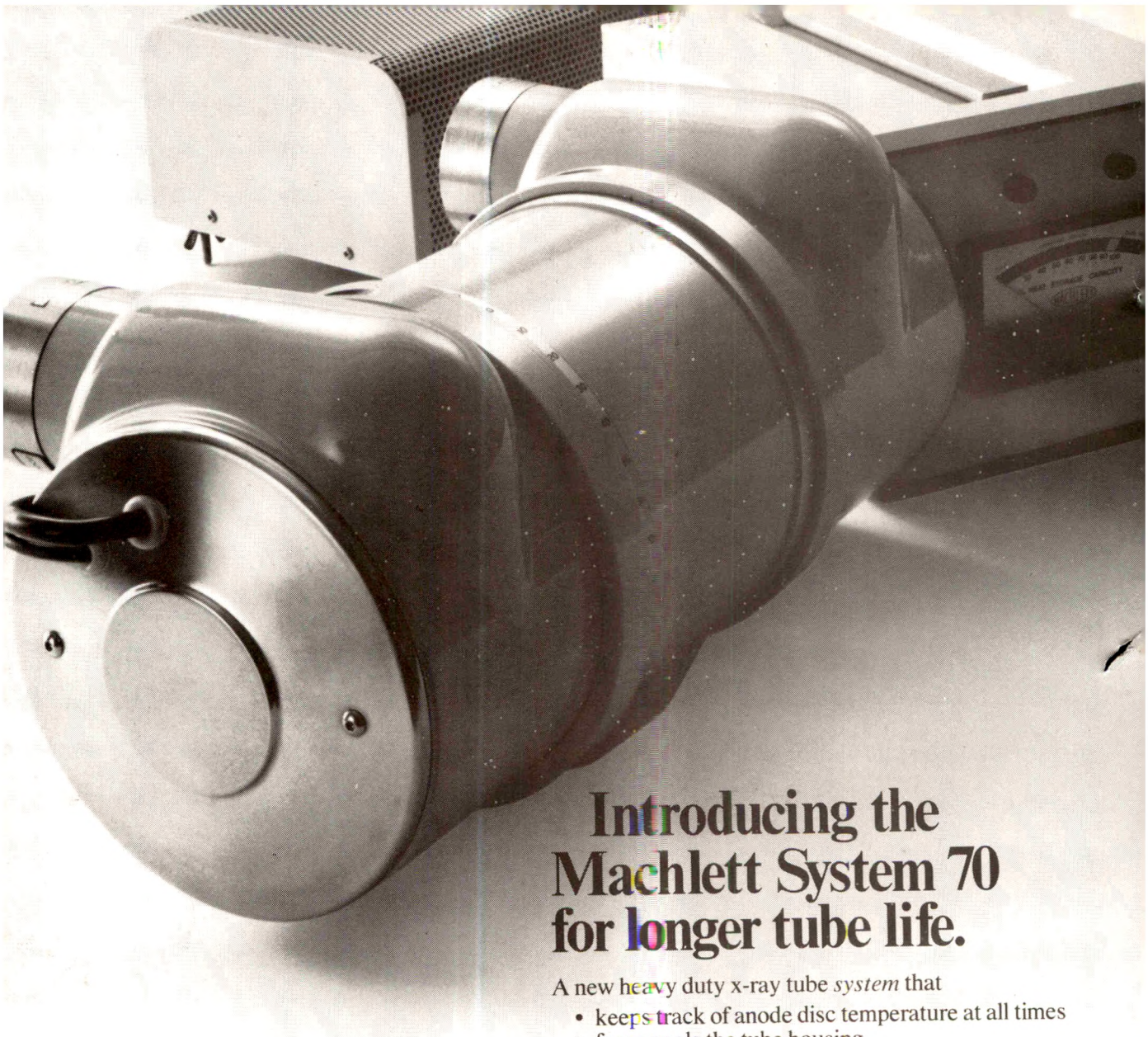
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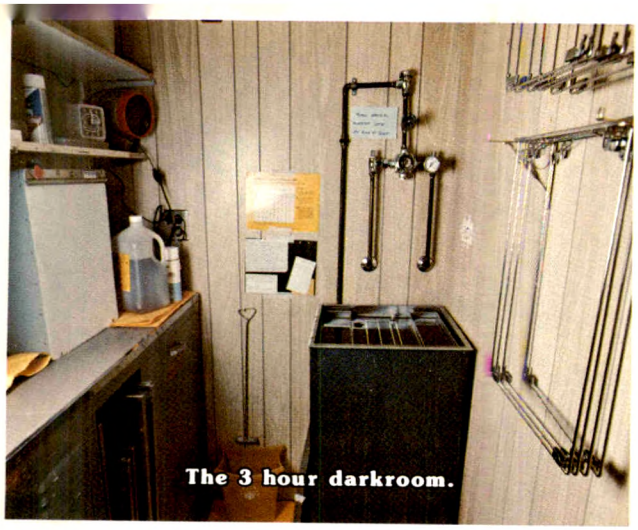
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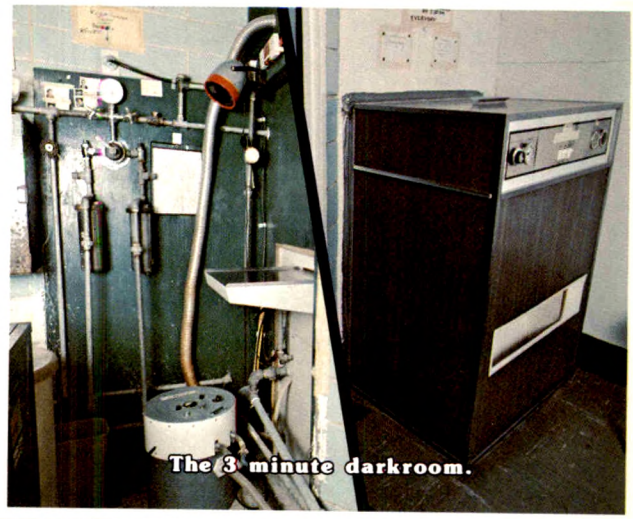
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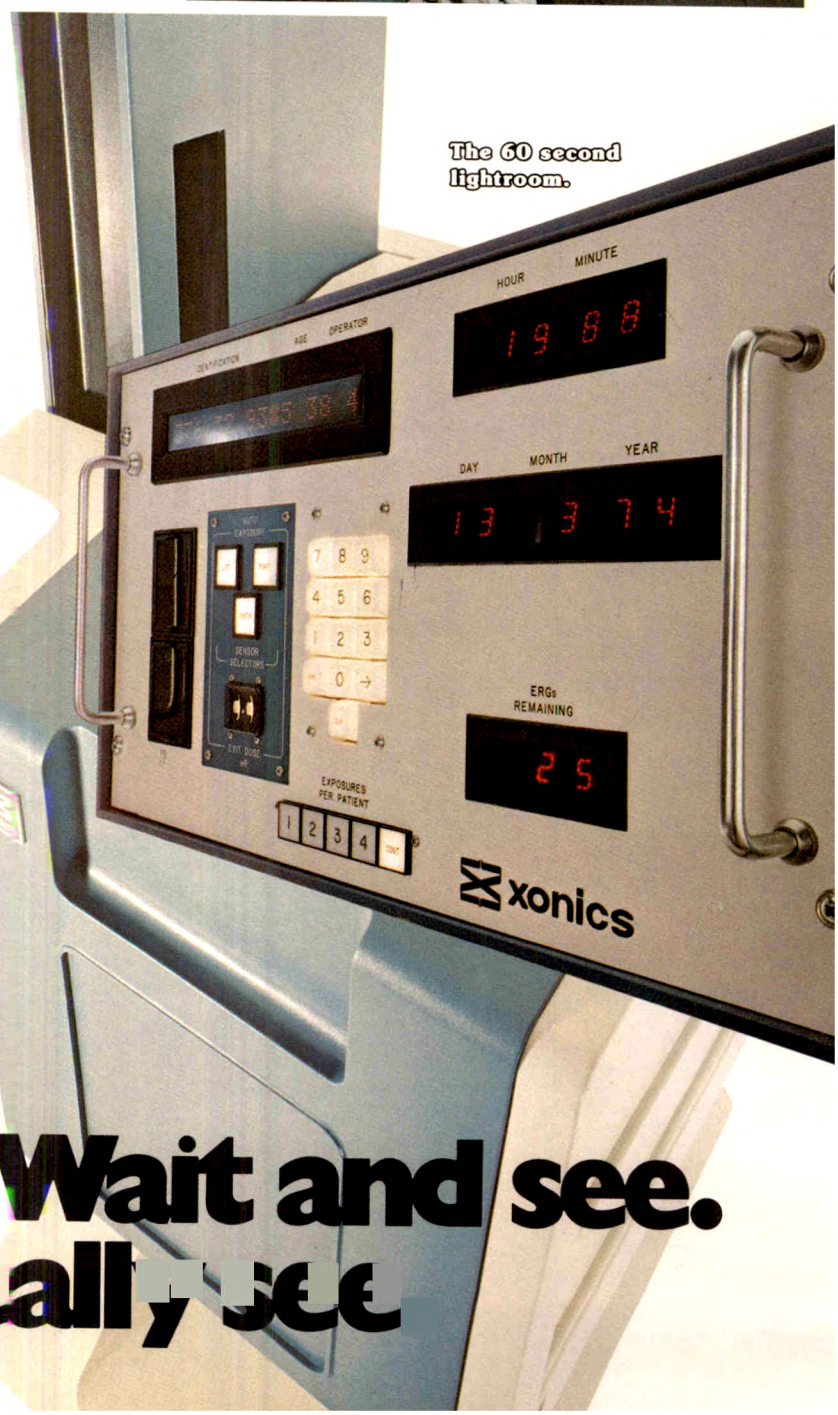
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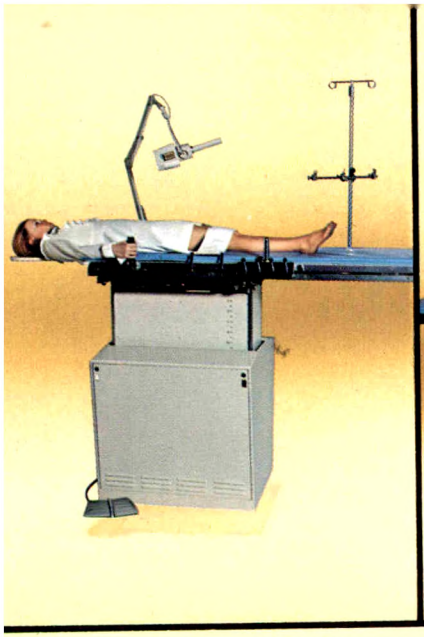
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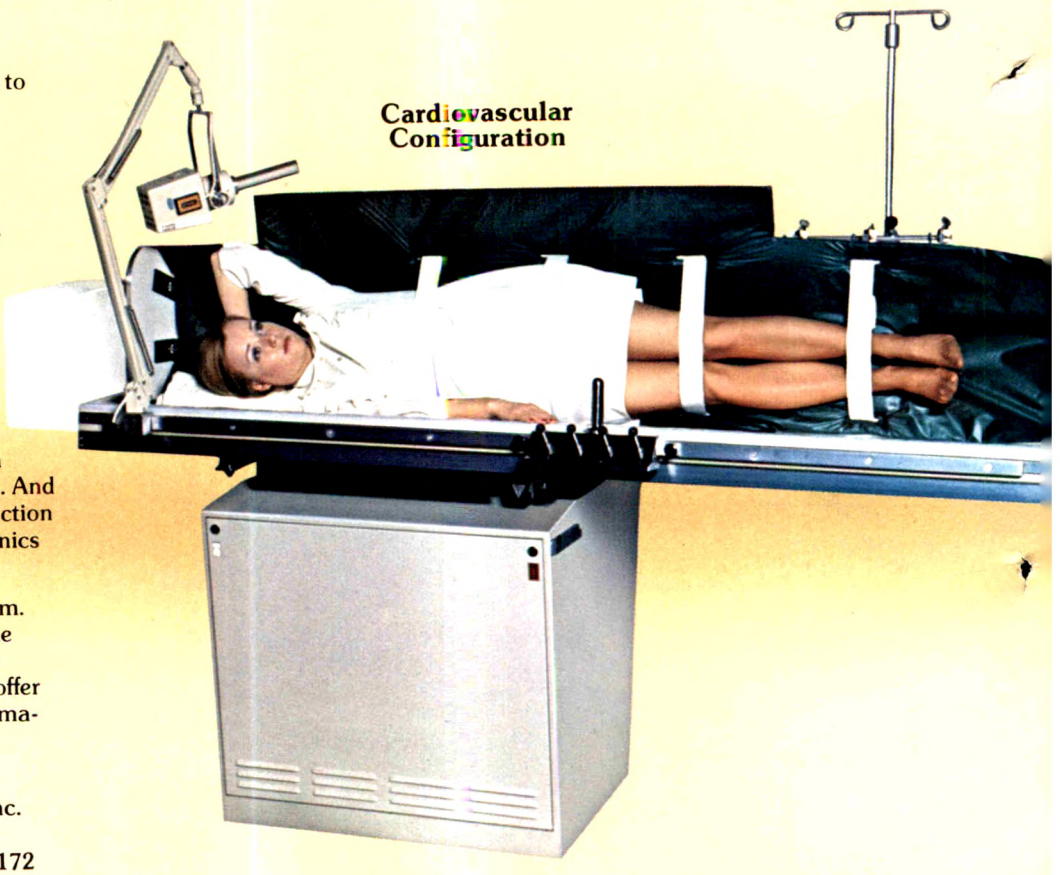
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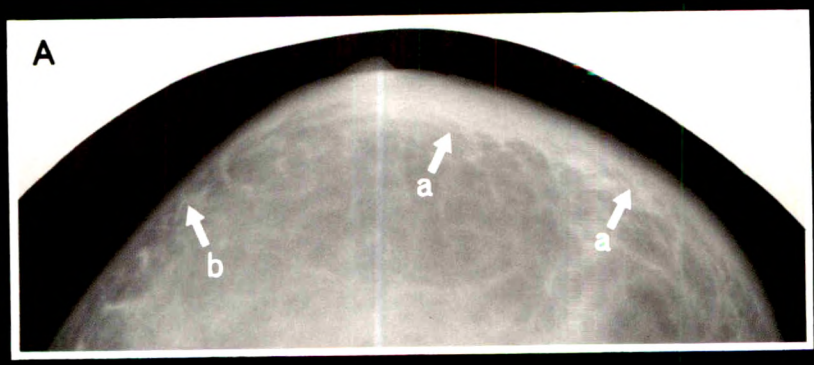
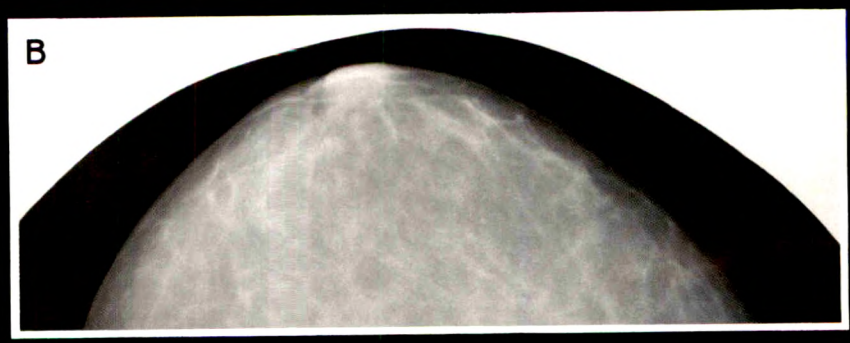


Figure I—Inflammatory Carcinoma
A. Craniocaudal View: The skin of the breast is markedly thickened at (a) and fades very perceptibly toward (b). Clinically, the patient complained of a hot, painful and swollen breast (peau d'orange), due to grade 4 scirrhous adenocarcinoma.



B. The same patient's contralateral breast was normal.

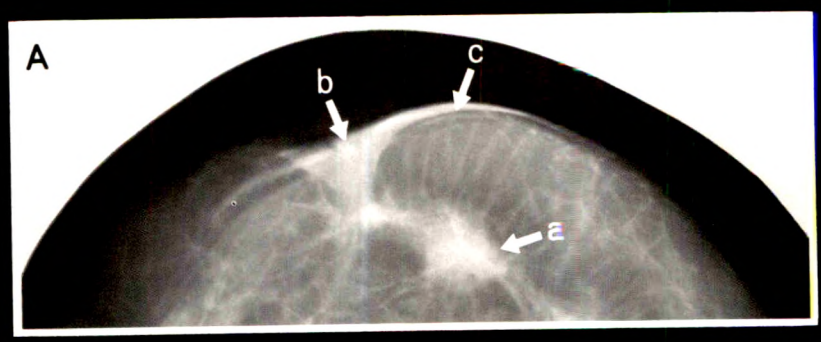
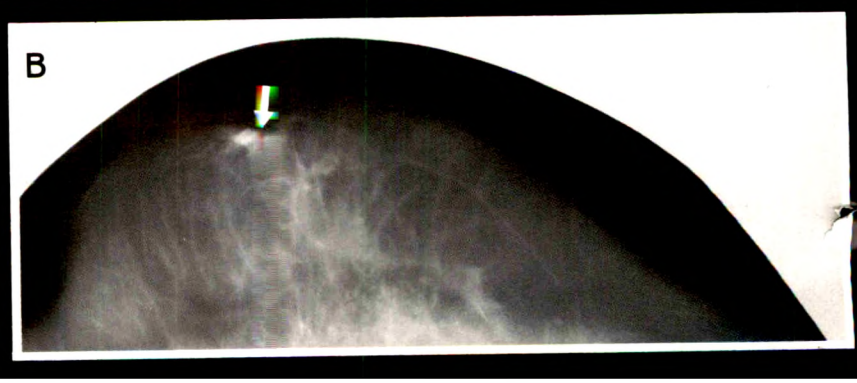


Figure II—Nipple Retraction Congenital and Malignant (Bilateral nipple retraction since childhood.)
A. Craniocaudal View: The large carcinoma (a) has invaded the nipple (b) and skin (c).



B. Normal contralateral breast reveals the congenitally inverted nipple (arrow); the skin is normal.

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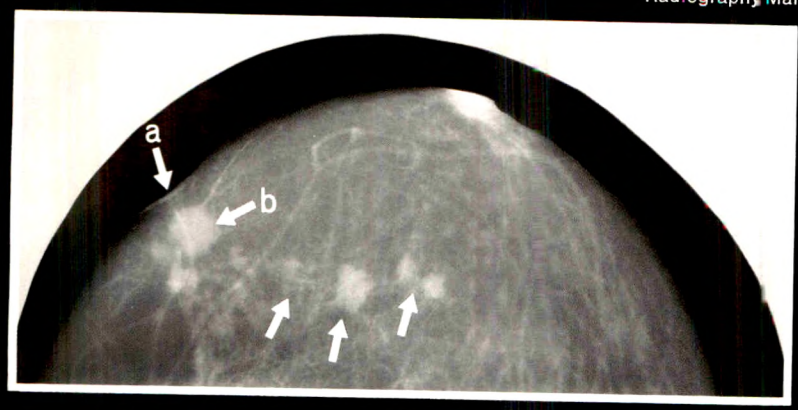


Figure III—Multicentric Carcinoma
Craniocaudal view reveals a cluster of grade 3 scirrhous adenocarcinomas (arrows) with focal skin thickening (a) immediately adjacent to the largest lesion (b).

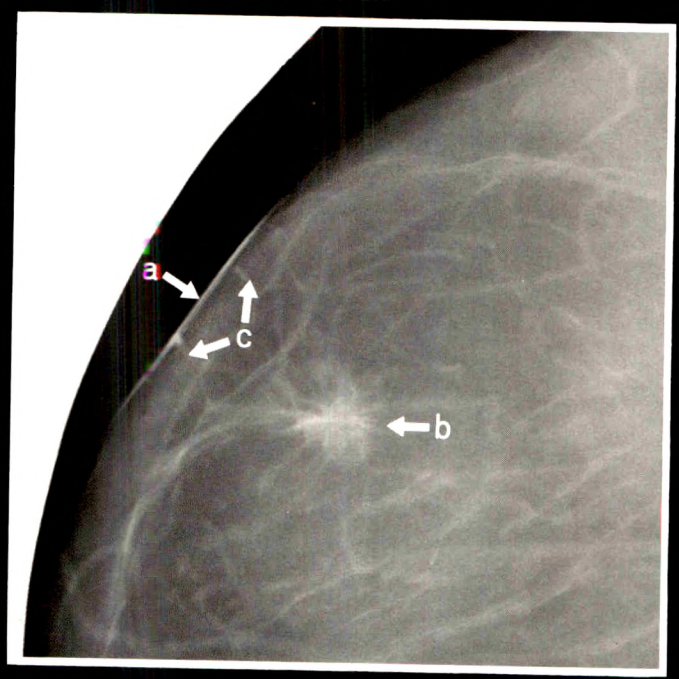


Figure IV—Carcinoma with Localized Skin Thickening
Mediolateral view reveals a limited area of skin thickening (a) due to the primary scirrhous adenocarcinoma (b) demonstrated well below skin level. Note the two prominent vessels (c) which could be dilated lymphatics.

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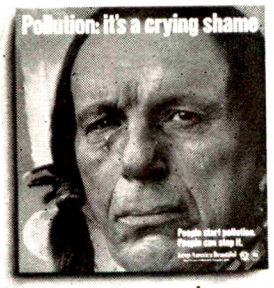
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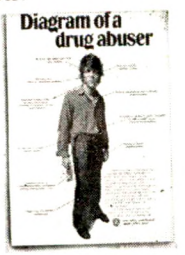


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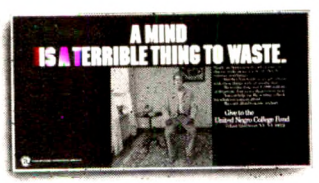




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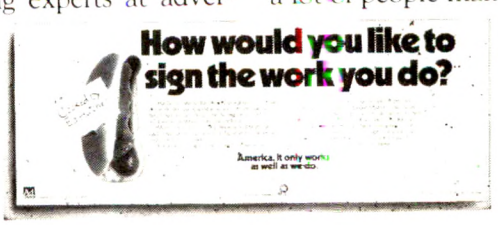
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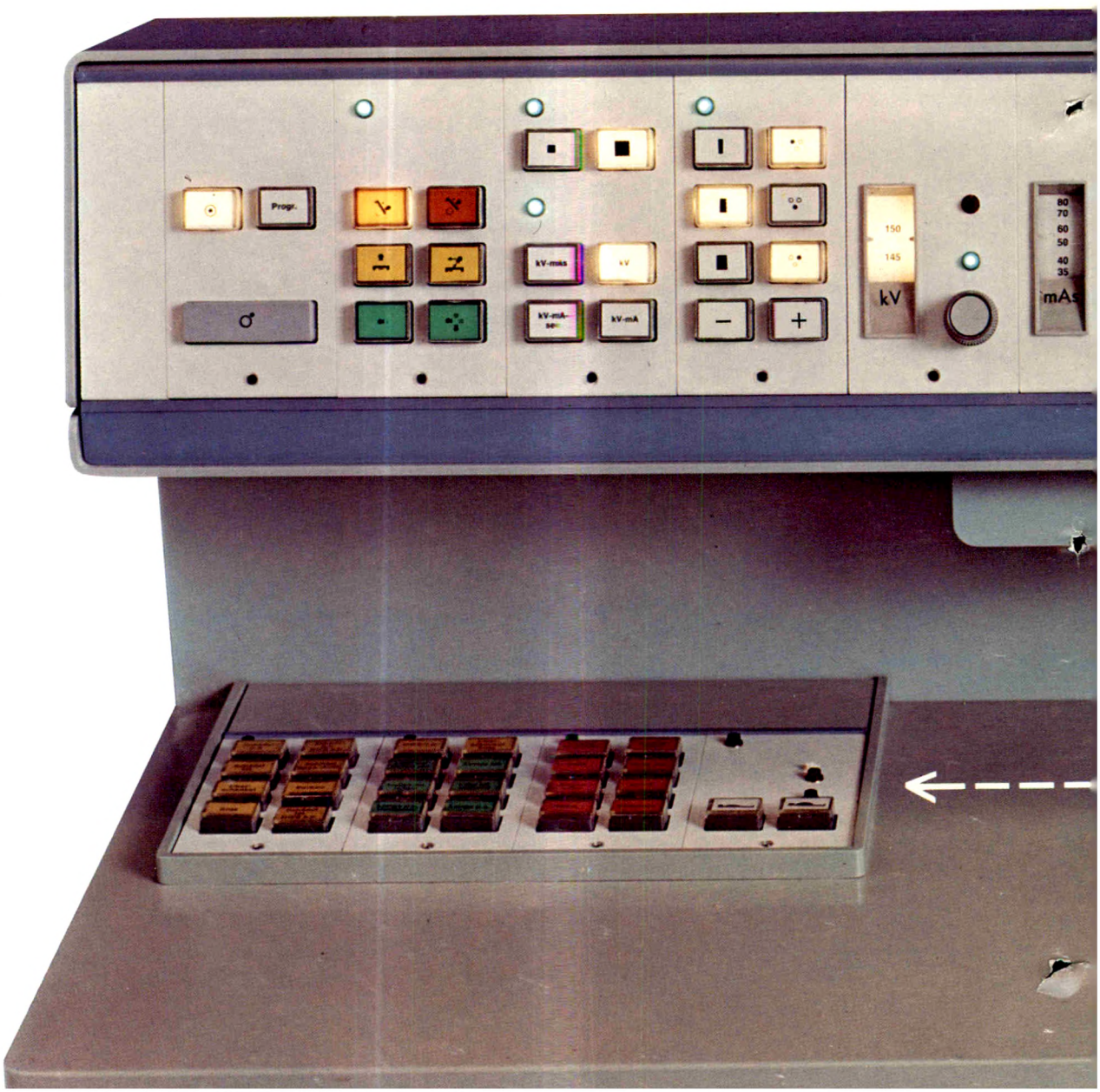
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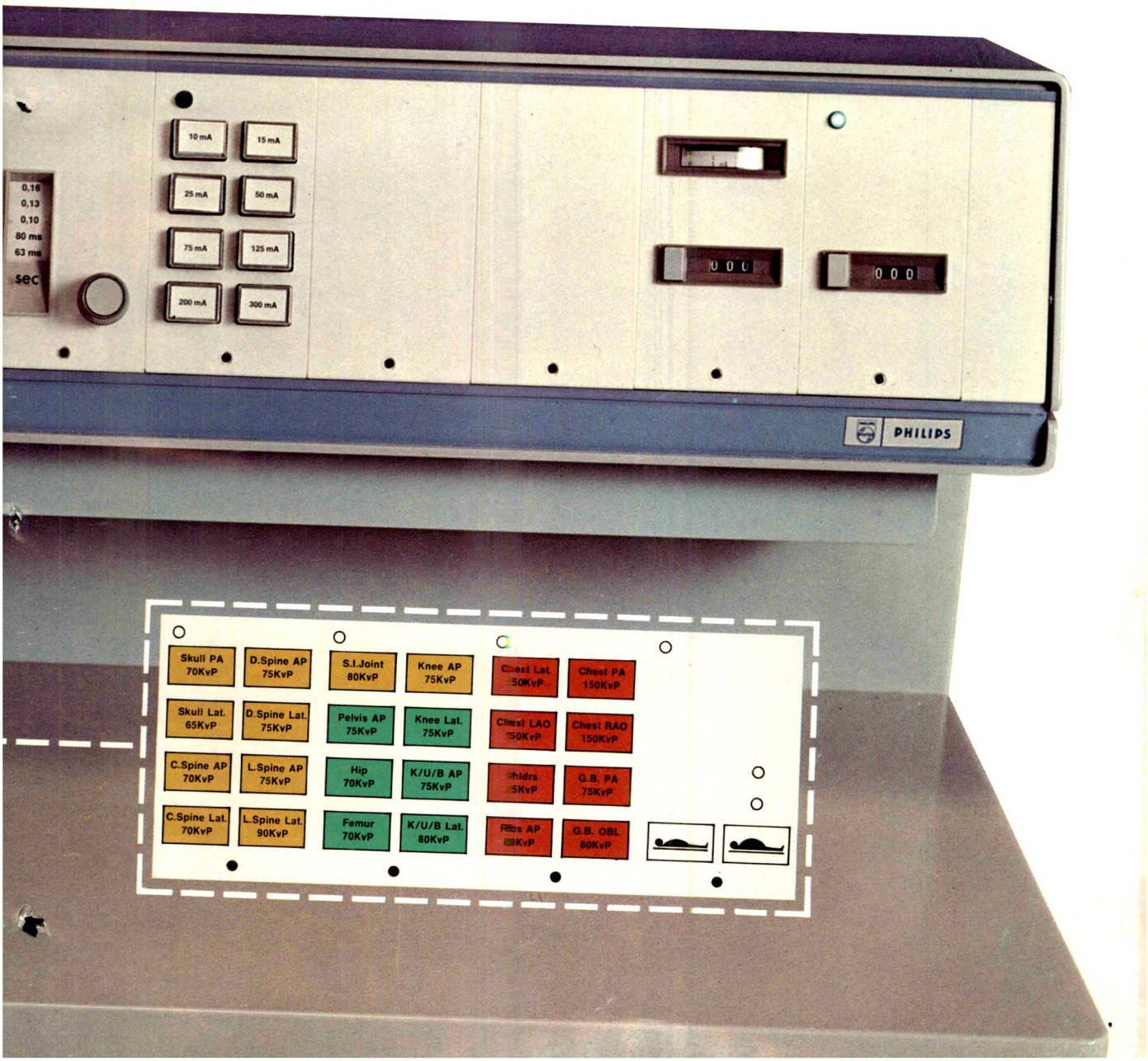
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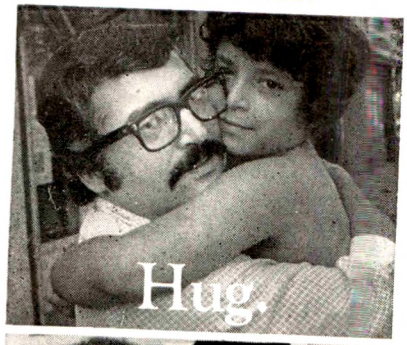
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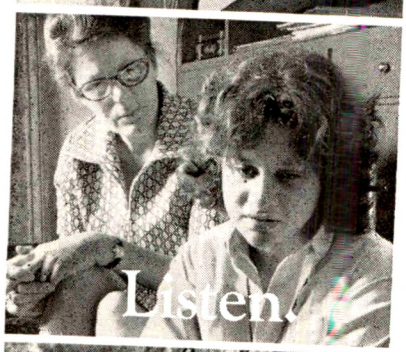
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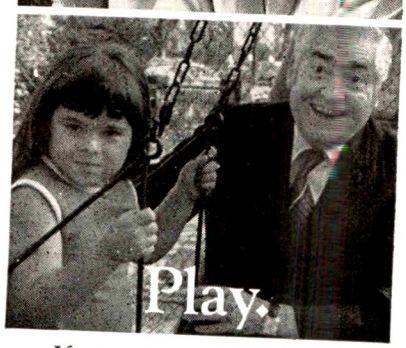
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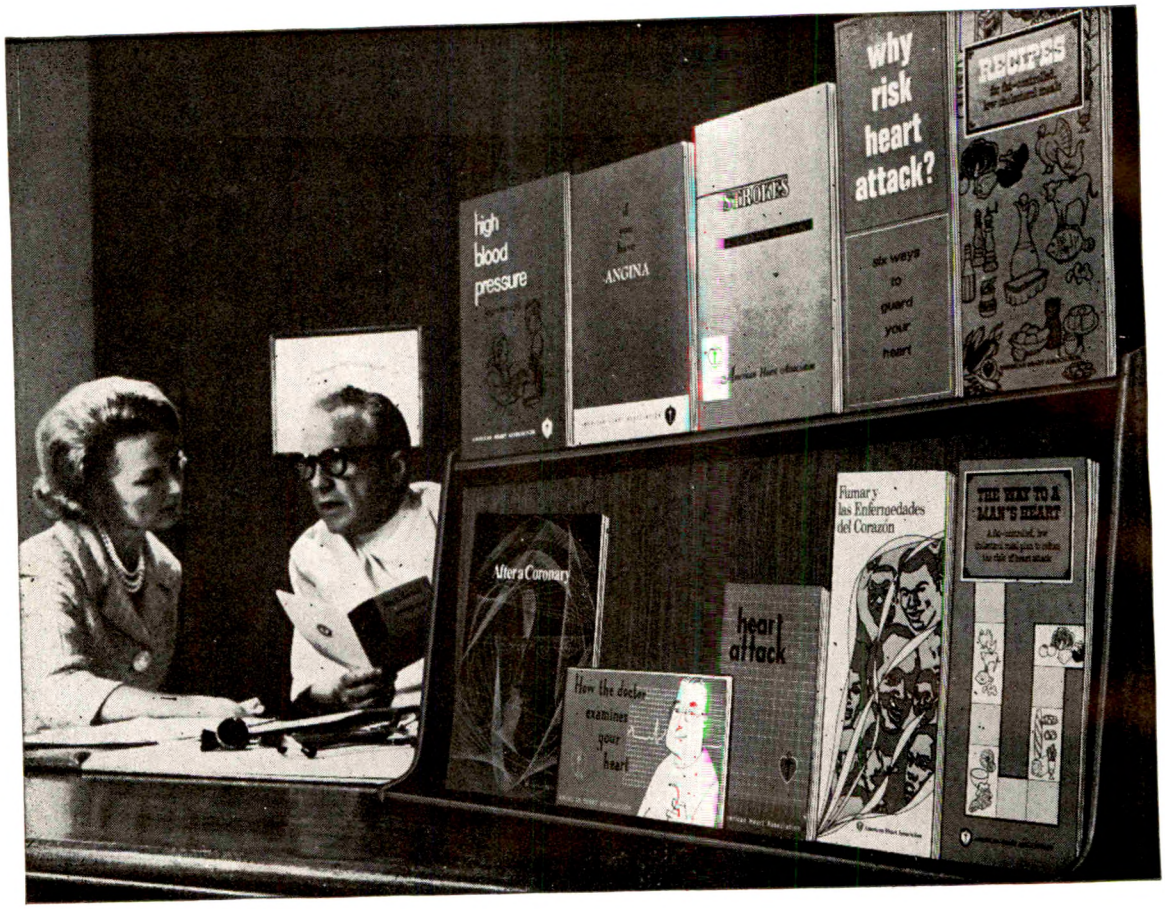
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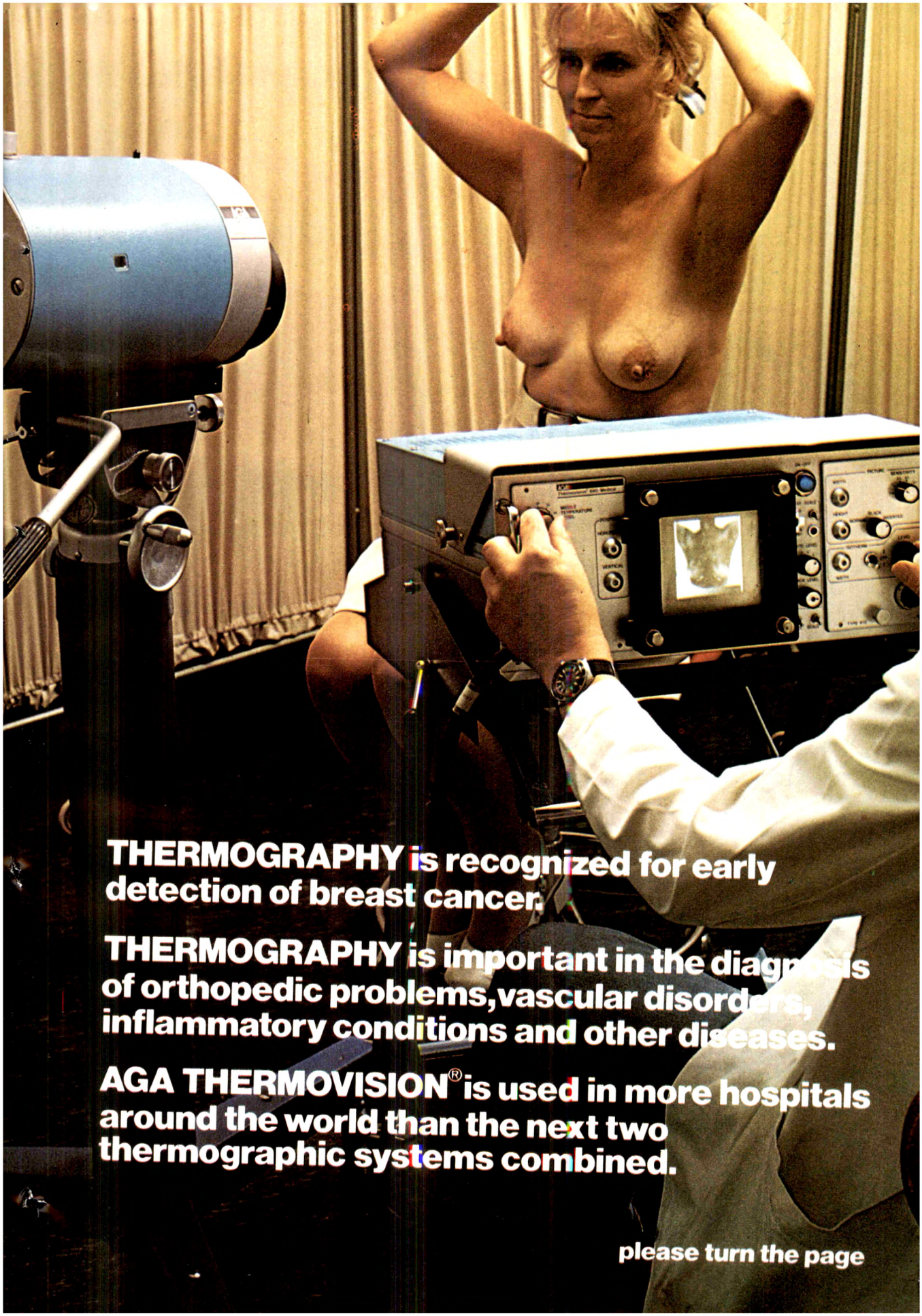
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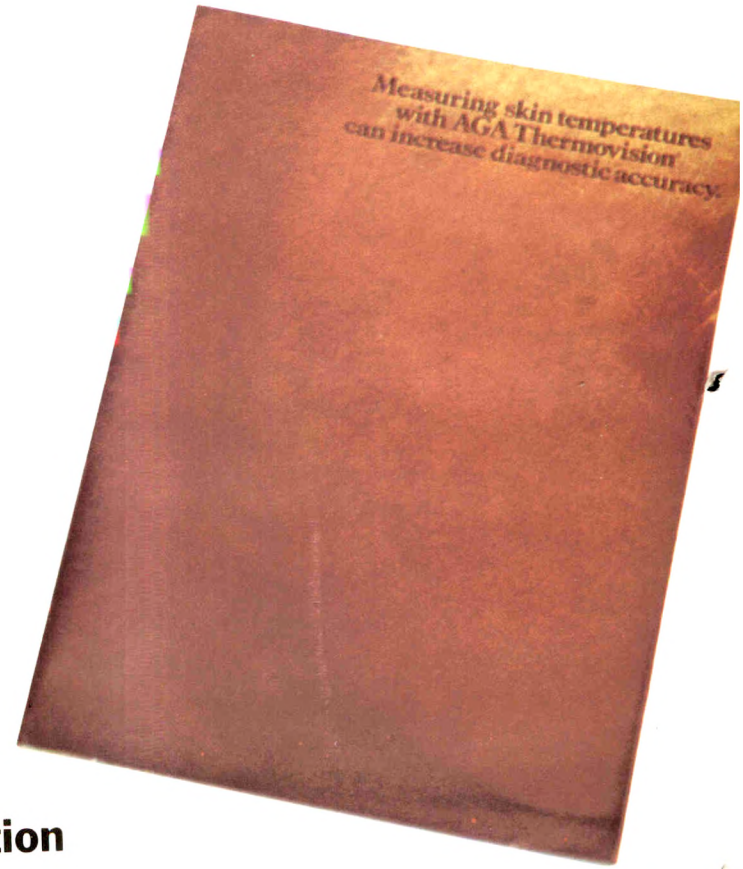
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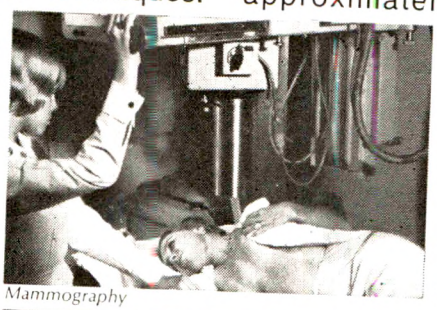
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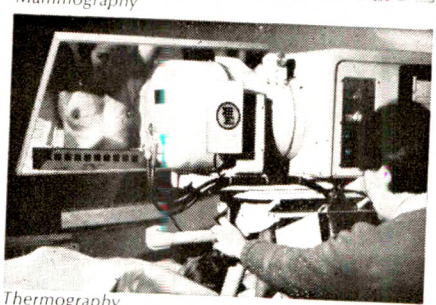
Breast Cancer: earlier warning system

Futility and frustration beset the physician confronted with breast cancer. For the last 35 years, the survival rate has not significantly changed despite intensive educational programs aimed at earlier detection, and improvement in treatment techniques.

What is the outlook? We know the key to reducing mortality from breast cancer is in the *earliest possible* diagnosis. The stage at which breast cancer is detected is *crucial* to the outcome of treatment. By the time a lump is discovered through BSE or clinical examination, critical time may have been lost.



Mammography

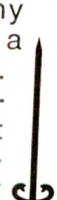


Thermography

And we *do* have the means to achieve earlier diagnosis. We do have an *earlier warning system.* Mammography and thermography can detect breast cancer *before* a lump is discernible by palpation. To demonstrate that it is practical and feasible to detect breast cancer earlier by using these modalities, the American Cancer Society and the National Cancer Institute are funding a network of breast cancer demonstration projects. Supported by grants of $2-million from the ACS and $4-million from the NCI, 20 such centers are expected to be operative across the country by the end of the year. Each will screen at no charge, approximately 5,000 women annually, in what is considered to be the ideal detection program — to include clinical examination, mammography and thermography. Each of these detection methods contributes independently to the detection of breast cancer, and none can be dispensed with in the search for early disease.

At present we cannot prevent breast cancer, but the potential for saving more lives is immense. The five-year survival rate changes dramatically from 53% when axillary nodes are positive, to 85% when the disease is localized, to nearly 100% for in-situ cancer.

We have an earlier warning system. Let's use it.



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- Controlling high blood pressure,
- Seeing your doctor regularly,

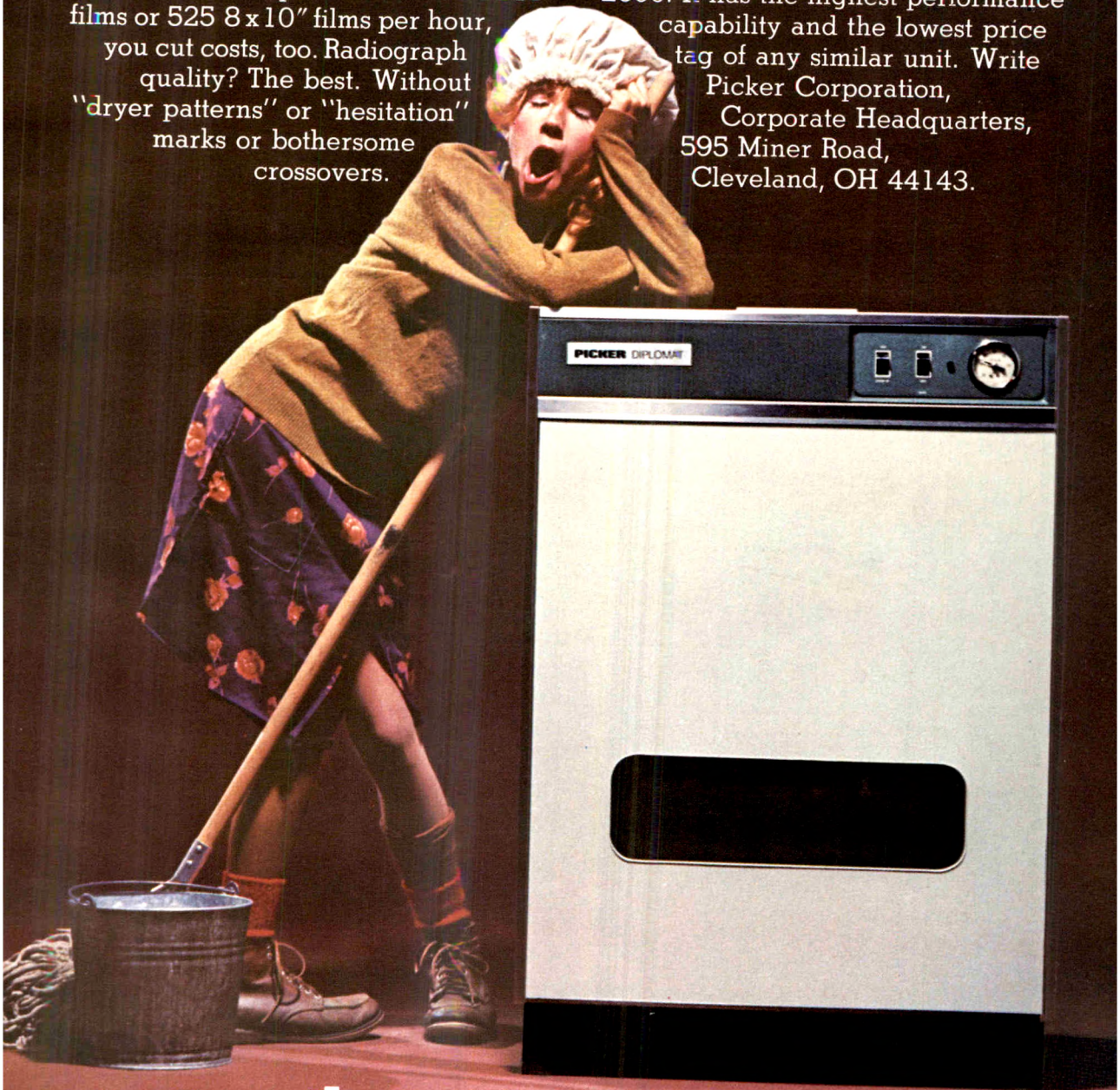
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